# NEET PG 2010

## Questions with Solutions

Detailed step-by-step solutions

### General Instructions

**(i)** This booklet contains 300 questions, each provided with a complete, step-by-step solution.

**(ii)** It comprises 300 single-correct multiple-choice questions.

**(iii)** These questions follow the NEET PG examination pattern.

**(iv)** Each correct answer is awarded +4 marks, with -1 for an incorrect answer.

**(v)** The actual NEET paper has 180 questions to be attempted in 3 hours 20 minutes.

**(vi)** Use of calculators is not permitted.

**(vii)** Attempt each question on your own before reviewing the given solution.

**1.** A patient with a bilirubin value of 8 mg/dl and a serum creatinine of 1.9 mg/dl is planned for surgery. What is the muscle relaxant of choice in this patient?

(A) Vecuronium

(B) Pancuronium

(C) Atracurium

(D) Rocuronium

**Correct Answer:** (C) Atracurium

**Solution:**

**Step 1:** Read the clinical picture. Bilirubin 8 mg/dl points to significant hepatic dysfunction, and creatinine 1.9 mg/dl points to impaired renal function. The ideal relaxant is one that does not depend on the liver or kidney for clearance.

**Step 2:** Recall the elimination routes. Atracurium is cleared by Hofmann elimination and non-specific plasma ester hydrolysis. Both are organ independent, so its clearance is unchanged in hepatic and renal failure.

**Step 3:** Eliminate the wrong options. Vecuronium is partly excreted in bile and partly renally, so it accumulates in hepatic and renal disease. Pancuronium is largely renally excreted and is strongly prolonged in renal failure. Rocuronium is mainly cleared by the liver, so its action is prolonged with raised bilirubin.

**Step 4:** Match drug to patient. With both liver and kidney impaired **(bilirubin = 8, creatinine = 1.9)**, only organ-independent clearance is safe.

The answer is **C. Atracurium**.

**Quick Tip:** Which relaxant clears by Hofmann elimination, independent of liver and kidney?

**2.** A 25 year old overweight female was given fentanyl-pancuronium anaesthesia for surgery. After surgery and extubation she was observed to have limited movement of the upper body and chest wall in the recovery room. She was conscious and alert but voluntary respiratory effort was limited. Her blood pressure and heart rate were normal. The likely diagnosis is:

(A) Incomplete reversal of pancuronium

(B) Pulmonary embolism

(C) Fentanyl induced chest wall rigidity

(D) Respiratory depression

**Correct Answer:** (A) Incomplete reversal of pancuronium

**Solution:**

**Step 1:** Identify the key findings. The patient is conscious and alert, so the brain is awake, yet she cannot move her upper body and chest wall and her voluntary respiratory effort is weak. Vital signs are normal.

**Step 2:** Awake but weak muscles is the signature of residual neuromuscular block. A patient with leftover pancuronium can be fully conscious but unable to

generate adequate muscle power, including for breathing.

**Step 3:** Pancuronium is long acting and largely renally cleared. In an overweight patient, dosing and recovery can be unpredictable, making incomplete reversal common, especially if reversal was inadequate.

**Step 4:** Exclude the distractors. Fentanyl induced chest wall rigidity occurs during induction when the patient is anaesthetised, not after extubation in an awake patient. Pulmonary embolism would cause tachycardia, hypotension and hypoxia, but her vitals are normal. Pure opioid respiratory depression would reduce alertness, yet she is conscious and alert.

**Step 5:** The combination of full consciousness with weak respiratory muscles fits residual paralysis.

The answer is **A. Incomplete reversal of pancuronium**.

**Quick Tip:** Awake and alert but weak breathing points to leftover muscle relaxant, not the opioid.

**3.** All of the following statements about the neuromuscular blockade produced by succinylcholine are true, except:

(A) No fade on train of four stimulation

(B) Fade on tetanic stimulation

(C) No post tetanic facilitation

(D) Train of four ratio $> 0.4$

**Correct Answer:** (B) Fade on tetanic stimulation

**Solution:**

**Step 1:** Recognise the drug type. Succinylcholine is a depolarising neuromuscular blocker. In a pure phase I (depolarising) block, the response to stimulation is

fundamentally different from a non-depolarising block.

**Step 2:** Recall the features of a phase I depolarising block. The four twitches of train of four are reduced but equally, so there is no fade. There is sustained but reduced response to tetanic stimulation, so there is no tetanic fade. There is no post tetanic facilitation. The train of four ratio stays high, typically near or above 0.7, well above 0.4.

**Step 3:** Test each option. Option A (no fade on train of four) is true. Option C (no post tetanic facilitation) is true. Option D (train of four ratio above 0.4) is true. Option B says there is fade on tetanic stimulation, which is a feature of a non-depolarising or phase II block, not of a pure depolarising block.

**Step 4:** The question asks for the exception, so we pick the false statement.

The answer is **B. Fade on tetanic stimulation**.

**Quick Tip:** Fade is the signature of a non-depolarising block, not a pure depolarising one.

**4.** A 27 year old female was brought to the emergency department for acute abdominal pain, following which she was shifted to the operation theatre for laparotomy. A speedy intubation was performed, but after the intubation breath sounds were observed to be decreased on the left side and a high end tidal CO2 was recorded. The likely diagnosis is:

(A) Endotracheal tube blockage

(B) Bronchospasm

(C) Esophageal intubation

(D) Endobronchial intubation

**Correct Answer:** (D) Endobronchial intubation

**Solution:**

**Step 1:** Pick out the two key signs. Breath sounds are decreased on the left side, and there is a recorded end tidal CO2.

**Step 2:** A present end tidal CO2 trace confirms the tube is in the airway, not the oesophagus. Oesophageal intubation gives little or no end tidal CO2, so option C is excluded.

**Step 3:** Decreased breath sounds on the left with the tube in the trachea suggests the tube has advanced too far. Because the right main bronchus is wider, shorter and more vertical, a tube pushed in too deep usually enters the right main bronchus. This ventilates only the right lung, so the left side has reduced breath sounds.

**Step 4:** Exclude the rest. Total tube blockage would cause no ventilation and a loss of the CO2 trace, not unilateral findings. Bronchospasm causes bilateral wheeze with raised airway pressures, not a one-sided silent chest after a hurried deep intubation.

**Step 5:** Unilateral decreased breath sounds with a maintained CO2 trace after rapid intubation is classic right endobronchial intubation.

The answer is **D. Endobronchial intubation**.

**Quick Tip:** A maintained CO2 trace with one quiet lung means the tube went too deep, not into the gullet.

**5.** The hypogastric sheath is a condensation of:

(A) Scarpa's fascia

(B) Colles' fascia

(C) Pelvic fascia

(D) Inferior layer of the urogenital diaphragm

**Correct Answer:** (C) Pelvic fascia

**Solution:**

**Step 1:** Define the structure. The hypogastric sheath is a layer of connective tissue in the pelvis that runs from the side wall of the pelvis to the pelvic organs and carries vessels and nerves with it.

**Step 2:** Locate its origin. It is a condensation of the visceral pelvic fascia (endopelvic fascia). It conveys the internal iliac vessels and their branches to the bladder, uterus and rectum, and it splits into laminae around these organs.

**Step 3:** Eliminate the wrong options. Scarpa's fascia is the deep membranous layer of the superficial fascia of the lower anterior abdominal wall. Colles' fascia is the membranous superficial fascia of the perineum. The inferior layer of the urogenital diaphragm (perineal membrane) lies in the perineum. None of these is the source of the hypogastric sheath.

**Step 4:** Only the pelvic fascia gives rise to this pelvic visceral condensation.

The answer is **C. Pelvic fascia**.

**Quick Tip:** It is a deep pelvic condensation carrying vessels to the bladder and uterus.

**6.** Which of the following positions best describes the location of the celiac plexus?

(A) Anterolateral to the aorta

(B) Posterolateral to the aorta

(C) Anterolateral to the sympathetic chain

(D) Anteromedial to the sympathetic chain

**Correct Answer:** (A) Anterolateral to the aorta

**Solution:**

**Step 1:** Place the plexus. The celiac plexus is the largest of the autonomic plexuses. It lies in the upper abdomen at the level of the first lumbar vertebra, around the root of the celiac trunk.

**Step 2:** Relate it to the aorta. The abdominal aorta lies in the midline against the vertebral bodies. The celiac plexus and its ganglia lie in front of and to the sides of the aorta, that is anterolateral to the aorta, surrounding the origins of the celiac trunk and superior mesenteric artery.

**Step 3:** Eliminate the wrong options. The plexus is not behind the aorta, so posterolateral to the aorta is wrong. The sympathetic chains lie further laterally along the sides of the vertebral bodies, so describing the plexus relative to the sympathetic chain does not give its best anatomical position; the plexus sits medial and anterior to the chains, not the way options C and D state.

**Step 4:** The clinical relevance is that a celiac plexus block, used for upper abdominal cancer pain, targets this anterolateral periaortic position.

The answer is **A. Anterolateral to the aorta**.

**Quick Tip:** It wraps the celiac trunk, which springs off the front of the aorta.

**7.** The gluteus medius muscle is supplied by which nerve?

(A) Superior gluteal nerve

(B) Inferior gluteal nerve

(C) Nerve to obturator internus

(D) Nerve to quadratus femoris

**Correct Answer:** (A) Superior gluteal nerve

**Solution:**

**Step 1:** Recall the gluteal nerve supply. The superior gluteal nerve (L4, L5, S1) leaves the pelvis above the piriformis through the greater sciatic foramen. The inferior gluteal nerve (L5, S1, S2) leaves below the piriformis.

**Step 2:** Match nerve to muscle. The superior gluteal nerve supplies the gluteus medius, gluteus minimus and tensor fasciae latae. The inferior gluteal nerve supplies only the gluteus maximus.

**Step 3:** So the gluteus medius is supplied by the superior gluteal nerve. Damage to this nerve weakens hip abduction and causes a positive Trendelenburg sign, where the pelvis drops on the unsupported side during walking.

**Step 4:** Exclude the rest. The inferior gluteal nerve supplies gluteus maximus, the nerve to obturator internus and the nerve to quadratus femoris supply those small lateral rotators, none of which is the gluteus medius.

**Step 5 (second part of the original question):** The arterial supply of the gluteus medius is from the superior gluteal artery, which accompanies the superior gluteal nerve above the piriformis, not the inferior gluteal, obturator or any ilioinguinal vessel.

The answer is **A. Superior gluteal nerve**.

**Quick Tip:** The nerve above piriformis that also causes a Trendelenburg sign when injured.

**8.** Which of the following tendons passes below the sustentaculum tali?

(A) Tibialis anterior

(B) Tibialis posterior

(C) Flexor digitorum longus

(D) Flexor hallucis longus

**Correct Answer:** (D) Flexor hallucis longus

**Solution:**

**Step 1:** Recall the sustentaculum tali. It is a shelf-like bony projection on the medial side of the calcaneus that supports the talus. Its under-surface carries a groove.

**Step 2:** Identify the tendon in that groove. The flexor hallucis longus tendon runs in the groove on the under-surface (inferior surface) of the sustentaculum tali. This is why the structure helps pulley the tendon toward the great toe.

**Step 3:** Exclude the other tendons. Tibialis anterior crosses the front of the ankle and inserts on the medial cuneiform and first metatarsal, nowhere near the sustentaculum. Flexor digitorum longus passes medial to and just above the sustentaculum, not under it.

**Step 4 (second part of the original question):** The tendon that gains attachment on the sustentaculum tali is the tibialis posterior. Tibialis posterior inserts mainly on the navicular tuberosity but also sends slips to the sustentaculum tali and other tarsal and metatarsal bones, so its attachment is on the sustentaculum, whereas flexor hallucis longus merely grooves its under-surface.

**Step 5:** So the tendon passing below the sustentaculum tali is flexor hallucis longus, and the tendon attaching to it is tibialis posterior.

The answer is **D. Flexor hallucis longus**.

**Quick Tip:** The shelf acts as a pulley for the tendon heading to the great toe.

**9.** Which of the following passes through the foramen magnum?

(A) Vertebral artery

(B) Sympathetic chain

(C) XIth cranial nerve

(D) Internal carotid artery

**Correct Answer:** (A) Vertebral artery

**Solution:**

**Step 1:** The foramen magnum is the large opening in the occipital bone that connects the posterior cranial fossa with the vertebral canal. Several structures pass through it.

**Step 2:** The structures passing through the foramen magnum are: the medulla oblongata with its meninges, both vertebral arteries, the anterior and posterior spinal arteries, the spinal (ascending) part of the accessory nerve, the apical ligament of the dens, the tectorial membrane, and the dural veins.

**Step 3:** The vertebral artery enters the cranial cavity by passing through the foramen magnum, so option A is correct.

**Step 4:** Why the others are wrong. The sympathetic chain runs in the neck and thorax along the vertebral column and does not pass through the foramen magnum. The XIth cranial nerve (accessory) is tricky: its *spinal root* ascends through the foramen magnum, but the accessory nerve as a whole exits the skull through the jugular foramen, so naming the cranial nerve XI as a foramen magnum structure is the distractor here. The internal carotid artery enters the skull through the carotid canal, not the foramen magnum.

**Answer: A. Vertebral artery**

**Quick Tip:** Think of the gateway where the medulla becomes the spinal cord, and which artery climbs in there.

**10.** All of the following movements occur during abduction of the shoulder, except:

(A) Elevation of humerus

(B) Axial rotation of clavicle

(C) Medial rotation of scapula

(D) Acromioclavicular joint movement

**Correct Answer:** (C) Medial rotation of scapula

**Solution:**

**Step 1:** Full abduction of the upper limb to 180 degrees is not a single joint action. It is a combined movement of the glenohumeral joint and the shoulder girdle, described by the scapulohumeral rhythm (about 2 degrees of glenohumeral movement for every 1 degree of scapular movement).

**Step 2:** What happens during abduction. The humerus elevates and rotates laterally at the glenohumeral joint (option A occurs). The scapula rotates so that the glenoid cavity turns upward; this is *lateral (upward) rotation* of the scapula, not medial rotation. To allow scapular rotation, movement must occur at the sternoclavicular and acromioclavicular joints (option D occurs).

**Step 3:** During this scapular rotation the clavicle also rotates about its own long axis (axial rotation of the clavicle, the so-called crankshaft mechanism at the sternoclavicular joint), so option B occurs.

**Step 4:** The exception is option C. The scapula undergoes **lateral rotation** during abduction, so describing it as *medial rotation of scapula* is the incorrect statement and is the answer to this except question.

**Answer: C. Medial rotation of scapula**

**Quick Tip:** During abduction the glenoid must turn upward, so which direction does the scapula really rotate?

**11.** All of the following are composite muscles, except:

(A) Flexor carpi ulnaris

(B) Flexor digitorum profundus

(C) Pectineus

(D) Biceps femoris

**Correct Answer:** (A) Flexor carpi ulnaris

**Solution:**

**Step 1:** A composite (or hybrid) muscle is a muscle that develops from two different muscle masses or is supplied by two different nerves, reflecting its dual embryological origin.

**Step 2:** Recognise the classic composite muscles. Flexor digitorum profundus is supplied by two nerves: the median nerve (anterior interosseous branch) to its radial half and the ulnar nerve to its medial half, so it is composite (option B). Pectineus is supplied by the femoral nerve and frequently also by the obturator (or accessory obturator) nerve, so it is composite (option C). Biceps femoris has two heads with two different nerve supplies: the long head from the tibial part of the sciatic nerve and the short head from the common peroneal (fibular) part of the sciatic nerve, so it is composite (option D).

**Step 3:** Flexor carpi ulnaris is supplied entirely by a single nerve, the ulnar nerve, and develops from the common flexor mass. It is therefore NOT a composite muscle.

**Step 4:** The except answer is the muscle with a single, uniform nerve supply.

**Answer: A. Flexor carpi ulnaris**

**Quick Tip:** A composite muscle has a dual nerve supply; find the one fed by only one nerve.

**12.** The urogenital diaphragm is made up of the following, except:

(A) Deep transverse perinei

(B) Perineal membrane

(C) Colles fascia

(D) Sphincter urethrae

**Correct Answer:** (C) Colles fascia

**Solution:**

**Step 1:** The urogenital diaphragm (deep perineal pouch in classical description) is a musculofascial sheet stretched across the anterior part of the perineum between the two ischiopubic rami.

**Step 2:** Its components are: the deep transverse perinei muscle and the sphincter urethrae muscle, sandwiched between two fascial layers, the superior fascia of the urogenital diaphragm above and the inferior fascia (the perineal membrane) below. So options A, B and D are all genuine parts of the urogenital diaphragm.

**Step 3:** Colles fascia is the membranous layer of the superficial fascia of the perineum. It forms the floor of the superficial perineal pouch, NOT a part of the deep urogenital diaphragm.

**Step 4:** Therefore the structure that is not part of the urogenital diaphragm is Colles fascia.

**Answer: C. Colles fascia**

**Quick Tip:** The diaphragm is two muscles between two fascial layers; one named

fascia belongs to the superficial pouch instead.

**13.** In post-ductal coarctation of the aorta, collaterals may be formed by all of the following, except:

(A) Vertebral artery

(B) Suprascapular artery

(C) Subscapular artery

(D) Posterior intercostal artery

**Correct Answer:** (A) Vertebral artery

**Solution:**

**Note on the key:** The recalled paper printed no answer for this item. The medically correct answer is determined below from the anatomy of the collateral circulation.

**Step 1:** In post-ductal coarctation, the narrowing lies distal to the left subclavian artery. Blood must bypass the obstruction to reach the descending aorta. The collaterals connect branches arising *above* the coarctation (mainly from the subclavian arteries) with branches that join the descending aorta *below* the coarctation (chiefly the posterior intercostal arteries).

**Step 2:** The main pathway uses the anterior intercostal branches of the internal thoracic artery and the branches of the subclavian artery (including the costocervical trunk, the lateral thoracic and the scapular anastomosis). Blood flows back through the posterior intercostal arteries into the descending aorta below the block. This rib-notching anastomosis is the hallmark.

**Step 3:** The scapular anastomosis (suprascapular and subscapular arteries, both ultimately fed from the subclavian/axillary system) carries blood toward the posterior intercostals, so the suprascapular artery (B), the subscapular artery (C) and the posterior intercostal arteries (D) are all genuine collateral channels.

**Step 4:** The vertebral artery passes upward into the skull to supply the brain; it does not contribute to the chest wall collateral pathway that bypasses an aortic coarctation. It is therefore the exception.

**Answer: A. Vertebral artery**

**Quick Tip:** Collaterals link subclavian branches to the intercostals; which option heads to the brain instead of the chest wall?

**14.** A left-sided superior vena cava drains into:

(A) Right atrium

(B) Left atrium

(C) Coronary sinus

(D) Pericardial space

**Correct Answer:** (C) Coronary sinus

**Solution:**

**Note on the key:** The recalled paper printed option A (right atrium). The classic and medically correct answer is the coronary sinus, so the key has been corrected to option C, with the reasoning below.

**Step 1:** A persistent left superior vena cava (LSVC) is the commonest thoracic venous anomaly. It results from failure of the left anterior cardinal vein to obliterate during development.

**Step 2:** The left common cardinal vein and the left horn of the sinus venosus normally regress; the left horn of the sinus venosus persists as the coronary sinus. When the left anterior cardinal vein fails to involute, it persists as an LSVC, which descends on the left, receives the left brachiocephalic territory, and drains into the coronary sinus.

**Step 3:** The dilated coronary sinus then empties into the right atrium in the usual way, so blood ultimately reaches the right atrium but through the coronary sinus. The direct termination of the LSVC is the coronary sinus, which is what this question asks.

**Step 4:** In the rare variant where the LSVC drains into the left atrium it causes a right-to-left shunt, but the typical, expected answer for this question is the coronary sinus, so options A, B and D are not the best answer.

**Answer: C. Coronary sinus**

**Quick Tip:** The left horn of the sinus venosus becomes the coronary sinus; the persistent left vein drains there.

**15.** All of the following statements about bronchial circulation are true, except:

(A) It contributes about 2% of systemic circulation

(B) It contributes to gaseous exchange

(C) It causes venous admixture of blood

(D) It provides nutritive function to the lung

**Correct Answer:** (B) It contributes to gaseous exchange

**Solution:**

**Step 1:** The lung has two circulations. The pulmonary circulation carries deoxygenated blood from the right ventricle and is responsible for gas exchange. The bronchial circulation is a small part of the systemic circulation that nourishes the lung tissue itself.

**Step 2:** Check each statement. The bronchial arteries carry about 1 to 2% of the systemic cardiac output, so option A is true. The bronchial circulation supplies oxygen and nutrients to the bronchi, bronchioles, connective tissue and pleura,

that is its nutritive role, so option D is true.

**Step 3:** Part of the bronchial venous blood (which is deoxygenated systemic blood) drains into the pulmonary veins. This adds a small amount of poorly oxygenated blood to the freshly oxygenated pulmonary venous blood, producing a physiological venous admixture (one cause of the normal small alveolar to arterial oxygen difference). So option C is true.

**Step 4:** Gas exchange (oxygenation of venous blood) is the job of the **pulmonary** circulation across the alveolar capillary membrane, NOT the bronchial circulation. So saying the bronchial circulation contributes to gaseous exchange is false, and that is the except answer.

**Answer: B. Contributes to gaseous exchange**

**Quick Tip:** Bronchial circulation feeds lung tissue; which job actually belongs to the pulmonary circulation?

**16.** An important non-respiratory function of the lungs is:

(A) Anion balance

(B) Sodium balance

(C) Potassium balance

(D) Calcium balance

**Correct Answer:** (B) Sodium balance

**Solution:**

**Note on the key:** The recalled paper printed option B (sodium balance). On strict physiology this is debatable, but the intended answer relates to the lung influencing the acid base and ionic status of blood; the printed key is retained as B with the reasoning below.

**Step 1:** Besides gas exchange, the lungs perform many non-respiratory functions: filtering small clots, metabolising vasoactive substances (converting angiotensin I to angiotensin II via ACE, inactivating bradykinin and serotonin), acting as a blood reservoir, and contributing to acid base balance by controlling carbon dioxide.

**Step 2:** By regulating the elimination of CO2, the lungs control the carbonic acid component of the blood and thereby the bicarbonate buffer. This respiratory control of acid base status influences the handling and distribution of body electrolytes, including sodium, by the linked renal and acid base systems.

**Step 3:** Of the options offered, sodium balance is the electrolyte most closely tied to the lung through the angiotensin converting enzyme step: pulmonary ACE generates angiotensin II, which drives aldosterone release and sodium retention. This places the lung in the loop that governs sodium handling.

**Step 4:** Potassium, calcium and the broad anion balance are governed mainly by the kidney, gut and parathyroid hormone, not by the lung. So the best single answer among the choices is sodium balance.

**Answer: B. Sodium balance**

**Quick Tip:** Recall the enzyme on lung endothelium that activates angiotensin II and drives aldosterone.

**17.** Maximum water reabsorption in the gastrointestinal tract occurs in:

(A) Stomach

(B) Jejunum

(C) Ileum

(D) Colon

**Correct Answer:** (B) Jejunum

**Solution:**

**Step 1:** Each day the gut handles a huge fluid load. About 2 litres come from food and drink, and the remaining 7 litres are secretions (saliva, gastric juice, bile, pancreatic juice, intestinal juice). So roughly 9 litres of water enter the lumen daily.
**Step 2:** Of this, the small intestine reabsorbs the bulk, close to 8 to 8.5 litres. The colon recovers only about 0.5 to 1 litre, and barely 0.1 litre is lost in stool.
**Step 3:** Within the small intestine, water follows the osmotic gradient set up mainly by sodium and nutrient (glucose, amino acid) absorption. This solute uptake is most active in the proximal small intestine, so the **jejunum** reabsorbs the largest share of water.
**Step 4:** The other options absorb far less. The stomach absorbs negligible water. The ileum reabsorbs bile salts and vitamin $B_{12}$ but less water than the jejunum. The colon concentrates stool but handles a much smaller volume.
**Conclusion:** Maximum water reabsorption occurs in the jejunum. Answer is option B.

**Quick Tip:** The proximal small bowel handles the largest fluid volume, not the colon.

**18.** The primary action of Nitric oxide (NO) in the gastrointestinal tract is:

(A) Vasodilatation

(B) Vasoconstriction

(C) Gastrointestinal smooth muscle relaxation

(D) Gastrointestinal slow smooth muscle contraction

**Correct Answer:** (C) Gastrointestinal smooth muscle relaxation

**Solution:**

**Step 1:** Nitric oxide in the gut is released by enteric inhibitory neurons. These belong to the non-adrenergic, non-cholinergic (NANC) nerves of the enteric nervous system.

**Step 2:** NO diffuses into smooth muscle cells and activates the enzyme guanylyl cyclase. This raises intracellular cyclic GMP, which lowers cytosolic calcium and causes the muscle to relax.
**Step 3:** In the gut this relaxation is essential for normal motility. It mediates receptive relaxation of the stomach, descending relaxation ahead of a peristaltic wave, and relaxation of sphincters such as the lower oesophageal sphincter and internal anal sphincter.
**Step 4:** Although NO is a vasodilator in blood vessels, the question asks about its **primary gastrointestinal action**, which is relaxation of GI smooth muscle. So vasodilatation (A) is a vascular effect, not the principal gut role here. Vasoconstriction (B) and slow contraction (D) are the opposite of what NO does.
**Conclusion:** The primary GI action of NO is gastrointestinal smooth muscle relaxation. Answer is option C.

**Quick Tip:** NO is the chief NANC inhibitory transmitter of the gut wall.

**19.** Hot water bottle relieves pain of abdominal spasm by:

(A) Stimulation of adrenergic fibers

(B) Stimulation of cholinergic fibers

(C) Inhibition of cold receptors

(D) Inhibition of heat receptors

**Correct Answer:** (A) Stimulation of adrenergic fibers

**Solution:**

**Step 1:** Abdominal cramp pain comes from spasm of the smooth muscle in the gut wall. Relief means relaxing that muscle and damping the pain signals.
**Step 2:** Applying surface heat with a hot water bottle warms the abdominal wall and the underlying viscera. Local warmth promotes smooth muscle relaxation and increases blood flow, which helps wash out the pain-producing metabolites of the cramp.
**Step 3:** In the gut, sympathetic (adrenergic) activity inhibits motility and relaxes

smooth muscle, whereas parasympathetic (cholinergic) activity increases contraction. So a response that eases spasm aligns with an adrenergic, muscle-relaxing effect, while cholinergic stimulation would worsen the spasm. This is why option B is wrong.

**Step 4:** Heat also reduces pain by counterirritation, a gate-control type mechanism in which warm-sensitive input dampens the transmission of pain. Heat does not work by inhibiting cold receptors (C) or by inhibiting heat receptors (D); in fact heat receptors are stimulated, not inhibited.

**Note on the key:** The recalled answer key is A. Of the four options offered, stimulation of adrenergic fibres is the choice consistent with smooth muscle relaxation and spasm relief, so the printed key is retained.

**Conclusion:** Answer is option A.

**Quick Tip:** Spasm relief means smooth muscle relaxation, which in the gut is the adrenergic effect.

**20.** Vitamin K dependent coagulation factors include:

(A) II and III

(B) IX and X

(C) III and V

(D) VIII and XII

**Correct Answer:** (B) IX and X

**Solution:**

**Step 1:** Vitamin K acts as a cofactor for the enzyme that carboxylates glutamate residues on certain clotting factors. This gamma-carboxylation lets these factors bind calcium and attach to phospholipid surfaces, which they need to work.

**Step 2:** The vitamin K dependent clotting factors are **II, VII, IX and X** (a useful memory aid is the numbers 1972, that is factors 10, 9, 7, 2). Protein C and protein S, two anticoagulants, are also vitamin K dependent.

**Step 3:** Now check the options. Option B lists IX and X, both of which are on the

list, so it is correct.

**Step 4:** The other options each contain a factor that is not vitamin K dependent. Option A pairs II (dependent) with III, which is tissue factor and is not vitamin K dependent. Option C has III and V, neither of which is vitamin K dependent. Option D has VIII and XII, neither of which is vitamin K dependent.

**Conclusion:** Factors IX and X are vitamin K dependent. Answer is option B.

**Quick Tip:** Remember the numbers 1972: factors 10, 9, 7 and 2.

**21.** During heavy exercise the cardiac output (CO) increases up to five fold while pulmonary arterial pressure rises very little. This physiological ability of the pulmonary circulation is best explained by:

(A) Increase in the number of open capillaries

(B) Sympathetically mediated greater distensibility of pulmonary vessels

(C) Large amount of smooth muscle in pulmonary arteries

(D) Smaller surface area of pulmonary circulation

**Correct Answer:** (A) Increase in the number of open capillaries

**Solution:**

**Step 1:** The pulmonary circulation is a low pressure, low resistance, high compliance system. It must accept the same output as the systemic side, yet it can take a large rise in flow without much rise in pressure.

**Step 2:** At rest many lung capillaries, especially at the apices, are collapsed or carry little flow because pressure there is low. There is a large reserve of unused capillary bed.

**Step 3:** When cardiac output rises in exercise, the pulmonary pressure increases slightly, and this small rise opens previously closed capillaries. This is called **recruitment**. Already open capillaries also widen, called distension. Both lower pulmonary vascular resistance.

**Step 4:** Because recruitment and distension drop resistance just as flow increases, pulmonary arterial pressure barely changes despite a five fold rise in

output. Among the options, increase in the number of open capillaries (recruitment) is the best explanation.
**Step 5:** The wrong options: pulmonary vessels actually have little smooth muscle and are not the main reason here (C is wrong), the protective fall in resistance is passive and not chiefly sympathetic distensibility (B is wrong), and the pulmonary capillary surface area is huge, not small (D is wrong).
**Conclusion:** Answer is option A.

**Quick Tip:** Think recruitment of resting capillaries that lowers pulmonary vascular resistance.

**22.** Venous return to the heart during quiet standing is facilitated by all of the following factors, except:

(A) Calf muscle contraction during standing

(B) Valves in perforators

(C) Sleeves of deep fascia

(D) Gravitational increase in arterial pressure

**Correct Answer:** (D) Gravitational increase in arterial pressure

**Solution:**

**Step 1:** This is a negative question. Three options help return venous blood to the heart against gravity while standing, and one does not.
**Step 2:** The calf muscle pump (A) is the main mechanism. Contraction of the calf muscles squeezes the deep veins and pushes blood upward toward the heart, helped by the one way valves.
**Step 3:** Valves in the perforating veins (B) allow flow only from superficial to deep veins and prevent backflow, so they aid upward return and stop pooling.
**Step 4:** The tight sleeve of deep fascia around the leg muscles (C) makes the muscle pump more effective by ensuring contractions compress the deep veins rather than letting them bulge.
**Step 5:** Gravity during standing does raise the arterial pressure in the feet, but it

equally raises the venous pressure and makes blood pool in the legs. Gravity opposes venous return to the heart, it does not assist it. So option D is the exception.

**Conclusion:** The factor that does not facilitate venous return is the gravitational increase in arterial pressure. Answer is option D.

**Quick Tip:** Gravity makes blood pool in the legs while standing, so it hinders venous return.

**23.** During cardiac imaging the phase of minimum motion of the heart is:

(A) Late systole

(B) Mid systole

(C) Late diastole

(D) Mid diastole

**Correct Answer:** (D) Mid diastole

**Solution:**

**Step 1:** In cardiac imaging such as CT coronary angiography, motion blur is least when the heart is most still. The goal is to time image capture to the quietest part of the cardiac cycle.

**Step 2:** The heart has two relatively still windows: end systole, and mid diastole during the phase called diastasis. Diastasis is the slow filling period that sits between the early rapid filling of the ventricle and the late atrial contraction.

**Step 3:** At normal and slow heart rates the longest and most reliable rest period is mid diastole (diastasis), so this is the phase used for the best images of the coronary arteries.

**Step 4:** Comparing the options, late systole and mid systole still involve vigorous wall motion, and late diastole includes the atrial kick which moves the heart again. Mid diastole, the diastasis phase, has the least motion.

**Conclusion:** The phase of minimum cardiac motion is mid diastole. Answer is option D.

**Quick Tip:** The diastasis period of slow filling in mid diastole is the quietest moment.

**24.** Insulin secretion is inhibited by:

(A) Secretin

(B) Epinephrine

(C) Growth hormone

(D) Gastrin

**Correct Answer:** (B) Epinephrine

**Solution:**

**Step 1:** Insulin is released from the beta cells of the pancreatic islets. Its secretion is controlled by nutrients, hormones, and the autonomic nervous system.
**Step 2:** Epinephrine inhibits insulin secretion. It acts on alpha-2 adrenergic receptors on the beta cell, which lower cyclic AMP and suppress insulin release. This makes sense during stress and the fight or flight response, when the body needs to keep blood glucose high for energy rather than store it.
**Step 3:** The other options actually promote insulin secretion. Secretin and gastrin are gut hormones with an incretin-like effect that augments insulin release after a meal. Growth hormone, although it antagonises insulin action over time and is diabetogenic, stimulates insulin secretion and over the long term tends to raise insulin levels.
**Step 4:** So the only option that directly inhibits insulin secretion is epinephrine, acting through alpha-2 receptors.
**Conclusion:** Insulin secretion is inhibited by epinephrine. Answer is option B.

**Quick Tip:** Stress hormones acting on alpha-2 receptors switch insulin off.

**25.** Which of the following is not seen in humans?

(A) Estrous cycle

(B) Menstrual cycle

(C) Endometrial cycle

(D) Ovarian cycle

**Correct Answer:** (A) Estrous cycle

**Solution:**

**Step 1:** The question asks which reproductive cycle is absent in humans.
**Step 2:** The estrous cycle is the reproductive cycle of most female mammals (rats, dogs, cows, etc.). In an estrous cycle the female is sexually receptive only during a defined period called estrus (heat), and if fertilisation does not occur the thickened endometrium is reabsorbed rather than shed.
**Step 3:** Humans and other higher primates instead have a menstrual cycle. Here the endometrium that is not used for implantation is shed as menstrual bleeding, and the female can be receptive throughout the cycle.
**Step 4:** The menstrual cycle is the overall cycle in humans, and it has component cycles described together: the ovarian cycle (follicular and luteal phases in the ovary) and the endometrial (uterine) cycle (proliferative, secretory and menstrual phases of the endometrium). All three are seen in humans.
**Step 5:** Therefore the cycle not seen in humans is the estrous cycle, while the menstrual, endometrial and ovarian cycles are all present.
Answer: Option A (Estrous cycle).

**Quick Tip:** Humans menstruate; they do not have a heat or estrus period.

**26.** Lesions of the lateral cerebellum cause all of the following, except:

(A) Incoordination

(B) Intention tremor

(C) Resting tremor

(D) Ataxia

**Correct Answer:** (C) Resting tremor

**Solution:**

**Step 1:** The lateral cerebellum (the cerebellar hemispheres, also called the cerebrocerebellum) plans and coordinates skilled, learned movements of the limbs. Damage here produces signs on the same side of the body.
**Step 2:** Typical cerebellar signs include incoordination of voluntary movement (asynergia/dysmetria), intention tremor (a tremor that appears and worsens as the hand approaches a target), and ataxia (an unsteady, broad-based gait and clumsy limb movement). Options A, B and D all fit cerebellar disease.
**Step 3:** A resting tremor is a tremor present when the limb is at rest and is the classic sign of basal ganglia disease, especially Parkinson disease, not of the cerebellum. In fact, cerebellar (intention) tremor is the opposite pattern: it is absent at rest and appears with movement.
**Step 4:** So the feature that does not belong to a lateral cerebellar lesion is resting tremor.
Answer: Option C (Resting tremor).

**Quick Tip:** Resting tremor is a basal ganglia sign; cerebellum gives action or intention tremor.

**27.** Basal Metabolic Rate depends most closely on:

(A) Lean body mass

(B) Body mass index

(C) Obesity

(D) Body surface area

**Correct Answer:** (A) Lean body mass

**Solution:**

**Step 1:** Basal metabolic rate (BMR) is the energy the body uses at complete rest, in the post-absorptive state, to keep basic functions going.

**Step 2:** Most of this resting energy is spent by metabolically active tissue: skeletal muscle, liver, brain, heart and kidney. These tissues make up the lean (fat-free) body mass. Fat tissue is relatively inactive metabolically and contributes little.
**Step 3:** Because the active tissues are concentrated in lean mass, BMR correlates more closely with lean body mass than with total body weight. A muscular person has a higher BMR than a fatter person of the same weight.
**Step 4:** BMR has traditionally been expressed per unit body surface area, and surface area is a useful and convenient way to standardise and report BMR. But the actual physiological determinant, the tissue doing the work, is the lean body mass. The question asks what BMR depends on most closely, so lean body mass is the best answer over surface area, BMI or obesity.
Answer: Option A (Lean body mass).

**Quick Tip:** Fat is metabolically lazy; resting energy is set by metabolically active lean tissue.

**28.** Decreased Basal Metabolic Rate is seen in:

(A) Obesity

(B) Hyperthyroidism

(C) Feeding

(D) Exercise

**Correct Answer:** (A) Obesity

**Solution:**

**Step 1:** BMR is set mainly by metabolically active lean tissue and is raised by anything that increases tissue metabolism.
**Step 2:** Hyperthyroidism raises BMR strongly because thyroid hormone increases oxygen consumption and heat production in almost all tissues. So option B raises, not lowers, BMR.
**Step 3:** Feeding raises energy expenditure through the specific dynamic action (thermic effect) of food, especially protein. Exercise increases muscle metabolism

and oxygen demand, so it also raises metabolic rate. Options C and D both increase metabolism.

**Step 4:** In obesity the extra weight is largely fat, which is metabolically inactive. When BMR is expressed per unit body weight or per unit surface area, obese subjects show a relatively low value, so BMR per unit mass is decreased in obesity. Among the four choices, obesity is the condition linked with a decreased BMR.

**Step 5:** Therefore the answer is obesity.

Answer: Option A (Obesity).

**Quick Tip:** Thyroid up, food and exercise all raise BMR; fat mass dilutes it.

**29.** Low CSF protein may be seen in all of the following conditions, except:

(A) Recurrent lumbar puncture

(B) Hypothyroidism

(C) Pseudotumor cerebri

(D) Infants

**Correct Answer:** (B) Hypothyroidism

**Solution:**

**Step 1:** Normal CSF protein in adults is roughly 15 to 45 mg/dL. The question asks which listed condition does not cause a low CSF protein.

**Step 2:** Recurrent or repeated lumbar punctures and chronic CSF leak drain protein-containing fluid faster than it is replaced, so CSF protein falls. This is a recognised cause of low CSF protein.

**Step 3:** Pseudotumor cerebri (idiopathic intracranial hypertension) classically shows raised CSF pressure with normal or low CSF protein; a low value is a typical accepted finding. Young infants normally have low CSF protein production patterns in some references and are listed as a low-protein group.

**Step 4:** Hypothyroidism (myxedema) is associated with an increased CSF protein, not a decreased one. It is a known cause of elevated CSF protein.

Therefore it does not belong with the low-protein conditions.
**Step 5:** The exception is hypothyroidism.
Answer: Option B (Hypothyroidism).

**Quick Tip:** Myxedema raises CSF protein, it does not lower it.

**30.** Sphingomyelinase deficiency is seen in:

(A) Niemann-Pick disease

(B) Farber's disease

(C) Tay-Sachs disease

(D) Krabbe's disease

**Correct Answer:** (A) Niemann-Pick disease

**Solution:**

**Step 1:** These are all sphingolipidoses, a group of lysosomal storage disorders. Each is defined by a specific missing enzyme and the substrate that then accumulates.
**Step 2:** Sphingomyelinase (acid sphingomyelinase) normally breaks down sphingomyelin into ceramide and phosphocholine. Its deficiency means sphingomyelin builds up. This is Niemann-Pick disease (types A and B), which presents with hepatosplenomegaly and, in type A, neurodegeneration and a cherry-red macular spot.
**Step 3:** The other options use different enzymes: Farber disease is ceramidase deficiency (ceramide accumulates); Tay-Sachs disease is hexosaminidase A deficiency (GM2 ganglioside accumulates); Krabbe disease is galactocerebrosidase (beta-galactocerebrosidase) deficiency (galactocerebroside accumulates).
**Step 4:** So sphingomyelinase deficiency maps to Niemann-Pick disease.
Answer: Option A (Niemann-Pick disease).

**Quick Tip:** Sphingomyelin builds up, so think of the disease named for that lipid.

**31.** Which of the following lipoproteins does not move towards the charged end in electrophoresis?

(A) VLDL

(B) LDL

(C) HDL

(D) Chylomicrons

**Correct Answer:** (D) Chylomicrons

**Solution:**

**Step 1:** On lipoprotein electrophoresis the particles separate by their net charge and size. Migration position gives each class its alternative Greek-letter name.
**Step 2:** HDL is the most charged and moves fastest, taking the alpha position. VLDL moves to the pre-beta position. LDL moves to the beta position. All three migrate away from the origin towards the charged (anode) end.
**Step 3:** Chylomicrons are huge, triglyceride-rich and have very little protein relative to their size, so they have very low surface charge and a high mass. They essentially do not migrate and stay at the point of application (the origin).
**Step 4:** Therefore the lipoprotein that does not move towards the charged end is the chylomicron.
Answer: Option D (Chylomicrons).

**Quick Tip:** The biggest, most fat-laden particle stays at the start line.

**32.** Inulin like fructans are used as prebiotics as they are non digestible. Resistance to digestion in the upper GI tract results from:

(A) Absence of digestive enzyme in the upper GIT

(B) Beta configuration of anomeric C2

(C) Low pH of the stomach

(D) Presence of alpha-osidic linkages

**Correct Answer:** (B) Beta configuration of anomeric C2

**Solution:**

**Step 1:** Inulin is a fructan, a chain of fructose units joined mostly by beta(2 to 1) glycosidic bonds, usually with a terminal glucose. Prebiotics work because the upper gut cannot digest them, so they reach the colon to feed beneficial bacteria.
**Step 2:** Human digestive enzymes such as salivary and pancreatic amylase and the brush-border maltases act on alpha glycosidic bonds (as in starch). They cannot hydrolyse beta linkages.
**Step 3:** In inulin the fructose units are linked through the anomeric carbon of fructose, which is C2, in the beta configuration. Because the bond is in the beta form at the anomeric C2, no human enzyme can cleave it, so inulin passes undigested through the upper GI tract.
**Step 4:** Checking the distractors: option A is too vague and not the chemical reason; humans simply lack a beta-fructosidase, but the bond chemistry is the precise cause. Option C, low stomach pH, does not break these bonds and is not why it resists. Option D is wrong because the linkage is beta, not alpha; alpha linkages would in fact be digestible.
**Step 5:** The correct chemical reason is the beta configuration at the anomeric C2. Answer: Option B (Beta configuration of anomeric C2).

**Quick Tip:** Human amylases break alpha bonds only; inulin is linked in the beta form.

**33.** Method of chromatography in which negatively charged molecules are selectively released from the stationary phase by positively charged molecules in the mobile phase is termed:

(A) Affinity chromatography

(B) Ion-exchange chromatography

(C) Adsorption chromatography

(D) Size-exclusion chromatography

**Correct Answer:** (B) Ion-exchange chromatography

**Solution:**

**Step 1:** Identify the separation principle in the stem. The molecules are separated based on their net electrical charge. The stationary phase holds charged molecules, and they are displaced (eluted) by other charged species in the mobile phase. Charge-based separation points to ion-exchange chromatography.
**Step 2:** In ion-exchange chromatography the stationary resin carries fixed charged groups. A cation-exchange resin carries negative groups and binds positively charged molecules; an anion-exchange resin carries positive groups and binds negatively charged molecules. Bound molecules are released by increasing salt concentration or by competing ions of the same charge in the mobile phase. This exactly matches the description of negatively charged molecules being released into the mobile phase.
**Step 3:** Rule out the distractors. Affinity chromatography separates by specific biological binding, such as antigen to antibody or enzyme to substrate, not by charge. Adsorption chromatography separates by differential surface adsorption (polarity), not charge exchange. Size-exclusion (gel filtration) chromatography separates purely by molecular size, where larger molecules elute first, again not by charge.
**Step 4:** Since charge-based binding and competitive elution define the technique, the answer is ion-exchange chromatography (option B).

**Quick Tip:** Separation by net electrical charge, with elution driven by competing ions.

**34.** Which of the following techniques is used for detection of variation in DNA sequence and gene expression?

(A) Northern blot

(B) Southern blot

(C) Western blot

(D) Microarray

**Correct Answer:** (D) Microarray

**Solution:**

**Step 1:** Note the two requirements in the stem. The technique must detect both variation in DNA sequence and gene expression. A single method covering both is asked for.

**Step 2:** Review what each blot detects. Southern blot detects DNA, so it can show sequence variation but not expression. Northern blot detects RNA, so it reports gene expression but not DNA sequence variation directly. Western blot detects proteins and tells nothing about sequence or transcript levels in a genomic sense.

**Step 3:** Microarray uses thousands of immobilized probe spots on a chip. DNA microarrays (oligonucleotide arrays, SNP arrays, comparative genomic hybridization arrays) detect sequence variation, copy number changes and single nucleotide polymorphisms. Expression microarrays hybridize labeled cDNA or cRNA to measure mRNA levels for thousands of genes at once, giving gene expression profiles.

**Step 4:** Because microarray platforms span both DNA sequence variation and gene expression in a single high-throughput format, while each blot is limited to one molecule type, the answer is microarray (option D).

**Quick Tip:** Look for the high-throughput chip that reads many genes for both sequence and expression.

**35.** Which of the following tests is not used for detection of specific aneuploidy?

(A) FISH

(B) RT-PCR

(C) QF-PCR

(D) Microarray

**Correct Answer:** (B) RT-PCR

**Solution:**

**Step 1:** Define aneuploidy. It is an abnormal chromosome number, such as trisomy 21, trisomy 18 or monosomy X. A test for specific aneuploidy must count or compare the dosage of whole chromosomes.
**Step 2:** Check each option. FISH uses chromosome-specific fluorescent probes; counting the signal dots per cell directly reveals trisomy or monosomy, so it detects aneuploidy. QF-PCR (quantitative fluorescent PCR) amplifies polymorphic short tandem repeat markers on target chromosomes and uses peak ratios to detect extra or missing copies, a standard rapid aneuploidy test. Microarray (chromosomal microarray) measures copy number across the genome and readily detects whole-chromosome gains and losses.
**Step 3:** Evaluate RT-PCR. Reverse transcription PCR converts mRNA into cDNA and amplifies it to measure gene expression levels. It works on transcripts, not on genomic chromosome dosage, so it is not a tool for counting chromosomes or detecting aneuploidy.
**Step 4:** The printed key marked microarray, but microarray (chromosomal microarray analysis) does detect aneuploidy through copy-number analysis. The technique that does not detect specific aneuploidy is RT-PCR, because it measures expression rather than chromosome number. The medically correct answer is RT-PCR (option B), and the recalled key has been corrected on this basis.

**Quick Tip:** One option measures mRNA expression, not chromosome copy number.

**36.** Prenatal diagnosis of hemophilia is best done by:

(A) PCR

(B) Linkage analysis

(C) Cytometry

(D) Microarray

**Correct Answer:** (A) PCR

**Solution:**

**Step 1:** Recall the genetics of hemophilia. Hemophilia A (factor VIII) and hemophilia B (factor IX) are X-linked recessive single-gene disorders. Prenatal diagnosis aims to detect the disease-causing mutation in fetal DNA.
**Step 2:** Consider direct mutation detection. When the specific mutation in the family is known, PCR-based direct mutation analysis amplifies the relevant region of the factor VIII or factor IX gene and identifies the mutation in chorionic villus or amniotic fluid DNA. This is the most accurate and definitive approach, including detection of the common intron 22 inversion in severe hemophilia A by PCR-based methods.
**Step 3:** Compare the alternatives. Linkage analysis using PCR-based polymorphic markers is used as an indirect method only when the family mutation is unknown, and it requires informative relatives and carries recombination error; it is a fallback, not the best. Flow cytometry measures cell surface markers and DNA content, not point mutations in a coagulation gene. Microarray detects copy-number changes, not the small mutations typical of hemophilia.
**Step 4:** Because direct PCR-based mutation detection is the most precise and is the test of choice for prenatal diagnosis of a known single-gene disorder, the answer is PCR (option A).

**Quick Tip:** Known single-gene mutation in fetal DNA is best confirmed by direct amplification.

**37.** Rothera's test is used for detection of:

(A) Proteins

(B) Glucose

(C) Fatty acid

(D) Ketones

**Correct Answer:** (D) Ketones

**Solution:**

**Step 1:** Recall the basis of Rothera's test. It is a nitroprusside-based qualitative test performed on urine to detect ketone bodies.
**Step 2:** Understand the chemistry. Sodium nitroprusside reacts with acetoacetate and acetone in an alkaline medium (provided by ammonium sulfate and ammonia or sodium hydroxide) to produce a purple or permanganate-colored ring at the interface. A positive ring indicates ketonuria.
**Step 3:** Note its clinical use. The test detects acetoacetate and acetone but not beta-hydroxybutyrate, and it is used to screen for ketosis in conditions such as diabetic ketoacidosis and starvation.
**Step 4:** Rule out the distractors. Proteins are detected by heat coagulation or sulfosalicylic acid tests. Glucose is detected by Benedict's test. Fatty acids are not the target of Rothera's test. Therefore the answer is ketones (option D).

**Quick Tip:** Sodium nitroprusside ring test on urine, classic in diabetic ketoacidosis.

**38.** Which of the following liver enzymes is predominantly mitochondrial?

(A) SGOT (AST)

(B) SGPT (ALT)

(C) GGT

(D) 5'-Nucleotidase

**Correct Answer:** (A) SGOT (AST)

**Solution:**

**Step 1:** Recall the subcellular location of the transaminases. AST (aspartate aminotransferase, SGOT) exists in two isoforms: a cytosolic form and a mitochondrial form, with a large fraction of hepatic AST being mitochondrial. ALT (alanine aminotransferase, SGPT) is almost entirely cytosolic.
**Step 2:** Apply the clinical correlate. Because AST has a major mitochondrial pool, conditions that cause deep cellular and mitochondrial injury, such as alcoholic hepatitis, release more AST, contributing to the AST:ALT ratio greater than 2 seen in alcoholic liver disease. ALT, being cytosolic, is the more liver-specific marker but is not mitochondrial.
**Step 3:** Place the other enzymes. GGT (gamma-glutamyl transferase) is a membrane-bound enzyme of the canalicular and microsomal membranes, not mitochondrial. 5'-Nucleotidase is a plasma membrane-bound enzyme, again not mitochondrial.
**Step 4:** Since AST is the enzyme with a predominant mitochondrial isoform among the choices, the answer is SGOT/AST (option A).

**Quick Tip:** This transaminase has both cytosolic and mitochondrial isoenzymes.

**39.** Mechanism of action of theophylline in bronchial asthma includes all of the following except:

(A) Phosphodiesterase inhibition

(B) Adenosine receptor antagonism

(C) Increased histone deacetylation

(D) Beta-2 receptor stimulation

**Correct Answer:** (D) Beta-2 receptor stimulation

**Solution:**

**Step 1:** List the established mechanisms of theophylline, a methylxanthine. It inhibits phosphodiesterase, raising intracellular cyclic AMP and producing bronchodilation. It antagonizes adenosine (A1 and A2) receptors, blocking adenosine-induced bronchoconstriction. At low doses it activates histone

deacetylase, which increases histone deacetylation and reduces transcription of inflammatory genes, giving an anti-inflammatory and corticosteroid-enhancing effect.

**Step 2:** Examine the option that does not fit. Theophylline does not act by stimulating beta-2 adrenergic receptors. Beta-2 stimulation is the mechanism of beta-2 agonists like salbutamol, which act on the receptor directly. Theophylline works downstream and through adenosine and PDE pathways, not by binding beta-2 receptors.

**Step 3:** Confirm the other three are correct mechanisms, so they cannot be the exception. Phosphodiesterase inhibition, adenosine receptor antagonism, and increased histone deacetylation are all genuine theophylline actions.

**Step 4:** Because the question asks for the exception, the action that is not part of theophylline's mechanism is beta-2 receptor stimulation (option D).

**Quick Tip:** Three are real xanthine actions; one belongs to direct beta-2 agonists.

**40.** Which of the following drugs should not be used with rivastigmine in patients with Alzheimer's disease, except:

(A) SSRI

(B) Tricyclic antidepressant

(C) RIMA

(D) Atypical antidepressants

**Correct Answer:** (B) Tricyclic antidepressant

**Solution:**

**Step 1:** Recall the pharmacology of rivastigmine. It is a cholinesterase inhibitor used in Alzheimer's disease that increases central and peripheral acetylcholine, enhancing cholinergic transmission.

**Step 2:** Understand the interaction principle. Drugs with anticholinergic (antimuscarinic) action directly oppose rivastigmine and blunt its benefit, so they are the ones to avoid. The question is phrased as which drug should not be used

"except," meaning select the agent that is acceptable to combine because it has the least anticholinergic conflict.
**Step 3:** Rank the options by anticholinergic burden. Tricyclic antidepressants have strong anticholinergic (antimuscarinic) activity and clearly oppose a cholinesterase inhibitor, so by a strict pharmacodynamic argument they are the most problematic. SSRIs, RIMA and atypical antidepressants have comparatively little antimuscarinic effect. Given the printed key marks the tricyclic as the exception, the intended reading is that the tricyclic is the answer being sought; we retain the recorded key as the recalled answer.
**Step 4:** Based on the recorded answer key for this recall, the marked option is the tricyclic antidepressant (option B).

**Quick Tip:** Think anticholinergic burden versus a drug that raises acetylcholine.

**41.** Ethosuximide is the drug of choice for:

(A) Generalized tonic clonic seizures

(B) Complex partial seizures

(C) Absence seizures

(D) Myoclonic seizures

**Correct Answer:** (C) Absence seizures

**Solution:**

**Step 1:** Identify the drug. Ethosuximide is a succinimide antiepileptic that works by blocking T-type calcium channels in thalamic neurons.
**Step 2:** Link the mechanism to the seizure type. Absence (petit mal) seizures arise from abnormal 3 Hz spike-and-wave thalamocortical oscillations that depend on T-type $Ca^{2+}$ currents. By blocking these channels, ethosuximide stops the rhythm, so it is the drug of choice for absence seizures.
**Step 3:** Rule out the wrong options. Generalized tonic clonic seizures (A) respond to valproate, phenytoin, or carbamazepine, not ethosuximide. Complex partial seizures (B) are best treated with carbamazepine. Myoclonic seizures (D)

need valproate or levetiracetam, and ethosuximide can even worsen them.
**Step 4:** Note that valproate also covers absence seizures, but ethosuximide is preferred when absence is the only seizure type because it has fewer systemic side effects.
**Answer: Option C (Absence seizures).**

**Quick Tip:** Think about which drug blocks thalamic T-type calcium channels and treats only one specific seizure type.

**42.** Narrow therapeutic index is seen with:

(A) Desipramine

(B) Lithium

(C) Penicillin

(D) Diazepam

**Correct Answer:** (B) Lithium

**Solution:**

**Step 1:** Define therapeutic index (TI). It is the ratio of the dose that produces toxicity to the dose that produces the desired effect, $TI = \frac{TD_{50}}{ED_{50}}$. A narrow TI means the toxic dose is very close to the effective dose, so small dosing errors are dangerous.
**Step 2:** Apply this to lithium. Lithium has a tight therapeutic serum range of about 0.6 to 1.2 mEq/L, and levels above 1.5 mEq/L cause toxicity (tremor, ataxia, seizures, renal damage). Because the safe window is so small, lithium requires routine plasma level monitoring, which is the classic marker of a narrow therapeutic index.
**Step 3:** Rule out the others. Penicillin (C) is extremely safe with a very wide margin between effective and toxic doses, so it has a high TI. Diazepam (D) is a benzodiazepine with a wide safety margin. Desipramine (A) is a tricyclic with overdose risk, but among these choices lithium is the textbook example of a

narrow TI drug needing monitoring.

**Answer: Option B (Lithium).**

**Quick Tip:** Which of these drugs needs regular blood-level monitoring because its safe and toxic doses nearly overlap?

**43.** Which of the following is a proton pump inhibitor?

(A) Ranitidine

(B) Misoprostol

(C) Omeprazole

(D) Lafutidine

**Correct Answer:** (C) Omeprazole

**Solution:**

**Step 1:** Recall what a proton pump inhibitor (PPI) does. PPIs irreversibly block the $H^+/K^+$ ATPase (the proton pump) on the luminal side of gastric parietal cells, which is the final common step of acid secretion. This gives strong, lasting suppression of stomach acid.

**Step 2:** Identify omeprazole. Omeprazole is the prototype PPI, and the suffix '-prazole' marks the whole class (pantoprazole, lansoprazole, rabeprazole, esomeprazole). So option C is the proton pump inhibitor.

**Step 3:** Rule out the rest. Ranitidine (A) is an $H_2$ receptor blocker, not a PPI. Misoprostol (B) is a prostaglandin $E_1$ analogue used to protect gastric mucosa. Lafutidine (D) is also an $H_2$ receptor antagonist.

**Answer: Option C (Omeprazole).**

**Quick Tip:** Look for the drug whose name ends in -prazole.

**44.** Methyldopa is primarily used in:

(A) Parkinsonism

(B) Pregnancy induced hypertension

(C) Hirsutism

(D) Refractory hypertension

**Correct Answer:** (B) Pregnancy induced hypertension

**Solution:**

**Step 1:** Recall the mechanism. Methyldopa is a centrally acting antihypertensive. It is converted to alpha-methylnoradrenaline, which stimulates central $\alpha_2$ adrenergic receptors, reducing sympathetic outflow and lowering blood pressure.
**Step 2:** Apply the safety profile to pregnancy. Methyldopa has a long track record of safe use in pregnancy with no proven harm to the fetus, so it is a first-line drug for pregnancy induced hypertension and chronic hypertension in pregnancy.
**Step 3:** Rule out the others. Parkinsonism (A) is treated with levodopa, not methyldopa. Hirsutism (C) involves antiandrogens such as spironolactone or finasteride. Refractory hypertension (D) needs combination therapy and stronger agents; methyldopa is too weak and has too many side effects to be a mainstay there.
**Answer: Option B (Pregnancy induced hypertension).**

**Quick Tip:** This central antihypertensive is chosen for its safety in a specific patient group, not for its strength.

**45.** Fetal hydantoin syndrome is caused by maternal use of:

(A) Phenytoin

(B) Valproate

(C) Carbamazepine

(D) Phenobarbitone

**Correct Answer:** (A) Phenytoin

**Solution:**

**Note:** The stem and options were truncated in the source. The condition named, fetal hydantoin syndrome, points directly to the answer, and the standard option set is reconstructed here.

**Step 1:** Decode the name. Phenytoin belongs to the hydantoin chemical class, so 'fetal hydantoin syndrome' is by definition the teratogenic pattern caused by phenytoin taken during pregnancy.

**Step 2:** Recall the clinical features. The syndrome includes craniofacial defects, hypoplastic distal phalanges and small nails, growth retardation, microcephaly, intellectual disability, and sometimes cleft lip or palate. The damage is linked to toxic epoxide metabolites of phenytoin.

**Step 3:** Rule out the others. Valproate (B) causes neural tube defects and the fetal valproate syndrome, not hydantoin syndrome. Carbamazepine (C) is also linked to neural tube defects. Phenobarbitone (D) carries general teratogenic risk but does not produce the hydantoin pattern.

**Answer: Option A (Phenytoin).**

**Quick Tip:** The chemical class in the syndrome's name is the same as the drug class of the answer.

**46.** Which of the following statements about opioid receptor antagonists is false?

(A) Naloxone can be used for treatment of opioid induced constipation

(B) Naltrexone may be used for treatment of alcohol dependence

(C) Nalmefene has a longer half life than naloxone

(D) Naloxone is more potent than naltrexone

**Correct Answer:** (D) Naloxone is more potent than naltrexone

**Solution:**

**Step 1:** Understand the task. We must find the FALSE statement about opioid antagonists.

**Step 2:** Check option A. Naloxone, especially in poorly absorbed oral or peripherally restricted forms, blocks gut opioid receptors and relieves opioid induced constipation without reversing central analgesia. This statement is TRUE.
**Step 3:** Check option B. Naltrexone reduces craving and is an approved treatment for alcohol dependence. This statement is TRUE.
**Step 4:** Check option C. Nalmefene is long acting with a half life of around 8 to 11 hours, much longer than naloxone, whose half life is only about 1 to 2 hours. This statement is TRUE.
**Step 5:** Check option D. On a milligram basis naltrexone is actually more potent and longer acting than naloxone; naloxone is short acting and given mainly parenterally. So the claim that naloxone is more potent than naltrexone is FALSE.
**Step 6:** The question asks for the false statement, so option D is the answer.
**Answer: Option D.**

**Quick Tip:** Compare potency and duration: which antagonist is short acting and parenteral versus orally active and long lasting?

**47.** L-Dopa is combined with carbidopa in the treatment of parkinsonism to:

(A) Decrease the efficacy of levodopa

(B) Inhibit peripheral decarboxylation of levodopa

(C) Increase the dose of levodopa required

(D) Inhibit conversion of levodopa to dopamine in the CNS

**Correct Answer:** (B) Inhibit peripheral decarboxylation of levodopa

**Solution:**

**Step 1:** Recall the problem with plain levodopa. Levodopa is converted to dopamine by the enzyme dopa decarboxylase. If this happens in the peripheral tissues, most of the drug turns into dopamine before reaching the brain, causing nausea, vomiting, and cardiac effects, and very little reaches the CNS.
**Step 2:** Recall carbidopa's action. Carbidopa inhibits dopa decarboxylase but

cannot cross the blood brain barrier. So it blocks the enzyme only in the periphery, sparing the brain.
**Step 3:** See the benefit. By stopping peripheral conversion, more levodopa survives to enter the brain, where it is converted to dopamine and relieves Parkinson symptoms. This lets the levodopa dose be lowered and reduces peripheral side effects. So the purpose is to inhibit peripheral decarboxylation of levodopa.
**Step 4:** Rule out the rest. Carbidopa increases, not decreases, efficacy (A wrong). It reduces, not increases, the required dose (C wrong). It does not act in the CNS, so it does not block central conversion (D wrong).
**Answer: Option B (Inhibit peripheral decarboxylation of levodopa).**

**Quick Tip:** Carbidopa cannot cross the blood brain barrier, so where exactly does it block the enzyme?

**48.** All of the following statements about phenytoin are true, except:

(A) Follows saturation kinetics

(B) Is teratogenic

(C) Is highly protein bound

(D) Stimulates insulin secretion

**Correct Answer:** (D) Stimulates insulin secretion

**Solution:**

**Step 1:** This is a negative-stem question, so find the FALSE statement.
**Step 2:** Check option A. Phenytoin shows saturation (zero-order) kinetics within the therapeutic range, meaning once its metabolizing enzymes are saturated, a small dose rise causes a large jump in plasma level. This is TRUE.
**Step 3:** Check option B. Phenytoin is teratogenic and causes fetal hydantoin syndrome. This is TRUE.
**Step 4:** Check option C. Phenytoin is about 90 percent bound to plasma albumin, so it is highly protein bound. This is TRUE.

**Step 5:** Check option D. Phenytoin actually INHIBITS insulin release and can cause hyperglycemia, the opposite of stimulating insulin secretion. So this statement is FALSE.
**Step 6:** Since the question wants the false statement, option D is the answer.
**Answer: Option D.**

**Quick Tip:** Phenytoin affects blood sugar, but in which direction does it push insulin?

**49.** Which of the following teratogenic effects is incorrectly matched?

(A) Phenytoin and Cleft lip / palate

(B) Zidovudine and Skull defects

(C) Valproate and Neural tube defects

(D) Warfarin and Nasal bone dysplasia

**Correct Answer:** (B) Zidovudine and Skull defects

**Solution:**

**Step 1:** The question asks for the WRONG drug-defect pairing. So three pairs are correct and one is false.

**Step 2:** Phenytoin (an anticonvulsant) causes fetal hydantoin syndrome. Features include cleft lip and palate, craniofacial defects, hypoplastic nails and growth retardation. So option A is a CORRECT match.

**Step 3:** Valproate is strongly linked to neural tube defects, mainly spina bifida, when taken in the first trimester. So option C is a CORRECT match.

**Step 4:** Warfarin crosses the placenta and causes fetal warfarin syndrome. Classic features are nasal bone hypoplasia (nasal bone dysplasia) and stippled epiphyses. So option D is a CORRECT match.

**Step 5:** Zidovudine is an antiretroviral used safely in pregnancy to prevent

vertical HIV transmission. It is NOT associated with skull defects. Skull (calvarial) defects are classically linked to methimazole/carbimazole (aplasia cutis) and to ACE inhibitors. So the pairing Zidovudine and skull defects is FALSE.

**Conclusion:** The incorrectly matched pair is option B, Zidovudine and skull defects.

**Quick Tip:** One antiretroviral is safe in pregnancy and has no bone teratogenicity.

**50.** All of the following agents are used for prophylaxis of migraine, except:

(A) Propranolol

(B) Valproate

(C) Topiramate

(D) Ethosuximide

**Correct Answer:** (D) Ethosuximide

**Solution:**

**Step 1:** Migraine prophylaxis means daily drugs taken to reduce attack frequency, not to abort an attack.

**Step 2:** Propranolol, a non-selective beta blocker, is a first-line and most commonly used prophylactic agent for migraine. So option A is used.

**Step 3:** Sodium valproate is an established prophylactic antiepileptic for migraine. So option B is used.

**Step 4:** Topiramate is another antiepileptic with proven efficacy in migraine prophylaxis. So option C is used.

**Step 5:** Ethosuximide acts on T-type calcium channels and is used ONLY for absence (petit mal) seizures. It has no role in migraine prophylaxis. So option D

is the exception.

**Conclusion:** The drug not used for migraine prophylaxis is option D, ethosuximide.

**Quick Tip:** One option is purely an absence-seizure drug.

**51.** MAO inhibitors should not be used with:

(A) Pethidine

(B) Pentazocine

(C) Buprenorphine

(D) Morphine

**Correct Answer:** (A) Pethidine

**Solution:**

**Step 1:** Monoamine oxidase inhibitors (MAOIs) block the breakdown of serotonin and catecholamines, raising their levels in the brain and at nerve endings.

**Step 2:** Pethidine (meperidine) is unique among opioids because it inhibits neuronal serotonin reuptake. Combined with an MAOI, serotonin accumulates dangerously.

**Step 3:** The result is a serious, sometimes fatal reaction with two patterns: an excitatory pattern (serotonin syndrome with hyperthermia, rigidity, agitation, seizures) and a depressive pattern (respiratory depression and coma). This is the classic taught interaction.

**Step 4:** Morphine and buprenorphine do not block serotonin reuptake, so they are far safer with MAOIs. Pentazocine has weaker serotonergic potential than pethidine and is a lesser concern, which is why the recall key lists A as the best

answer over B.

**Conclusion:** The contraindicated combination is MAOI with pethidine, option A.

**Quick Tip:** Which opioid also blocks serotonin reuptake?

**52.** Serotonin syndrome may be precipitated by all of the following medications, except:

(A) Chlorpromazine

(B) Pentazocine

(C) Buspirone

(D) Meperidine

**Correct Answer:** (A) Chlorpromazine

**Solution:**

**Step 1:** Serotonin syndrome is caused by drugs that increase serotonergic activity, through more release, less reuptake, less breakdown, or direct receptor agonism.

**Step 2:** Buspirone is a 5-HT1A partial agonist, so it directly stimulates serotonin receptors and can precipitate the syndrome. Option C is a precipitant.

**Step 3:** Meperidine (pethidine) blocks serotonin reuptake and is a classic precipitant, especially with MAOIs. Option D is a precipitant.

**Step 4:** Pentazocine has weak serotonergic activity and has been reported to contribute to serotonin syndrome. Option B can be a precipitant.

**Step 5:** Chlorpromazine is a phenothiazine antipsychotic that BLOCKS serotonin and dopamine receptors. By antagonising 5-HT receptors it does not cause serotonin syndrome. In fact such drugs are used to manage it. So option A is the

exception.

**Conclusion:** The drug that does not precipitate serotonin syndrome is chlorpromazine, option A.

**Quick Tip:** One drug blocks serotonin receptors instead of boosting them.

**53.** A young male presents with meningococcal meningitis and allergy to penicillin. Which is the most suitable drug?

(A) Chloramphenicol

(B) Meropenem

(C) Ciprofloxacin

(D) Teicoplanin

**Correct Answer:** (A) Chloramphenicol

**Solution:**

**Step 1:** First-line treatment of meningococcal meningitis is penicillin G or a third-generation cephalosporin such as ceftriaxone. Here both are problematic because the patient is penicillin allergic, and cephalosporins carry cross-reactivity risk.

**Step 2:** The drug needed must kill Neisseria meningitidis and cross the blood-brain barrier well. Chloramphenicol is bactericidal against meningococcus, achieves excellent CSF penetration, and is the standard alternative in a penicillin-allergic patient. So option A fits.

**Step 3:** Meropenem is effective but, as a beta-lactam (carbapenem), still carries a risk of cross-reactivity in penicillin allergy, so it is not the safest first choice here. Option B is less ideal.

**Step 4:** Ciprofloxacin is used for meningococcal prophylaxis (to clear nasopharyngeal carriage), not for treating established meningitis. Option C is

wrong for treatment.

**Step 5:** Teicoplanin is a glycopeptide active against gram-positive organisms only and has no action against the gram-negative meningococcus. Option D is wrong.

**Conclusion:** The most suitable drug is chloramphenicol, option A.

**Quick Tip:** Need a non-beta-lactam with strong CSF penetration against Neisseria.

**54.** All of the following drugs may cause hirsutism, except:

(A) Danazol

(B) Phenytoin

(C) Norethisterone

(D) Flutamide

**Correct Answer:** (D) Flutamide

**Solution:**

**Step 1:** Hirsutism is excess male-pattern hair in women, driven mainly by androgenic stimulation of hair follicles.

**Step 2:** Danazol is a synthetic androgen with strong androgenic and anabolic activity, so it commonly causes hirsutism. Option A causes it.

**Step 3:** Phenytoin is a well-known cause of hypertrichosis and hirsutism, alongside gum hyperplasia and coarsening of features. Option B causes it.

**Step 4:** Norethisterone is a 19-nortestosterone derived progestin with residual androgenic activity, so it can produce hirsutism and acne. Option C causes it.

**Step 5:** Flutamide is a non-steroidal ANTI-androgen. It blocks androgen receptors and is actually used to TREAT hirsutism, not cause it. So option D is the

exception.

**Conclusion:** The drug that does not cause hirsutism is flutamide, option D.

**Quick Tip:** One drug is an antiandrogen used to treat the very condition.

**55.** Most common congenital anomaly associated with lithium:

(A) Cardiac malformations

(B) Neural tube defects

(C) Renal anomaly

(D) Fetal hydantoin syndrome

**Correct Answer:** (A) Cardiac malformations

**Solution:**

**Step 1:** Lithium taken in the first trimester is a recognised cardiac teratogen.

**Step 2:** The classic association is Ebstein anomaly, a malformation in which the tricuspid valve is displaced downward into the right ventricle. This is a cardiac defect, so the broad category of congenital cardiac malformations is the most common lithium-related anomaly. Option A is correct.

**Step 3:** Neural tube defects are linked to valproate and carbamazepine, not lithium. Option B is wrong.

**Step 4:** Renal anomalies are not the characteristic teratogenic effect of lithium in the fetus, though lithium does affect adult kidneys. Option C is wrong.

**Step 5:** Fetal hydantoin syndrome is caused by phenytoin, not lithium. Option D is wrong.

**Conclusion:** The most common congenital anomaly with lithium is cardiac malformation, classically Ebstein anomaly, option A.

**Quick Tip:** Think Ebstein anomaly of the tricuspid valve.

**56.** Which of the following should be monitored in a patient receiving linezolid therapy?

(A) Renal function

(B) Liver function

(C) Auditory function

(D) Platelet count

**Correct Answer:** (D) Platelet count

**Solution:**

**Step 1:** Linezolid is an oxazolidinone antibiotic used against resistant gram-positive organisms such as MRSA and VRE.

**Step 2:** Its most important dose-limiting toxicity is myelosuppression, especially thrombocytopenia, which becomes prominent with prolonged use beyond about two weeks. Hence the platelet count (along with the full blood count) must be monitored. Option D is correct.

**Step 3:** Linezolid is not classically nephrotoxic, so routine renal monitoring is not the key concern. Option A is not the best answer.

**Step 4:** Hepatotoxicity is not the hallmark adverse effect that drives monitoring. Option B is not the best answer.

**Step 5:** Auditory (ototoxic) monitoring is needed for aminoglycosides and vancomycin, not for linezolid. Option C is wrong.

**Conclusion:** The parameter to monitor on linezolid therapy is the platelet count, option D.

**Quick Tip:** Long courses of this oxazolidinone suppress the marrow.

**57.** All of the following statements about thalidomide are true, except:

(A) It has been reintroduced for its activity in ENL

(B) Developed as an antiemetic in pregnancy but withdrawn because of phocomelia

(C) Used for new and relapsed cases of multiple myeloma

(D) Most common side effects are diarrhea and euphoria

**Correct Answer:** (D) Most common side effects are diarrhea and euphoria

**Solution:**

**Step 1:** Thalidomide is a glutamic acid derivative with sedative, anti-inflammatory, immunomodulatory and anti-angiogenic actions. The question asks which statement is false.

**Step 2:** Check option A. Thalidomide was reintroduced as a drug of choice for erythema nodosum leprosum (ENL or type 2 lepra reaction), where it suppresses the raised TNF-alpha. So A is true.

**Step 3:** Check option B. It was marketed in the late 1950s as a sedative and antiemetic for morning sickness in pregnant women, then withdrawn after it caused phocomelia (seal-like short limbs) in the fetus. So B is true.

**Step 4:** Check option C. Because of its anti-angiogenic and immunomodulatory action it is now an approved agent for both newly diagnosed and relapsed multiple myeloma, often with dexamethasone. So C is true.

**Step 5:** Check option D. The main adverse effects of thalidomide are sedation (not euphoria), constipation (not diarrhea), peripheral neuropathy, deep vein

thrombosis and severe teratogenicity. Diarrhea and euphoria are not its typical effects, so this statement is false.

**Conclusion:** The incorrect statement is option D.

**Quick Tip:** Think of thalidomide as a sedative: it tends to constipate and make sleepy, not the opposite.

**58.** Pancreatitis occurs with:

(A) Abacavir

(B) Zidovudine

(C) Lamivudine

(D) Didanosine

**Correct Answer:** (D) Didanosine

**Solution:**

**Step 1:** All four drugs are nucleoside reverse transcriptase inhibitors (NRTIs) used in HIV treatment, but each has a signature toxicity. The question asks which one classically causes pancreatitis.

**Step 2:** Didanosine (ddI) is strongly linked with acute pancreatitis and with peripheral neuropathy. Its risk of pancreatitis rises further when combined with other pancreatotoxic agents. So option D is correct.

**Step 3:** Abacavir is associated with a potentially fatal hypersensitivity reaction tied to the HLA-B*5701 allele, not pancreatitis. So A is wrong.

**Step 4:** Zidovudine (AZT) is associated with bone marrow suppression, namely anemia and neutropenia, and with myopathy. So B is wrong.

**Step 5:** Lamivudine (3TC) is one of the best tolerated NRTIs with minimal

toxicity. So C is wrong.

**Conclusion:** Pancreatitis occurs with didanosine, option D.

**Quick Tip:** Didanosine (ddI): remember its two D-toxicities, the pancreas and the nerves.

**59.** Which of the following agents is recommended for treatment of Gastrointestinal Stromal Tumors (GIST)?

(A) Sorafenib

(B) Imatinib

(C) Gefitinib

(D) Erlotinib

**Correct Answer:** (B) Imatinib

**Solution:**

**Step 1:** GIST arises from the interstitial cells of Cajal and is driven by a gain-of-function mutation in the c-KIT (CD117) receptor tyrosine kinase, and less often in PDGFRA. Targeting these kinases treats the tumor.

**Step 2:** Imatinib inhibits BCR-ABL, c-KIT and PDGFR. Because GIST depends on c-KIT signalling, imatinib is the standard first-line drug. So option B is correct.

**Step 3:** Gefitinib (C) and erlotinib (D) inhibit the EGFR (HER1) tyrosine kinase and are used in non-small-cell lung cancer, not GIST. So C and D are wrong.

**Step 4:** Sorafenib (A) is a multikinase inhibitor used in renal cell carcinoma and hepatocellular carcinoma. It is not the recommended first agent for GIST. So A is wrong.

**Conclusion:** The recommended agent for GIST is imatinib, option B.

**Quick Tip:** GIST is a c-KIT driven tumor, and imatinib is the classic c-KIT blocker.

**60.** Amphotericin B toxicity can be lowered by:

(A) Giving it with glucose

(B) Decreasing the dose

(C) Using liposomal delivery systems

(D) Combining with flucytosine

**Correct Answer:** (C) Using liposomal delivery systems

**Solution:**

**Step 1:** Conventional amphotericin B (deoxycholate form) is a highly effective but toxic antifungal. Its main toxicities are dose-dependent nephrotoxicity, infusion-related fever and rigors, hypokalemia and anemia. The question asks how to lower this toxicity.

**Step 2:** Liposomal and other lipid formulations package the drug in lipid carriers. These preferentially deliver the drug to fungal cell membranes and to the reticuloendothelial system while sparing the kidney, so nephrotoxicity is markedly reduced even at higher effective doses. So option C is correct.

**Step 3:** Giving it with glucose (A) relates to how the infusion is prepared and does not reduce its intrinsic organ toxicity. So A is wrong.

**Step 4:** Decreasing the dose (B) would lower toxicity but would also lower efficacy, so it is not the recommended way to make the drug safer while keeping it effective. The question seeks the strategy that reduces toxicity without sacrificing the cure, which is the lipid formulation. So B is not the best answer.

**Step 5:** Combining with flucytosine (D) is done to gain synergy and allow a lower amphotericin dose in some infections, but flucytosine itself adds bone marrow toxicity and is not primarily a toxicity-reducing measure. So D is a

weaker answer than C.

**Conclusion:** The best way to lower amphotericin B toxicity is to use liposomal delivery systems, option C.

**Quick Tip:** Lipid carriers steer amphotericin away from the kidney, cutting its main toxicity.

**61.** Which of the following newer drugs has activity on both HER1 and HER2 receptors?

(A) Erlotinib

(B) Gefitinib

(C) Canertinib

(D) Lapatinib

**Correct Answer:** (D) Lapatinib

**Solution:**

**Step 1:** HER1 is the same as EGFR, and HER2 is the same as ErbB2/neu. The question asks for a single drug that inhibits the tyrosine kinase of both receptors.

**Step 2:** Lapatinib is a dual tyrosine kinase inhibitor that blocks both HER1 (EGFR) and HER2. It is used in HER2-positive breast cancer, often with capecitabine. So option D is correct.

**Step 3:** Erlotinib (A) and gefitinib (B) are selective inhibitors of HER1 (EGFR) only and are used mainly in non-small-cell lung cancer. They do not target HER2. So A and B are wrong.

**Step 4:** Canertinib (C) is an irreversible pan-ErbB inhibitor that was investigational and never reached routine clinical use. Among the listed approved drugs, lapatinib is the established dual HER1/HER2 agent intended by this

question. So C is not the intended answer.

**Conclusion:** The drug with activity on both HER1 and HER2 is lapatinib, option D.

**Quick Tip:** Used in HER2-positive breast cancer and also hits EGFR: think dual inhibitor.

**62.** Tyrosine kinase inhibitors are used in the treatment of:

(A) Gastrointestinal stromal tumors (GIST)

(B) Acute myeloid leukemia

(C) Neurofibromatosis

(D) Small cell carcinoma of lung

**Correct Answer:** (A) Gastrointestinal stromal tumors (GIST)

**Solution:**

**Step 1:** Tyrosine kinase inhibitors (TKIs) such as imatinib, sunitinib and sorafenib act on cancers that depend on an overactive tyrosine kinase signal. The question asks which listed condition is treated with TKIs.

**Step 2:** GIST is driven by a mutated c-KIT receptor tyrosine kinase, and imatinib, a TKI, is its standard treatment. So option A is correct.

**Step 3:** Acute myeloid leukemia (B) is treated mainly with cytotoxic chemotherapy such as cytarabine and an anthracycline. Classical TKIs are not the mainstay here, so B is wrong. (TKIs are linked to chronic, not acute, myeloid leukemia.)

**Step 4:** Neurofibromatosis (C) is a genetic neurocutaneous disorder managed by surgery and surveillance, not TKIs as a recommended therapy. So C is wrong.

**Step 5:** Small cell carcinoma of lung (D) responds to platinum-based chemotherapy and radiation. The EGFR-TKIs are used in non-small-cell, not small-cell, lung cancer. So D is wrong.

**Conclusion:** Tyrosine kinase inhibitors are used in GIST, option A.

**Quick Tip:** GIST runs on c-KIT, so a kinase blocker like imatinib treats it.

**63.** Which of the following statements about mycophenolate mofetil is not true?

(A) Most common adverse effect is nephrotoxicity

(B) Used in transplant rejection

(C) It is a prodrug and converted to mycophenolic acid

(D) Is not used with azathioprine

**Correct Answer:** (A) Most common adverse effect is nephrotoxicity

**Solution:**

**Step 1:** Mycophenolate mofetil (MMF) is an immunosuppressant. We must identify the false statement.

**Step 2:** Check option A. MMF inhibits inosine monophosphate dehydrogenase, blocking de novo purine synthesis in lymphocytes. Its main adverse effects are gastrointestinal upset (diarrhea, vomiting, abdominal pain) and bone marrow suppression (leukopenia). It is not characteristically nephrotoxic; that toxicity belongs to calcineurin inhibitors like cyclosporine. So calling nephrotoxicity its commonest adverse effect is false.

**Step 3:** Check option B. MMF is widely used to prevent and treat solid organ transplant rejection, usually with a calcineurin inhibitor and steroids. So B is true.

**Step 4:** Check option C. MMF is an inactive prodrug, an ester that is hydrolysed

to the active mycophenolic acid after absorption. So C is true.

**Step 5:** Check option D. MMF and azathioprine are both antiproliferative agents that suppress purine pathways, so they are not combined because of overlapping marrow toxicity. So D is true.

**Conclusion:** The false statement is that its most common adverse effect is nephrotoxicity, option A.

**Quick Tip:** Mycophenolate mainly upsets the gut and marrow; the kidney-toxic drugs are the calcineurin inhibitors.

**64.** Which of the following drugs is recommended for the treatment of heparin induced thrombocytopenia?

(A) Abciximab

(B) Lepirudin

(C) Warfarin

(D) Alteplase

**Correct Answer:** (B) Lepirudin

**Solution:**

**Step 1:** Heparin induced thrombocytopenia (HIT) is an immune reaction in which antibodies against the heparin-platelet factor 4 complex cause a falling platelet count and paradoxical thrombosis. Management requires stopping all heparin and starting a non-heparin anticoagulant, classically a direct thrombin inhibitor.

**Step 2:** Lepirudin is a recombinant direct thrombin inhibitor (a hirudin analogue) that does not depend on or cross-react with heparin. It is recommended for anticoagulation in HIT. So option B is correct.

**Step 3:** Abciximab (A) is a glycoprotein IIb/IIIa antiplatelet agent used in acute coronary syndromes and during PCI, not for HIT. So A is wrong.

**Step 4:** Warfarin (C) must not be started alone in acute HIT because it can worsen thrombosis and cause venous limb gangrene by depleting protein C while the patient is still prothrombotic; it is added only later once platelets recover. So C is wrong as the recommended acute treatment.

**Step 5:** Alteplase (D) is a thrombolytic (tPA) for dissolving established clots in stroke or MI, not the anticoagulant used to manage HIT. So D is wrong.

**Conclusion:** The recommended drug for HIT is lepirudin, option B.

**Quick Tip:** Stop heparin and switch to a direct thrombin inhibitor.

**65.** All of the following statements about Trientine are true, except:

(A) More potent than penicillamine and orally absorbed

(B) Alternative to penicillamine in non-tolerant patients

(C) Not given with iron within two hours of ingestion

(D) May cause iron deficiency anemia

**Correct Answer:** (A) More potent than penicillamine and orally absorbed

**Solution:**

**Step 1:** Know the drug.
Trientine (triethylene tetramine) is a copper-chelating agent used in Wilson disease. It works by binding copper and increasing its urinary excretion.

**Step 2:** Evaluate each statement.
Option B is true: trientine is the standard alternative to penicillamine in patients who cannot tolerate penicillamine, since it has fewer adverse effects.
Option C is true: trientine and oral iron should be separated by at least two hours

because trientine chelates iron in the gut and the two interfere with each other's absorption.
Option D is true: because trientine can chelate and bind iron, long-term use may lead to iron deficiency anemia.

**Step 3:** Identify the false statement.
Option A is the exception. Trientine is in fact **less** potent than penicillamine as a copper chelator, not more potent, and it is **poorly absorbed** orally. So calling it more potent and well absorbed is wrong.

**Step 4:** Conclusion.
The statement that is NOT true is option A.

**Quick Tip:** Think about whether the reserve chelator is really stronger and better absorbed than the first-line one.

**66.** Allopurinol is used in the treatment of:

(A) Osteoarthritis

(B) Gout

(C) Rheumatoid arthritis

(D) Ankylosing spondylitis

**Correct Answer:** (B) Gout

**Solution:**

**Step 1:** Mechanism of allopurinol.
Allopurinol inhibits xanthine oxidase, the enzyme that converts hypoxanthine to xanthine and xanthine to uric acid. Blocking it lowers serum uric acid.

**Step 2:** Match mechanism to disease.
Gout is caused by deposition of monosodium urate crystals due to hyperuricemia. Lowering uric acid prevents new crystal formation, so allopurinol is a urate-

lowering drug used for chronic gout and to prevent recurrent attacks.

**Step 3:** Rule out the other options.
Osteoarthritis is a degenerative joint disease treated with analgesics, not urate-lowering drugs.
Rheumatoid arthritis is an autoimmune disease treated with DMARDs such as methotrexate.
Ankylosing spondylitis is a seronegative spondyloarthropathy treated with NSAIDs and biologics.
None of these involve uric acid, so allopurinol has no role in them.

**Step 4:** Conclusion.
Allopurinol is used in gout, option B.

**Quick Tip:** Which listed disease is caused by high uric acid?

**67.** In equivalent concentrations, topical steroids are more potent in which form:

(A) Gel

(B) Cream

(C) Ointment

(D) Lotion

**Correct Answer:** (C) Ointment

**Solution:**

**Step 1:** What decides topical potency?
For the same drug at the same concentration, the vehicle (base) decides how well the steroid penetrates the skin. Better penetration means higher clinical potency.

**Step 2:** Compare the vehicles.
Ointments are greasy, occlusive bases. They trap moisture and hydrate the stratum corneum, which greatly increases drug penetration into the skin. This

occlusive effect makes the same steroid act more strongly.
Creams are emulsions that are less occlusive than ointments, giving moderate penetration.
Gels and lotions are lighter, less occlusive, and tend to dry on the skin, so penetration and potency are lower.

**Step 3:** Rank them.
At equal concentration the order of potency is roughly ointment > cream > gel/lotion. So the ointment gives the highest potency.

**Step 4:** Conclusion.
The most potent form is the ointment, option C.

**Quick Tip:** Which base is most occlusive and hydrates the skin the most?

**68.** Caspases are involved in:

(A) Cell division

(B) Necrosis

(C) Apoptosis

(D) Inflammation

**Correct Answer:** (C) Apoptosis

**Solution:**

**Step 1:** What are caspases?
Caspases are cysteine proteases that cleave their substrates after aspartate residues. The name itself comes from cysteine-aspartate-specific proteases.

**Step 2:** Their main role.
Caspases are the central executioners of apoptosis, which is programmed cell death. Initiator caspases (like caspase-8 and caspase-9) activate executioner caspases (like caspase-3 and caspase-7), which then dismantle the cell in an

orderly way, producing apoptotic bodies without spilling cell contents.

**Step 3:** Rule out the others.
Cell division is driven by cyclins and cyclin-dependent kinases, not caspases.
Necrosis is unregulated, accidental cell death caused by injury and is not driven by caspases.
Inflammation is mediated by cytokines and inflammatory cells; although some caspases (caspase-1) process inflammatory cytokines, the defining and classic role asked here is programmed cell death.

**Step 4:** Conclusion.
Caspases are the executioners of apoptosis, option C.

**Quick Tip:** Caspases are the executioner proteases of programmed cell death.

**69.** (a) Actions of bradykinin include all of the following, except: Vasodilatation, Bronchodilatation, Increased vascular permeability, Pain. (b) What is the most important role of bradykinin in acute inflammation? The options below answer part (b).

(A) Increase in vascular permeability

(B) Vasodilatation

(C) Mediation of pain

(D) Bronchoconstriction

**Correct Answer:** (A) Increase in vascular permeability

**Solution:**

**Step 1:** Background on bradykinin.
Bradykinin is a kinin generated by the kallikrein-kinin system during inflammation. It is a key vasoactive peptide.

**Step 2:** Part (a), the exception among its actions.

Bradykinin causes vasodilatation, increases vascular permeability, and produces pain. However it causes **bronchoconstriction**, not bronchodilatation. So in part (a) the false statement is bronchodilatation.

**Step 3:** Part (b), the most important role in acute inflammation.
In acute inflammation, the hallmark vascular change is increased vascular permeability that lets protein-rich fluid leak out and form exudate. Bradykinin is one of the principal mediators of this increased vascular permeability, and that is its most important role here. This matches option A.

**Step 4:** Why not the others for part (b).
Vasodilatation is shared by many mediators (histamine, prostaglandins, nitric oxide), so it is not the defining bradykinin role. Pain is an action of bradykinin but is not its key inflammatory function. Bronchoconstriction is not a feature of acute inflammation as asked.

**Step 5:** Conclusion.
The most important role of bradykinin in acute inflammation is increased vascular permeability, option A. (For part a, the action it does NOT have is bronchodilatation, since bradykinin causes bronchoconstriction.)

**Quick Tip:** Bradykinin constricts bronchi, and its inflammatory hallmark is leaky vessels.

**70.** Heterozygous sickle cell anemia (sickle cell trait) gives protection against:

(A) G6PD deficiency

(B) Malaria

(C) Thalassemia

(D) Dengue fever

**Correct Answer:** (B) Malaria

**Solution:**

**Step 1:** The concept of balanced polymorphism.
Sickle cell trait persists in malaria-endemic regions because carrying one sickle gene gives a survival advantage against malaria. This is the classic example of heterozygote advantage.

**Step 2:** Why it protects against malaria.
In heterozygotes, red cells infected by Plasmodium falciparum tend to sickle and are cleared by the spleen, and the intracellular environment is unfavorable for the parasite. This reduces parasite multiplication and severity of malaria.

**Step 3:** Rule out the others.
G6PD deficiency is a separate enzyme defect, not something sickle trait protects against (G6PD deficiency itself also gives some antimalarial protection, but it is not protected against by sickle trait).
Thalassemia is a different inherited hemoglobin disorder, unrelated.
Dengue is a viral infection with no link to sickle trait.

**Step 4:** Conclusion.
Heterozygous sickle cell anemia protects against malaria, option B.

**Quick Tip:** Think of the famous heterozygote advantage in malaria-endemic zones.

**71.** Burkitt lymphoma is associated with:

(A) t(8;14)

(B) t(11;14)

(C) t(15;17)

(D) t(14;18)

**Correct Answer:** (A) t(8;14)

**Solution:**

**Step 1:** Recall the Burkitt translocation.
Burkitt lymphoma is defined by a translocation that places the MYC oncogene on chromosome 8 next to an immunoglobulin gene. The classic and most common form is t(8;14), which juxtaposes MYC with the immunoglobulin heavy chain locus, causing constant MYC overexpression and rapid cell proliferation.

**Step 2:** Match the other translocations.
t(11;14) involves cyclin D1 and is seen in mantle cell lymphoma.
t(15;17) creates the PML-RARA fusion of acute promyelocytic leukemia (AML M3).
t(14;18) involves BCL2 and is the hallmark of follicular lymphoma.

**Step 3:** Conclusion.
Burkitt lymphoma is associated with t(8;14), option A.

**Quick Tip:** Which translocation puts MYC on chromosome 8 next to the IgH gene?

**72.** Translocation t(2;8)(p12;q24) is associated with:

(A) Chronic myeloid leukemia (CML)

(B) Acute myeloid leukemia (AML)

(C) T cell acute lymphoblastic leukemia (T-cell ALL)

(D) Burkitt lymphoma

**Correct Answer:** (D) Burkitt lymphoma

**Solution:**

**Step 1:** Decode the translocation.
The breakpoint 8q24 is the location of the MYC oncogene. Any translocation that involves 8q24 in a lymphoma points to MYC deregulation, which is the hallmark of Burkitt lymphoma.

**Step 2:** Identify this variant.
Besides the classic t(8;14), Burkitt lymphoma has two variant translocations. t(2;8) moves MYC next to the kappa light chain gene on chromosome 2, and t(8;22) moves it next to the lambda light chain gene. So t(2;8)(p12;q24) is the kappa light chain variant of Burkitt lymphoma.

**Step 3:** Rule out the others.
CML is associated with t(9;22), the Philadelphia chromosome (BCR-ABL).
AML subtypes have their own translocations, such as t(15;17) in APL and t(8;21) in AML M2.
T-cell ALL involves rearrangements of T-cell receptor loci, not 8q24.

**Step 4:** Conclusion.
t(2;8)(p12;q24) is the variant Burkitt lymphoma translocation, option D.

**Quick Tip:** 8q24 is the MYC locus, so think which lymphoma is driven by MYC.

**73.** All of the following are features of apoptosis, except:

(A) Cellular swelling

(B) Nuclear compaction

(C) Intact cell membrane

(D) Cytoplasmic eosinophilia

**Correct Answer:** (A) Cellular swelling

**Solution:**

**Step 1:** Define the two patterns of cell death. Apoptosis is programmed, energy-dependent death of single cells. Necrosis is accidental death of groups of cells after severe injury. The morphology differs, and that difference is the key to this question.
**Step 2:** List the true features of apoptosis. The cell shrinks, so the cytoplasm

becomes dense and stains deeply pink, giving **cytoplasmic eosinophilia**. The chromatin condenses and clumps under the nuclear membrane, which is **nuclear compaction** (pyknosis). The plasma membrane stays whole, so the cell breaks into membrane-bound apoptotic bodies, meaning the **cell membrane is intact**. There is no leakage of contents and no inflammation.

**Step 3:** Find the odd feature. **Cellular swelling** is the hallmark of **necrosis** and reversible injury, not apoptosis. It is caused by failure of the membrane ion pumps and water entry. So swelling does not belong to apoptosis.

**Step 4:** Reject the other options. Nuclear compaction, intact membrane, and cytoplasmic eosinophilia are all genuine apoptotic findings, so none of them is the exception.

**Conclusion:** The feature that is NOT seen in apoptosis is cellular swelling. The answer is option A.

**Quick Tip:** Shrinkage defines apoptosis; swelling defines necrosis.

**74.** Paroxysmal nocturnal hemoglobinuria (PNH) is associated with deficiency of:

(A) DAF (Decay accelerating factor)

(B) MIRL (Membrane inhibitor of reactive lysis)

(C) GPI anchored protein (Glycosyl phosphatidyl inositol anchored proteins)

(D) LFA (Lymphocyte function associated antigen)

**Correct Answer:** (C) GPI anchored protein (Glycosyl phosphatidyl inositol anchored proteins)

**Solution:**

**Step 1:** Recall the basic defect in PNH. PNH is an acquired clonal stem cell disorder caused by a somatic mutation in the **PIGA** gene. This gene is needed to make the **glycosyl phosphatidyl inositol (GPI) anchor**.

**Step 2:** Understand the GPI anchor. The GPI anchor is a small lipid stalk that fixes many surface proteins onto the red cell membrane. If the anchor cannot be made, every protein that depends on it is lost from the cell surface.

**Step 3:** Connect the anchor to hemolysis. Two of the lost proteins are complement regulators: **DAF (CD55)** and **MIRL (CD59)**. Without CD55 and CD59 the red cells cannot block complement, so complement attacks them and causes intravascular hemolysis, especially at night, producing hemoglobinuria.
**Step 4:** Choose the best single answer. DAF and MIRL are both deficient, but they are deficient only because the upstream GPI anchor is missing. The root and most complete answer is the **GPI anchored protein** defect, which explains the loss of both regulators at once.
**Step 5:** Reject the wrong option. LFA is a leukocyte adhesion molecule and has no role in PNH hemolysis, so option D is wrong.
**Conclusion:** The single best answer is deficiency of the GPI anchored protein. The answer is option C.

**Quick Tip:** The missing anchor explains loss of both CD55 and CD59 at once.

**75.** Plasmacytoid lymphomas (lymphoplasmacytic lymphoma) may be associated with:

(A) IgG

(B) IgM

(C) IgA

(D) IgE

**Correct Answer:** (B) IgM

**Solution:**

**Step 1:** Identify the tumour. A plasmacytoid or lymphoplasmacytic lymphoma is a B-cell lymphoma whose cells differentiate toward plasma cells and secrete a monoclonal immunoglobulin.
**Step 2:** Link it to the clinical syndrome. When this lymphoma secretes monoclonal **IgM**, it produces **Waldenstrom macroglobulinemia**. Macroglobulin literally means the large IgM molecule.
**Step 3:** Explain why IgM fits. IgM is a large pentamer. High levels of IgM thicken the blood, causing the hyperviscosity syndrome of Waldenstrom: visual blurring,

headache, and bleeding. This is the classic association tested here.
**Step 4:** Reject the other classes. IgG is the class of typical multiple myeloma, not plasmacytoid lymphoma. IgA myeloma also exists but is a plasma cell tumour, not a lymphoplasmacytic lymphoma. IgE myeloma is exceedingly rare and unrelated.
**Conclusion:** Plasmacytoid (lymphoplasmacytic) lymphoma is associated with IgM. The answer is option B.

**Quick Tip:** Think Waldenstrom macroglobulinemia and its large pentameric protein.

**76.** Which of the following has the most friable vegetation?

(A) Infective endocarditis

(B) Libman-Sacks endocarditis

(C) Rheumatic heart disease

(D) SLE

**Correct Answer:** (A) Infective endocarditis

**Solution:**

**Step 1:** Understand what friable means. A friable vegetation is soft, crumbly, and easily breaks off to throw emboli.
**Step 2:** Characterize each vegetation. **Infective endocarditis**, especially the acute form from Staphylococcus aureus, forms large bulky vegetations packed with fibrin, platelets, and live organisms. These are loosely held and crumble easily, so they embolize often and can destroy the valve.
**Step 3:** Compare the non-infective lesions. Rheumatic vegetations are small, firm, warty (verrucous) nodules sitting along the line of valve closure and are firmly attached, so they rarely embolize. Libman-Sacks endocarditis of SLE produces small sterile vegetations on either side of the valve; they are sterile and far less friable than infected ones.
**Step 4:** Note the overlap in options. Libman-Sacks endocarditis is the cardiac lesion of SLE, so options B and D describe the same entity; neither has friable vegetations as dramatic as infection.

**Conclusion:** The most friable vegetations are those of infective endocarditis. The answer is option A.

**Quick Tip:** Living bacteria make a soft crumbly mass that embolizes easily.

**77.** The characteristic pathological finding in carcinoid heart disease is:

(A) Fibrous endocardial thickening of the right ventricle, tricuspid valve and pulmonary valve

(B) Endometrial thickening of the tricuspid valve with severe tricuspid stenosis

(C) Collagen rich, elastic deposits in endocardium of right ventricle and pulmonary valve

(D) Calcification of the tricuspid and pulmonary valve

**Correct Answer:** (A) Fibrous endocardial thickening of the right ventricle, tricuspid valve and pulmonary valve

**Solution:**

**Step 1:** Recall the mechanism. Carcinoid tumours release serotonin and other vasoactive amines. When these reach the heart, they trigger deposition of fibrous tissue on the endocardium and valves.
**Step 2:** Locate the lesions. The mediators are inactivated in the lung, so the **right side of the heart** bears the damage while the left side is usually spared. The classic targets are the **right ventricular endocardium, the tricuspid valve, and the pulmonary valve**.
**Step 3:** Describe the lesion. The deposits are plaque-like fibrous (smooth muscle and collagen) thickenings of the endocardium and valve leaflets. This leads to tricuspid regurgitation and pulmonary stenosis.
**Step 4:** Match to the option. Option A states fibrous endocardial thickening of the right ventricle, tricuspid valve, and pulmonary valve, which is exactly the described lesion.
**Step 5:** Reject the others. Option B uses endometrial which is a typo and unrelated, and stenosis is not the typical tricuspid lesion (regurgitation is).

Option C wrongly stresses elastic deposits; the plaques are poor in elastic fibres. Option D describes calcific degeneration, which is not the carcinoid pattern.
**Conclusion:** The finding is fibrous endocardial thickening of the right heart structures. The answer is option A.

**Quick Tip:** Serotonin hits the right heart; the lung protects the left.

**78.** A female presents with a history of progressive breathlessness. Histology shows heterogeneous patchy fibrosis with several fibroblastic foci. The most likely diagnosis is:

(A) Cryptogenic organizing pneumonia

(B) Non-specific interstitial pneumonia

(C) Usual interstitial pneumonia

(D) Desquamative interstitial pneumonia

**Correct Answer:** (C) Usual interstitial pneumonia

**Solution:**

**Step 1:** Pick out the diagnostic clues. The two key phrases are **heterogeneous patchy fibrosis** and **fibroblastic foci**.
**Step 2:** Match these to a pattern. **Usual interstitial pneumonia (UIP)**, the histologic substrate of idiopathic pulmonary fibrosis, is defined by patchy, non-uniform fibrosis with areas of old scarring next to normal lung (temporal and spatial heterogeneity), plus scattered **fibroblastic foci** of active young collagen. This is the exact description given.
**Step 3:** Exclude non-specific interstitial pneumonia. NSIP shows **uniform, homogeneous** fibrosis or inflammation and characteristically lacks fibroblastic foci. The patchy heterogeneous pattern rules NSIP out.
**Step 4:** Exclude the others. Cryptogenic organizing pneumonia shows plugs of granulation tissue in the airspaces (Masson bodies), not interstitial fibroblastic foci. Desquamative interstitial pneumonia shows airspaces filled with pigmented macrophages and is smoking related, again without the heterogeneous fibrosis

pattern.
**Conclusion:** Heterogeneous fibrosis with fibroblastic foci is UIP. The answer is option C.

**Quick Tip:** Patchy heterogeneous fibrosis plus fibroblastic foci equals UIP.

**79.** The chromophobe variant of renal cell carcinoma is associated with:

(A) VHL gene mutations

(B) Trisomy of 7 and 17 (+7, +17)

(C) 3p deletions (3p-)

(D) Monosomy of 1 and Y (-1, -Y)

**Correct Answer:** (D) Monosomy of 1 and Y (-1, -Y)

**Solution:**

**Step 1:** Recall the genetics of the renal cell carcinoma subtypes. Each major subtype has its own chromosomal signature, which is the basis of this question.
**Step 2:** Assign the signatures. Clear cell carcinoma, the commonest type, is linked to the **VHL gene on chromosome 3p**, so it shows **3p deletions** and VHL mutations. Papillary carcinoma shows **trisomy of chromosomes 7 and 17** and loss of Y.
**Step 3:** Identify chromophobe. The **chromophobe** variant is characterized by multiple chromosome losses, classically **monosomy of chromosomes 1 and Y** and other monosomies, producing a hypodiploid genome.
**Step 4:** Eliminate the wrong options. VHL mutation and 3p deletion belong to clear cell carcinoma. Trisomy 7 and 17 belongs to papillary carcinoma. Only monosomy of 1 and Y fits chromophobe.
**Conclusion:** The chromophobe variant is associated with monosomy of 1 and Y. The answer is option D.

**Quick Tip:** Chromophobe loses chromosomes; think monosomy, not trisomy.

**80.** All of the following conditions are associated with granulomatous pathology, except:

(A) Wegener's granulomatosis (granulomatosis with polyangiitis)

(B) Takayasu arteritis

(C) Polyarteritis nodosa (classic PAN)

(D) Giant cell arteritis

**Correct Answer:** (C) Polyarteritis nodosa (classic PAN)

**Solution:**

**Step 1:** Define granulomatous inflammation. A granuloma is a focal collection of activated macrophages (epithelioid cells), often with giant cells. The question asks which listed vasculitis does NOT show granulomas.
**Step 2:** Check the granulomatous vasculitides. **Wegener's granulomatosis** shows necrotizing granulomatous inflammation of the respiratory tract and kidneys. **Takayasu arteritis** is a granulomatous large-vessel vasculitis of the aorta. **Giant cell (temporal) arteritis** is a granulomatous vasculitis with multinucleated giant cells in the arterial wall. So A, B, and D are all granulomatous.
**Step 3:** Examine polyarteritis nodosa. Classic **PAN** is a necrotizing vasculitis of medium-sized arteries marked by fibrinoid necrosis and a mixed inflammatory infiltrate. It is **not** granulomatous.
**Step 4:** Select the exception. Since PAN lacks granulomas while the other three are granulomatous, PAN is the odd one out.
**Conclusion:** The condition not associated with granulomatous pathology is polyarteritis nodosa. The answer is option C.

**Quick Tip:** Three are granulomatous; classic PAN shows fibrinoid necrosis instead.

**81.** Electron microscopy is diagnostic in:

(A) Goodpasture's syndrome

(B) Alport's syndrome

(C) Wegener's syndrome

(D) Churg-Strauss syndrome

**Correct Answer:** (B) Alport's syndrome

**Solution:**

**Step 1:** The question asks which renal disease is diagnosed mainly by the ultrastructural (electron microscopy) appearance of the glomerular basement membrane (GBM), rather than by light microscopy or immunofluorescence alone.
**Step 2:** Alport's syndrome is a hereditary nephritis caused by a mutation in the gene coding for the alpha-5 chain of type IV collagen. The defect produces a characteristic GBM change that is only visible on electron microscopy.
**Step 3:** On EM, the GBM in Alport's syndrome shows irregular thinning and thickening with splitting and lamellation of the lamina densa, giving a basket-weave appearance. This finding is essentially diagnostic of the disease.
**Step 4:** Why the others are wrong. Goodpasture's syndrome is diagnosed by linear immunofluorescence (anti-GBM antibody) and crescents on light microscopy, not by EM. Wegener's (granulomatosis with polyangiitis) and Churg-Strauss are ANCA-associated vasculitides diagnosed by serology, light microscopy (necrotizing granulomas, pauci-immune crescents) and clinical features.
**Step 5:** Therefore the disease in which EM is the diagnostic tool is Alport's syndrome.
Answer: Option B (Alport's syndrome).

**Quick Tip:** Look for the hereditary type IV collagen defect with a basket-weave GBM.

**82.** Which of the following is the most characteristic ultrastructural feature of paraganglioma on electron microscopy?

(A) Shrunken mitochondria

(B) Large Golgi apparatus

(C) Frequent mitoses

(D) Dense core neuroendocrine granules

**Correct Answer:** (D) Dense core neuroendocrine granules

**Solution:**

**Step 1:** Paraganglioma is a tumour arising from the paraganglia, which are clusters of neuroendocrine cells. The phaeochromocytoma of the adrenal medulla is the best known example of this family.
**Step 2:** Neuroendocrine cells store catecholamines and peptide hormones inside membrane-bound secretory granules. These are called dense-core granules because their centre is electron dense on EM.
**Step 3:** So under the electron microscope the defining feature of a paraganglioma is the presence of numerous membrane-bound dense-core neuroendocrine (neurosecretory) granules in the cytoplasm.
**Step 4:** Why the others are wrong. Shrunken mitochondria and a large Golgi apparatus are nonspecific and are not a defining feature of any particular tumour. Frequent mitoses are a feature seen on light microscopy and indicate proliferation, not neuroendocrine differentiation, so they cannot be the characteristic ultrastructural marker.
**Step 5:** Hence the answer is dense-core neuroendocrine granules.
Answer: Option D (Dense core neuroendocrine granules).

**Quick Tip:** Neuroendocrine cells store catecholamines in membrane-bound granules with a dark centre.

**83.** Which of the following is a marker of Langerhans cell histiocytosis?

(A) CD1a

(B) CD10

(C) CD30

(D) CD56

**Correct Answer:** (A) CD1a

**Solution:**

**Step 1:** Langerhans cell histiocytosis (LCH) is a proliferation of Langerhans-type dendritic cells. Diagnosis rests on identifying these cells by their characteristic markers and ultrastructure.
**Step 2:** The Langerhans cell expresses CD1a on its surface and stains positively for S-100 and for langerin (CD207). On EM it contains Birbeck granules, which look like tennis rackets.
**Step 3:** Among the options, CD1a is the surface marker that confirms Langerhans cell lineage, so it is the marker of LCH.
**Step 4:** Why the others are wrong. CD10 (CALLA) is a marker of precursor B cells, germinal-centre B-cell lymphomas and some renal and endometrial tumours. CD30 marks Reed-Sternberg cells of Hodgkin lymphoma and anaplastic large cell lymphoma. CD56 is a natural killer (NK) cell and neuroendocrine marker. None of these identify Langerhans cells.
**Step 5:** Therefore the correct marker is CD1a.
Answer: Option A (CD1a).

**Quick Tip:** Pair it with S-100 and Birbeck granules to spot the Langerhans cell.

**84.** Hypercoagulation in nephrotic syndrome is caused by:

(A) Loss of antithrombin III

(B) Decreased fibrinogen

(C) Decreased metabolism of vitamin K

(D) Increase in protein C

**Correct Answer:** (A) Loss of antithrombin III

**Solution:**

**Step 1:** In nephrotic syndrome the glomerular filter leaks protein, so the urine loses large amounts of plasma proteins. The pattern of which proteins are lost and which rise explains the clinical complications.
**Step 2:** Antithrombin III is a small natural anticoagulant of similar molecular size to albumin, so it is lost in the urine. Falling antithrombin III removes a key brake on coagulation and tips the balance towards clotting.
**Step 3:** At the same time the liver increases synthesis of large clotting proteins such as fibrinogen, and there is increased platelet activity, all of which further promote thrombosis (for example renal vein thrombosis).
**Step 4:** Why the others are wrong. Fibrinogen is increased, not decreased, in nephrotic syndrome, so option B is the opposite of the truth. Vitamin K metabolism is not the driving abnormality. Protein C is an anticoagulant, so an increase would reduce clotting rather than cause hypercoagulability, making option D the reverse of what happens.
**Step 5:** The main cause of the hypercoagulable state is therefore urinary loss of antithrombin III.
Answer: Option A (Loss of antithrombin III).

**Quick Tip:** An anticoagulant similar in size to albumin slips into the urine.

**85.** Which of the following markers is specific for gastrointestinal stromal tumors (GIST)?

(A) CD117

(B) CD34

(C) CD23

(D) S-100

**Correct Answer:** (A) CD117

**Solution:**

**Step 1:** GIST arises from the interstitial cells of Cajal, the pacemaker cells of the gut wall. Most cases carry an activating mutation in the c-KIT gene.
**Step 2:** The c-KIT gene codes for a tyrosine kinase receptor whose protein is detected by the antibody CD117. Because nearly all GISTs overexpress this receptor, CD117 positivity is the defining and most specific marker of the tumour.
**Step 3:** This marker is also clinically important because CD117 positive tumours respond to imatinib, a tyrosine kinase inhibitor.
**Step 4:** Why the others are wrong. CD34 is positive in many GISTs but it is not specific, since it also marks vascular and other mesenchymal tumours. CD23 is a marker of follicular dendritic cells and small lymphocytic lymphoma/CLL. S-100 marks nerve sheath tumours and melanoma. None of these is specific for GIST.
**Step 5:** Therefore the specific marker is CD117.
Answer: Option A (CD117).

**Quick Tip:** Think c-KIT receptor and the drug imatinib.

**86.** Down's syndrome is most commonly caused by:

(A) Maternal nondisjunction

(B) Paternal nondisjunction

(C) Translocation

(D) Mosaicism

**Correct Answer:** (A) Maternal nondisjunction

**Solution:**

**Step 1:** Down's syndrome is trisomy 21, meaning the patient has three copies of chromosome 21. The mechanisms that produce this extra chromosome differ in frequency.
**Step 2:** About 95 percent of cases result from nondisjunction, where the two copies of chromosome 21 fail to separate during meiosis, giving a gamete with an

extra chromosome 21.
**Step 3:** This nondisjunction occurs far more often in the egg than in the sperm, mostly during meiosis I, and its frequency rises sharply with advancing maternal age. So maternal nondisjunction is the single most common cause.
**Step 4:** Why the others are wrong. Paternal nondisjunction accounts for only a small minority of nondisjunction cases. Robertsonian translocation (commonly 14;21) causes about 4 percent of cases and is the type that can be inherited. Mosaicism accounts for only about 1 to 2 percent.
**Step 5:** Therefore the most common cause is maternal nondisjunction.
Answer: Option A (Maternal nondisjunction).

**Quick Tip:** About 95 percent are nondisjunction, and the egg is the usual source.

**87.** Cystic fibrosis is inherited as an autosomal recessive condition. A normal couple has one daughter affected with the disease. They are now planning to have another child. What is the chance of their next child being affected by the disease?

(A) 0

(B) 1/2

(C) 1/4

(D) 3/4

**Correct Answer:** (C) 1/4

**Solution:**

**Step 1:** Cystic fibrosis is autosomal recessive, so a person needs two copies of the mutant allele to be affected. Let the alleles be A (normal) and a (mutant).
**Step 2:** Both parents are clinically normal but have already produced an affected daughter (genotype aa). The daughter inherited one a from each parent, so each parent must carry one a. Both parents are therefore carriers with genotype Aa.
**Step 3:** Cross Aa with Aa. The offspring ratio is 1 AA to 2 Aa to 1 aa. The aa genotype is the affected one.
**Step 4:** So the probability that the next child is affected (aa) is 1 in 4, that is 1/4.

Each pregnancy is independent, so the earlier affected child does not change this risk for the next child.
**Step 5:** The printed key gives option C, which matches this calculation.
Answer: Option C (1/4).

**Quick Tip:** Both healthy parents of an affected child must be Aa carriers.

**88.** Males are more commonly affected than females in:

(A) Autosomal dominant

(B) Autosomal recessive

(C) X-linked dominant

(D) X-linked recessive

**Correct Answer:** (D) X-linked recessive

**Solution:**

**Step 1:** The question asks which inheritance pattern shows a clear male predominance among affected people.
**Step 2:** Males have only one X chromosome (XY), so they are hemizygous for X-linked genes. A single recessive mutant allele on the X is enough to make a male affected, because he has no second X to mask it.
**Step 3:** A female (XX) needs the mutant allele on both X chromosomes to be affected by an X-linked recessive disease, which is far less likely. So in X-linked recessive conditions (for example haemophilia A and B, Duchenne muscular dystrophy, red-green colour blindness) males are affected much more often than females.
**Step 4:** Why the others are wrong. Autosomal dominant and autosomal recessive disorders affect both sexes roughly equally because the genes are not on the sex chromosomes. X-linked dominant conditions actually tend to affect females more often or to be lethal in males, the opposite of male predominance.

**Step 5:** Therefore the pattern with male predominance is X-linked recessive.
Answer: Option D (X-linked recessive).

**Quick Tip:** A male has only one X, so a single recessive allele is enough.

**89.** All of the following statements about El Tor vibrios are true, except:

(A) Humans are the only reservoir

(B) Can survive in ice cold water for 2 to 4 weeks

(C) Killed by boiling for 30 seconds

(D) Enterotoxin can have direct effects on tissues other than intestinal epithelial cells

**Correct Answer:** (D) Enterotoxin can have direct effects on tissues other than intestinal epithelial cells

**Solution:**

**Step 1:** The question asks for the FALSE statement about the El Tor biotype of Vibrio cholerae.
**Step 2:** Humans are the only known natural reservoir of Vibrio cholerae. There is no animal or environmental reservoir that maintains the organism long term, so option A is a true statement.
**Step 3:** Vibrio cholerae, especially the El Tor biotype, is hardy in water. It can survive in ice cold water for 2 to 4 weeks, which helps explain water borne spread, so option B is true.
**Step 4:** Vibrios are killed easily by heat. Boiling for about 30 seconds destroys them, which is why boiled water is safe in cholera areas. Option C is true.
**Step 5:** The action of cholera toxin is highly specific. It binds the GM1 ganglioside receptor on intestinal epithelial cells and raises cyclic AMP, causing massive secretion of water and electrolytes into the gut lumen. The toxin does NOT act directly on other tissues besides the intestinal epithelium. So the claim that the enterotoxin has direct effects on other tissues is false.

**Conclusion:** The exception is option D, the enterotoxin acting on tissues besides intestinal epithelium.

**Quick Tip:** Cholera toxin binds GM1 on enterocytes only, not on other tissues.

**90.** The peptide binding site on class I MHC molecules for presenting processed antigen to CD8 T cells is formed by:

(A) Proximal domain of the alpha subunit

(B) Distal domains of the alpha subunit

(C) Proximal domains of the alpha and beta subunits

(D) Distal domains of the alpha and beta subunits

**Correct Answer:** (B) Distal domains of the alpha subunit

**Solution:**

**Step 1:** MHC class I molecules present endogenous peptides to CD8 positive cytotoxic T cells. The structure has one polymorphic alpha chain with three domains, alpha1, alpha2 and alpha3, plus a non polymorphic beta2 microglobulin chain.
**Step 2:** The peptide binding groove of MHC class I is formed by the alpha1 and alpha2 domains. These are the membrane distal domains of the alpha chain, sitting farthest from the cell surface.
**Step 3:** The alpha3 domain is membrane proximal and is the one that interacts with the CD8 co receptor. Beta2 microglobulin only stabilizes the molecule and does not form the groove.
**Step 4:** So the binding site is built only by distal domains of the alpha chain. Option B describes the distal domains of the alpha subunit, which is correct.
**Step 5:** Why the others are wrong. Option A, a single proximal domain, is the alpha3 region, which binds CD8, not peptide. Options C and D wrongly bring in the beta chain, but in class I the beta chain is beta2 microglobulin and it does not contribute to the groove. The combined alpha plus beta groove description fits MHC class II, not class I.

**Conclusion:** The peptide binding site is formed by the distal domains of the alpha subunit, option B.

**Quick Tip:** Class I groove is alpha1 plus alpha2, both distal domains of the same alpha chain.

**91.** All of the following statements about Staphylococcus aureus are true, except:

(A) The most common source of infection is cross infection from infected people

(B) About 30 percent of the general population are healthy nasal carriers

(C) Epidermolysin and toxic shock syndrome toxin are superantigens

(D) Methicillin resistance is chromosomally mediated

**Correct Answer:** (A) The most common source of infection is cross infection from infected people

**Solution:**

**Step 1:** The task is to find the false statement about Staphylococcus aureus.
**Step 2:** Carriage. The main reservoir of S. aureus is the human nose and skin. Around 30 percent of healthy people carry it in the anterior nares without symptoms. So option B is true.
**Step 3:** Source of infection. Because so many people are healthy carriers, most staphylococcal infections are endogenous, that is they come from the patient's own carrier flora rather than cross infection from another infected person. The statement that the most common source is cross infection from infected people is therefore incorrect for general staphylococcal disease.
**Step 4:** Toxins. Toxic shock syndrome toxin 1 is a classic superantigen, and the exfoliative or epidermolytic toxins also behave as superantigens. So option C is acceptable as true.
**Step 5:** Resistance. Methicillin resistance in MRSA is due to the mecA gene carried on the SCCmec element, which is integrated into the bacterial chromosome. So methicillin resistance is chromosomally mediated, and option D is true.

**Conclusion:** The exception is option A, since most infections arise from the patient's own nasal or skin carriage, not from cross infection from infected people.

**Quick Tip:** Most staph infections come from the patient's own nasal carriage, not from others.

**92.** A child presents with sepsis. The bacteria isolated showed beta hemolysis on blood agar, resistance to bacitracin, and a positive CAMP test. The most likely organism is:

(A) Streptococcus agalactiae (Group B Streptococcus)

(B) Streptococcus pyogenes (Group A Streptococcus)

(C) Streptococcus pneumoniae

(D) Enterococcus faecalis

**Correct Answer:** (A) Streptococcus agalactiae (Group B Streptococcus)

**Solution:**

**Note:** The block file truncated this stem and gave no printed key. The reconstructed lab profile (beta hemolytic, bacitracin resistant, with the classic CAMP reaction in a septic child) points clearly to Group B Streptococcus, so options and answer are filled from standard microbiology.

**Step 1:** Beta hemolysis on blood agar narrows the list to Group A and Group B streptococci among the common pyogenic cocci.

**Step 2:** Bacitracin sensitivity separates them. Group A Streptococcus (S. pyogenes) is bacitracin sensitive, while Group B Streptococcus (S. agalactiae) is bacitracin resistant. The isolate here is bacitracin resistant, which fits Group B and rules out S. pyogenes.

**Step 3:** The CAMP test is positive for Group B Streptococcus. S. agalactiae produces the CAMP factor, which enhances the hemolysis of Staphylococcus aureus and gives an arrowhead zone. This confirms Group B.

**Step 4:** Clinical fit. Group B Streptococcus is the leading cause of neonatal and early infant sepsis and meningitis, which matches a child presenting with sepsis.

**Step 5:** Why the others are wrong. S. pyogenes is bacitracin sensitive and CAMP negative. S. pneumoniae is alpha hemolytic and optochin sensitive. Enterococcus is usually non hemolytic or alpha and is bile esculin and salt tolerant, not CAMP defined.
**Conclusion:** The organism is Streptococcus agalactiae, option A.

**Quick Tip:** Beta hemolytic, bacitracin resistant, CAMP positive equals Group B Streptococcus.

**93.** Isolation of Chlamydia from a tissue specimen can be done by:

(A) ELISA (enzyme linked immunosorbent assay)

(B) Yolk sac inoculation

(C) Direct immunofluorescence antibody test (DFA)

(D) Polymerase chain reaction (PCR)

**Correct Answer:** (B) Yolk sac inoculation

**Solution:**

**Step 1:** The key word is isolation. Isolation means recovering the live organism by growing it, not just detecting its antigen or nucleic acid.
**Step 2:** Chlamydiae are obligate intracellular bacteria, so they cannot grow on ordinary cell free media. They need living cells. Classic methods are inoculation into the yolk sac of an embryonated hen egg or into cell cultures such as McCoy cells.
**Step 3:** Among the options, yolk sac inoculation is the only true culture or isolation method. It supports the intracellular growth cycle of Chlamydia.
**Step 4:** Why the others are wrong. ELISA detects chlamydial antigen, it is a detection test, not isolation. Direct immunofluorescence with antibody detects elementary bodies in a smear, again detection, not isolation. PCR detects chlamydial DNA, which is detection of nucleic acid and not recovery of a living organism.
**Conclusion:** Isolation is done by yolk sac inoculation, option B.

**Quick Tip:** Isolation means growing the organism; Chlamydia needs living cells like the egg yolk sac.

**94.** Varicella zoster virus remains latent in:

(A) Trigeminal ganglion (dorsal root and cranial sensory ganglia)

(B) T cells

(C) B cells

(D) Macrophages

**Correct Answer:** (A) Trigeminal ganglion (dorsal root and cranial sensory ganglia)

**Solution:**

**Step 1:** Varicella zoster virus is a member of the herpesvirus family. A defining feature of herpesviruses is lifelong latency in specific host cells after the primary infection.

**Step 2:** The primary infection with VZV is chickenpox. After it resolves, the virus travels along sensory nerves and becomes dormant in sensory nerve cell bodies, that is in the dorsal root ganglia and cranial sensory ganglia such as the trigeminal ganglion.

**Step 3:** Reactivation. When immunity wanes, the virus reactivates and spreads down the sensory nerve to the skin, producing herpes zoster (shingles) in a dermatomal pattern. Involvement of the trigeminal ganglion causes ophthalmic zoster.

**Step 4:** Why the others are wrong. T cells, B cells and macrophages are sites of latency for other viruses. For example, Epstein Barr virus is latent in B lymphocytes and HIV in CD4 T cells and macrophages, but VZV is neurotropic and hides in sensory ganglia, not in these cells.

**Conclusion:** Varicella zoster virus remains latent in the sensory ganglia, represented by the trigeminal ganglion, option A.

**Quick Tip:** VZV is neurotropic and stays dormant in sensory ganglia like the trigeminal

ganglion.

**95.** The most common genital lesion in an HIV patient is:

(A) Chlamydia

(B) Herpes

(C) Syphilis

(D) Candida

**Correct Answer:** (B) Herpes

**Solution:**

**Step 1:** The question asks which genital lesion is most common in people with HIV. HIV impairs cell mediated immunity, which strongly favors reactivation and persistence of latent viral infections.

**Step 2:** Genital herpes is caused by herpes simplex virus, which establishes lifelong latency in sacral ganglia. In HIV the weakened T cell response leads to frequent, severe and sometimes chronic ulcerative herpes, making it the commonest genital ulcerative lesion in these patients.

**Step 3:** There is also a two way link. Genital herpes ulcers break the mucosal barrier and increase the risk of acquiring and transmitting HIV, and HIV in turn worsens herpes. This association makes herpes the typical genital lesion seen in HIV.

**Step 4:** Why the others are less correct. Chlamydia commonly causes urethritis and cervicitis rather than a classic visible lesion. Syphilis does cause genital ulcers and is more aggressive in HIV, but it is less common overall than recurrent herpes. Candida causes vulvovaginitis or balanitis, an infection rather than the classic ulcerative genital lesion emphasized here.

**Conclusion:** The most common genital lesion in HIV is herpes, option B.

**Quick Tip:** Failing cell mediated immunity makes recurrent genital herpes the commonest lesion.

**96.** A diabetic patient presents with bloody nasal discharge, orbital swelling and pain. Culture of the periorbital pus shows branching septate hyphae. Which of the following is the most probable organism involved?

(A) Mucor

(B) Candida

(C) Aspergillus

(D) Rhizopus

**Correct Answer:** (C) Aspergillus

**Solution:**

**Step 1:** The clinical picture, a diabetic with bloody nasal discharge, orbital swelling and pain, is a rhino orbital fungal infection. Two main fungal groups cause this, the Mucorales (Mucor and Rhizopus) and Aspergillus.
**Step 2:** The deciding clue is the microscopy. The pus shows branching SEPTATE hyphae. Hyphal morphology separates the two groups.
**Step 3:** Aspergillus has septate hyphae that branch at acute angles, classically about 45 degrees, with regular cross walls. So septate hyphae point to Aspergillus.
**Step 4:** The Mucorales, that is Mucor and Rhizopus, show broad, aseptate or sparsely septate hyphae that branch at wide angles near 90 degrees. They would NOT be described as septate. So Mucor and Rhizopus are excluded by the word septate.
**Step 5:** Candida is a yeast that forms pseudohyphae and budding cells, not true branching septate hyphae of this filamentous type, so it does not fit.
**Conclusion:** Branching septate hyphae in this rhino orbital infection indicate Aspergillus, option C. If the hyphae had been broad and aseptate, the answer would have been Mucor or Rhizopus.

**Quick Tip:** Septate branching hyphae mean Aspergillus; broad aseptate hyphae would mean Mucor or Rhizopus.

**97.** All of the following statements about Penicillin binding proteins (PBPs) are true, except:

(A) PBPs are localized on the outer face of the cell wall

(B) PBPs are essential for cell wall synthesis

(C) PBPs act as carboxypeptidases and transpeptidases

(D) Alteration in PBPs is the primary basis of resistance in MRSA

**Correct Answer:** (A) PBPs are localized on the outer face of the cell wall

**Solution:**

**Step 1:** The question asks for the FALSE statement about penicillin binding proteins (PBPs).
**Step 2:** PBPs are enzymes that sit on the cytoplasmic membrane, not on the outer face of the cell wall. They face the periplasmic side and build the peptidoglycan layer from inside out. So option A is the wrong (false) statement, and that is the answer.
**Step 3:** Option B is true. PBPs catalyse the final cross-linking steps of peptidoglycan, so they are essential for cell wall synthesis. Blocking them stops the wall from forming and the cell lyses.
**Step 4:** Option C is true. PBPs work as transpeptidases (they cross-link the peptide chains) and as carboxypeptidases (they trim terminal D-alanine residues). Beta-lactams mimic the D-Ala-D-Ala terminus and bind the active site.
**Step 5:** Option D is true. MRSA carries the *mecA* gene that codes for PBP2a, a low-affinity PBP. Beta-lactams cannot bind it, so altered PBP is the main resistance mechanism in MRSA.
**Conclusion:** The false statement is that PBPs are localized on the outer face of the cell wall. Answer: option A.

**Quick Tip:** Ask where these enzymes physically sit relative to the membrane and the wall.

**98.** Which of the following may cause biliary obstruction?

(A) Ancylostoma

(B) Enterobius

(C) Strongyloides

(D) Clonorchis

**Correct Answer:** (D) Clonorchis

**Solution:**

**Step 1:** The question asks which parasite lives in the biliary tract and can block bile flow.
**Step 2:** *Clonorchis sinensis*, the Chinese liver fluke, lives in the intrahepatic bile ducts. The adult flukes and their eggs cause inflammation, fibrosis and obstruction of the bile ducts, and chronic infection is linked to cholangiocarcinoma. So option D is correct.
**Step 3:** Option A is wrong. *Ancylostoma duodenale* (hookworm) attaches to the small intestine and causes iron deficiency anaemia, not biliary disease.
**Step 4:** Option B is wrong. *Enterobius vermicularis* (pinworm) lives in the caecum and colon and causes perianal itching; it does not enter the bile ducts.
**Step 5:** Option C is wrong. *Strongyloides stercoralis* lives in the small intestine and causes autoinfection and hyperinfection in the gut and lungs, not biliary obstruction.
**Conclusion:** The liver fluke that obstructs bile ducts is Clonorchis. Answer: option D.

**Quick Tip:** Which of these is a liver fluke that lives inside the bile ducts?

**99.** A young woman complains of recurrent rhinitis, nasal discharge and bilateral nasal blockage since one year. She also had a history of allergy and asthma. On examination, multiple polyps with mucosal thickening and impacted secretions are seen in the nasal cavities. A biopsy was taken and the material on culture showed many hyphae with dichotomous branching typically at 45 degrees. Which of the following is the most likely organism responsible?

(A) Rhizopus

(B) Aspergillus

(C) Mucor

(D) Candida

**Correct Answer:** (B) Aspergillus

**Solution:**

**Step 1:** Read the clinical picture. A young, allergic, asthmatic woman with nasal polyps, thick mucosal disease and impacted secretions points toward allergic fungal rhinosinusitis.
**Step 2:** The decisive clue is the microscopy: septate hyphae that branch dichotomously (into two equal branches) at acute angles of about 45 degrees. This is the classic morphology of *Aspergillus*. So option B is correct.
**Step 3:** Option A is wrong. *Rhizopus* is a Mucorale. Its hyphae are broad, ribbon-like and aseptate (or sparsely septate) and branch at wide, roughly 90 degree angles.
**Step 4:** Option C is wrong. *Mucor* is also a Mucorale with the same broad, aseptate, wide-angle hyphae and typically causes invasive rhino-orbital disease in diabetics, not allergic polyposis.
**Step 5:** Option D is wrong. *Candida* shows budding yeasts with pseudohyphae, not regular acute-angle dichotomous branching, and it does not cause this allergic fungal sinusitis picture.
**Conclusion:** Septate, acute-angle (45 degree) dichotomously branching hyphae in an allergic patient mean Aspergillus. Answer: option B.

**Quick Tip:** Septate hyphae branching at a 45 degree acute angle in an allergic patient.

**100.** Primary impact injuries (in a pedestrian hit by a vehicle) are commonly seen on:

(A) Chest

(B) Abdomen

(C) Legs

(D) Head

**Correct Answer:** (C) Legs

**Solution:**

**Step 1:** Understand the term. In a road traffic accident where a pedestrian is struck by a car, the first point of contact between the vehicle and the body produces the primary impact injury.
**Step 2:** The leading part of a car, the bumper, strikes the standing pedestrian at the level of the lower limbs. So the primary impact (bumper) injuries are seen on the legs, usually below the knees. So option C is correct.
**Step 3:** Why not the others. After the primary impact, the victim is thrown onto the bonnet, producing secondary impact injuries on the trunk, chest and head. So chest (A), abdomen (B) and head (D) belong to the secondary impact, not the primary one.
**Step 4:** Sequence reminder: primary impact (legs from bumper), secondary impact (thrown onto vehicle, hitting bonnet or windscreen), then secondary injuries (when thrown to the ground).
**Conclusion:** Primary impact from the bumper lands on the legs. Answer: option C.

**Quick Tip:** Which part of the body does a car bumper strike first?

**101.** A woman died within 5 years of marriage under suspicious circumstances. Her parents complained that her in-laws used to frequently demand dowry. Under which of the following sections can a magistrate authorize autopsy of the case?

(A) Section 174 CrPC

(B) Section 176 CrPC

(C) Section 304 IPC

(D) Section 302 IPC

**Correct Answer:** (B) Section 176 CrPC

**Solution:**

**Step 1:** Frame the scenario. Death of a woman within 7 years of marriage under suspicious circumstances, with dowry demand, is a dowry death. The question asks which section lets a magistrate order the autopsy.

**Step 2:** Section 176 CrPC empowers a magistrate (Executive or Judicial) to hold an inquiry into the cause of death and to direct the exhumation and post-mortem examination, including in cases of suspicious or custodial death. For a woman dying within 7 years of marriage in suspicious circumstances, magisterial inquiry under Section 176 is mandatory. So option B is correct.

**Step 3:** Option A is wrong. Section 174 CrPC deals with the police inquiry into unnatural or suspicious death; the investigating officer conducts inquest and sends the body for post-mortem. It is a police power, not the magistrate's authorisation of autopsy in this setting.

**Step 4:** Option C is wrong. Section 304 IPC is a punishment section (culpable homicide not amounting to murder; 304B is dowry death). It defines the offence and penalty, not the authority to order an autopsy.

**Step 5:** Option D is wrong. Section 302 IPC is the punishment for murder. Again it is a penal provision, not a section that authorises a post-mortem.

**Conclusion:** The magisterial power to inquire and order autopsy here is Section 176 CrPC. Answer: option B.

**Quick Tip:** Separate police inquest powers from a magistrate's inquiry power, and CrPC from IPC.

**102.** A factory worker presents with excessive salivation, blue lines on gums, tremors, disturbed personality, insomnia and loss of appetite. The most likely poisoning is:

(A) Mercury

(B) Lead

(C) Arsenic

(D) Phosphorus

**Correct Answer:** (A) Mercury

**Solution:**

**Step 1:** Identify the cluster of features. Excessive salivation, fine tremors, behavioural and personality change, insomnia and anorexia together point to chronic mercury poisoning (mercurialism).
**Step 2:** Chronic mercury exposure produces a classic triad: tremor (the mercurial tremor), erethism (timidity, irritability, personality change, insomnia, memory loss) and gingivitis with excessive salivation. A blue line on the gums can also be seen. So option A is correct.
**Step 3:** Option B is wrong. Lead poisoning gives a blue Burton line on the gums, but the rest of the picture differs: colic, wrist drop, anaemia with basophilic stippling and lead encephalopathy, not marked salivation with the erethism-tremor pattern. The combined salivation plus tremor plus personality change fits mercury better.
**Step 4:** Option C is wrong. Arsenic causes the rice-water diarrhoea, raindrop pigmentation, hyperkeratosis and Mees lines on the nails, not this salivation-tremor-erethism syndrome.
**Step 5:** Option D is wrong. Phosphorus poisoning causes garlic breath, vomiting and the typical staged hepatic damage, not these neuropsychiatric and salivary features.
**Conclusion:** Salivation, tremor and erethism in a factory worker indicate mercury. Answer: option A.

**Quick Tip:** Tremor plus erethism plus heavy salivation is one classic metal triad.

**103.** Which of the following bullets leaves a visible mark, so that a person can see its path:

(A) Tandem bullet

(B) Tracer bullet

(C) Dum dum bullet

(D) Incendiary bullet

**Correct Answer:** (B) Tracer bullet

**Solution:**

**Step 1:** The key word is a bullet whose path can be seen, that is, a glowing trail in flight.
**Step 2:** A tracer bullet has a pyrotechnic charge in its base that burns brightly while the bullet travels, producing a visible luminous trail. Shooters use this to follow and correct their aim. So option B is correct.
**Step 3:** Option A is wrong. A tandem (or piggyback) bullet is when a second bullet lodged in the barrel is fired out ahead of the next one; it has nothing to do with a visible flight path.
**Step 4:** Option C is wrong. A dum dum bullet is a soft-nosed expanding bullet that mushrooms on impact to cause greater wounding; it does not glow in flight.
**Step 5:** Option D is wrong. An incendiary bullet carries a substance that ignites on impact to start a fire at the target; the burning is at the target, not a continuous visible trail during flight as the question intends.
**Conclusion:** The bullet with a visible glowing track is the tracer bullet. Answer: option B.

**Quick Tip:** Which bullet is made to glow so the shooter can follow its flight?

**104.** Lightning flash can cause injury by all of the following, except:

(A) Direct effect of the electric current

(B) Superheated air

(C) Expanded and repelled air

(D) Compressed air pushed in front of the current

**Correct Answer:** (D) Compressed air pushed in front of the current

**Solution:**

**Step 1:** The question asks which listed mechanism does NOT cause lightning injury.
**Step 2:** Lightning injures through three accepted mechanisms. First, the direct passage of the high-voltage current through the body causes cardiac arrest, burns and neurological damage. So option A is a real mechanism (not the answer).
**Step 3:** Second, the lightning channel superheats the surrounding air to thousands of degrees, and this superheated air causes burns and flash injuries. So option B is a real mechanism (not the answer).
**Step 4:** Third, the explosive expansion of that air produces a blast (thunder) that expands and repels the air violently, and this blast effect can throw and injure the victim. So option C is a real mechanism (not the answer).
**Step 5:** The injury comes from the explosive expansion and the resulting repelled air, not from a column of compressed air being pushed ahead of the current. Compressed air pushed in front of the current is the description that does not fit the accepted mechanisms, so option D is the exception and is the answer.
**Conclusion:** Lightning injures by the current itself, superheated air and the expanding/repelled air blast, but not by compressed air pushed ahead of it. Answer: option D.

**Quick Tip:** Three mechanisms are real (current, superheated air, expanding blast); one wording is the odd one out.

**105.** Aconite poisoning causes all of the following except:

(A) Hypersalivation

(B) Tingling and numbness

(C) Increased blood pressure

(D) Chest pain

**Correct Answer:** (C) Increased blood pressure

**Solution:**

**Step 1:** Aconite (from Aconitum napellus, also called monkshood or Indian aconite, vatsanabha) is one of the most lethal plant poisons. Its active alkaloid is aconitine.
**Step 2:** Aconitine acts on voltage-gated sodium channels and keeps them open, causing persistent depolarisation of nerve and cardiac muscle. This explains the classic features.
**Step 3:** Local and early effects include tingling, numbness and burning of the mouth, tongue and skin (paraesthesia), along with marked hypersalivation. So options A and B are real features.
**Step 4:** On the heart it causes bradycardia, falling blood pressure (**hypotension**), arrhythmias and chest pain. Blood pressure goes **down**, not up. So an increase in blood pressure is NOT a feature of aconite poisoning.
**Step 5:** Therefore the exception is increased BP. The answer is option C.

**Quick Tip:** Aconitine keeps sodium channels open, so the heart slows and pressure drops, not rises.

**106.** Which of the following drugs is used for narcoanalysis?

(A) Atropine

(B) Phenobarbitone

(C) Scopolamine

(D) Pethidine

**Correct Answer:** (C) Scopolamine

**Solution:**

**Step 1:** Narcoanalysis (the so-called truth serum test) puts a person into a half-conscious, suggestible hypnotic state in which they are thought to speak more freely.
**Step 2:** The drugs classically used are the rapidly acting agents that produce this twilight sedation. These include scopolamine (hyoscine), and short-acting barbiturates such as thiopentone and sodium amytal.
**Step 3:** Among the given options, scopolamine is the recognised narcoanalysis agent. It was historically the original truth serum.
**Step 4:** Why the others are wrong. Atropine is an anticholinergic used for bradycardia and as an antidote, not for narcoanalysis. Phenobarbitone is a long-acting barbiturate used as an anticonvulsant, not the short-acting type used for the test. Pethidine is an opioid analgesic.
**Step 5:** The answer is option C, scopolamine.

**Quick Tip:** The original truth serum is the antimuscarinic hyoscine.

**107.** Which of the following is associated with emotional valence and is most likely to be influenced by motivation?

(A) Attitude

(B) Belief

(C) Practice

(D) Knowledge

**Correct Answer:** (C) Practice

**Solution:**

**Step 1:** This question comes from the KAP framework in community medicine, which describes Knowledge, Attitude and Practice, with belief sitting alongside.
**Step 2:** Knowledge is what a person knows, a purely cognitive item. Belief is what a person holds to be true. Attitude is the feeling or readiness to act, so it

carries emotional valence but is still an internal state.
**Step 3:** Practice is the actual behaviour or action a person carries out. Behaviour is the step that turns feeling into doing, so it is the component most directly driven by motivation. A person may know and feel correctly yet still not act unless motivated; motivation is the bridge to practice.
**Step 4:** So the item that both has an emotional driver and is most strongly moved by motivation is practice, the observable action.
**Step 5:** The answer is option C, practice.

**Quick Tip:** Think KAP: which component is the actual action that needs a push to happen?

**108.** ASHA (Accredited Social Health Activist) is posted at:

(A) Village level

(B) Primary Health Centre

(C) Community Health Centre

(D) Sub-centre

**Correct Answer:** (A) Village level

**Solution:**

**Step 1:** ASHA stands for Accredited Social Health Activist, a community health worker created under the National Rural Health Mission (NRHM) in 2005.
**Step 2:** The ASHA is selected from the village she serves. The norm is one ASHA per 1000 population at the village level, so she is a resident village-level volunteer who links the community to the health system.
**Step 3:** The other options name health facilities staffed by trained personnel. A sub-centre is the most peripheral facility (manned by an ANM and a male health worker), a Primary Health Centre is run by a medical officer, and a Community Health Centre is a 30-bedded first referral unit. The ASHA is not posted at any of these; she works in and from the village.
**Step 4:** The answer is option A, village level.

**Quick Tip:** One ASHA per 1000 population, chosen from and based in the village.

**109.** Movement across socioeconomic levels is termed as:

(A) Social equality

(B) Social upliftment

(C) Social mobility

(D) Social insurance

**Correct Answer:** (C) Social mobility

**Solution:**

**Step 1:** The question asks for the term that describes a person or group moving from one socioeconomic level to another.
**Step 2:** Social mobility is defined exactly as the movement of individuals or families between social or economic strata. It can be upward or downward, and across or within generations.
**Step 3:** The other terms do not mean movement between levels. Social equality means all members having the same status or rights. Social upliftment is a loose phrase for raising the disadvantaged, not the technical term. Social insurance is a financing mechanism that pools risk to protect against illness or loss of income.
**Step 4:** The precise sociological term for moving across socioeconomic levels is social mobility. The answer is option C.

**Quick Tip:** Moving up or down the social ladder has a precise sociological name.

**110.** JSY stands for:

(A) Janani Suraksha Yojana

(B) Jeevan Swastha Yojana

(C) Jan Sewa Yojana

(D) Jan Suraksha Yojana

**Correct Answer:** (A) Janani Suraksha Yojana

**Solution:**

**Step 1:** JSY is a well-known maternal health scheme launched in 2005 under the National Rural Health Mission.
**Step 2:** JSY expands to Janani Suraksha Yojana, where Janani means mother and Suraksha means protection. It is a safe motherhood and conditional cash transfer scheme.
**Step 3:** Its aim is to reduce maternal and neonatal mortality by promoting institutional delivery among poor pregnant women, giving them a cash incentive to deliver in a health facility. The ASHA helps link the mother to the scheme.
**Step 4:** The other expansions in the options are not real scheme names. The correct full form is Janani Suraksha Yojana, option A.

**Quick Tip:** Janani means mother; the scheme pays for safe institutional delivery.

**111.** The concept of primary health care was provided by:

(A) Bhore Committee

(B) Alma-Ata Declaration

(C) Shrivastava Committee

(D) National Health Policy

**Correct Answer:** (B) Alma-Ata Declaration

**Solution:**

**Step 1:** The term primary health care, with its formal definition and the slogan Health for All by 2000 AD, came from the International Conference on Primary

Health Care held at Alma-Ata in 1978.
**Step 2:** The Alma-Ata Declaration defined primary health care as essential health care made universally accessible to all, and laid out its elements and principles such as equity, community participation and intersectoral coordination.
**Step 3:** The other bodies are Indian and predate or differ from this concept. The Bhore Committee (1946) designed India's three-tier rural health structure. The Shrivastava Committee (1975) recommended community health workers and led to ideas later linked with India's primary care, but the global concept itself is Alma-Ata. National Health Policy documents adopt and apply the concept but did not originate it.
**Step 4:** The answer is option B, the Alma-Ata Declaration.

**Quick Tip:** Think 1978 and the slogan Health for All by 2000 AD.

**112.** Which of the following best reflects the highest level of community participation?

(A) Planning of intervention by the community

(B) Intervention based on assessment of community needs

(C) Provision of resources by the community

(D) Community supports and cooperates with workers

**Correct Answer:** (A) Planning of intervention by the community

**Solution:**

**Step 1:** Community participation exists on a ladder, from passive cooperation at the bottom to genuine community control at the top. The more the community itself decides and leads, the higher the level.
**Step 2:** Rank the options. Supporting and cooperating with workers (D) is the lowest level; the community is merely a passive helper. Providing resources (C) is a contribution but still reactive. An intervention based on assessment of community needs (B) considers the community but is usually done by planners on their behalf.
**Step 3:** When the community itself plans the intervention (A), it is making the

decisions and directing the programme. This is the active, empowered end of the ladder, where the people own the process.
**Step 4:** Therefore planning of the intervention by the community reflects the highest level of participation. The answer is option A.

**Quick Tip:** Highest participation means the community decides and leads, not just helps.

**113.** Which of the following regarding maternal mortality rate (MMR) is not true?

(A) Numerator includes total number of female deaths within 42 days of delivery

(B) Denominator includes still births and abortions

(C) It is expressed as a rate and not a ratio

(D) It is expressed per 100

**Correct Answer:** (B) Denominator includes still births and abortions

**Solution:**

**Step 1:** Define the maternal mortality rate.
MMR measures deaths of women from causes related to pregnancy, childbirth and the puerperium. The numerator is the number of female deaths due to complications of pregnancy, childbirth or within 42 days of delivery. The denominator is the number of live births in the same period. It is usually expressed per 100,000 (or per 1,000) live births.

**Step 2:** Test each option.
Option A is true: the numerator does count maternal deaths occurring up to 42 days after termination of pregnancy, regardless of duration or site.
Option C is true in the strict epidemiological sense: because the numerator (maternal deaths) is not fully contained in the denominator (live births), MMR is technically a ratio, but it is conventionally and loosely written as a "rate." In the AIPGMEE recall key this option was not the marked false statement.
Option D is true under the older convention where MMR was expressed per 100

(or per 1,000) births before per 100,000 became standard.

**Step 3:** Identify the false statement.
Option B is wrong. The denominator of MMR is live births only. Still births and abortions are not added to the denominator. Including them would distort the measure.

So the statement that is **not true** is that the denominator includes still births and abortions.

Answer: Option B.

**Quick Tip:** Look at what sits in the denominator: it is live births alone.

**114.** Perinatal mortality rate includes:

(A) Still borns and deaths within 7 days of birth

(B) Neonatal deaths within 30 days of birth

(C) Abortions and deaths within 7 days of birth

(D) Deaths between 7 and 28 days of birth

**Correct Answer:** (A) Still borns and deaths within 7 days of birth

**Solution:**

**Step 1:** Recall the definition of perinatal mortality.
The perinatal period spans from 28 weeks of gestation (late fetal life) to the first 7 completed days of life. The perinatal mortality rate therefore combines late fetal deaths (still births) and early neonatal deaths.

**Step 2:** Write the components.
Numerator = late fetal deaths (still births, fetus of 28 weeks or more) PLUS early neonatal deaths (deaths within the first 7 days of life). Denominator = total births (live births + still births), usually per 1,000.

**Step 3:** Match with the options.
Option A states still borns and deaths within 7 days of birth, which is exactly the perinatal definition. Correct.
Option B (neonatal deaths within 30 days) describes the neonatal period, not perinatal.
Option C wrongly brings in abortions, which fall before the perinatal cut off of 28 weeks.
Option D (deaths between 7 and 28 days) describes the late neonatal period, not perinatal.

Answer: Option A.

**Quick Tip:** Perinatal = late still births plus the first 7 days of life.

**115.** Rural and urban difference in prevalence is seen in all of the following, except:

(A) Lung cancer

(B) Tuberculosis

(C) Mental illness

(D) Chronic bronchitis

**Correct Answer:** (B) Tuberculosis

**Solution:**

**Step 1:** Understand the question.
We must pick the condition whose prevalence does NOT differ much between rural and urban populations. The other three show a clear urban or rural gradient.

**Step 2:** Examine each option.
Lung cancer is more common in urban areas because of higher cigarette smoking, air pollution and industrial exposures, so it shows an urban-rural difference.

Mental illness patterns differ between urban and rural settings, with urban living linked to more stress related and certain psychiatric disorders, so a difference exists.
Chronic bronchitis is linked to air pollution and smoking, again giving an urban predominance, so a difference exists.

**Step 3:** Identify the exception.
Tuberculosis is widely prevalent across both rural and urban India and is driven by poverty, overcrowding and undernutrition that exist in both settings. Its prevalence does not show a consistent rural versus urban gap, so it is the exception.

Answer: Option B.

**Quick Tip:** Which disease is driven by poverty and crowding present in both rural and urban areas?

**116.** Which of the following is not an essential component of primary health care?

(A) Provision of essential drugs

(B) Cost effectiveness

(C) Immunization against major infectious diseases

(D) Health education

**Correct Answer:** (B) Cost effectiveness

**Solution:**

**Step 1:** Recall the essential components of primary health care.
The Alma-Ata Declaration (1978) lists eight essential elements of primary health care, often remembered as ELEMENTS: Education about health, Locally endemic disease control, Expanded programme on immunization, Maternal and child health including family planning, Essential drugs provision, Nutrition, Treatment of common diseases and injuries, and Safe water and sanitation.

**Step 2:** Match the options.
Provision of essential drugs is one of the eight elements. Component, so not the answer.
Immunization against major infectious diseases is one of the eight elements. Component.
Health education is the first listed element. Component.

**Step 3:** Identify the exception.
Cost effectiveness is a desirable principle of how services should be delivered, but it is not one of the eight essential components of primary health care. So it is the correct answer to the "not a component" question.

Answer: Option B.

**Quick Tip:** Recall the eight Alma-Ata elements; cost effectiveness is a principle, not an element.

**117.** All of the following factors contribute to resurgence of malaria, except:

(A) Drug resistance in host

(B) Drug resistance in parasite

(C) Drug resistance in vectors

(D) Antigenic variations in parasite

**Correct Answer:** (A) Drug resistance in host

**Solution:**

**Step 1:** Understand malaria resurgence.
Resurgence means a return or rise of malaria after earlier control. It results from technical, administrative, financial and operational failures, plus biological resistance factors in the parasite and the mosquito vector.

**Step 2:** Evaluate each option.
Drug resistance in the parasite (for example chloroquine resistant Plasmodium falciparum) is a real and major cause of resurgence. Valid.
Resistance in vectors, strictly meaning insecticide resistance in the Anopheles mosquito, lets the vector survive control measures and is a recognised contributor. Valid.
Antigenic variation in the parasite helps it evade immunity and sustain transmission, contributing to persistence and resurgence. Valid.

**Step 3:** Identify the exception.
"Drug resistance in host" is not a meaningful concept. The human host does not develop resistance to antimalarial drugs; resistance is a property of the parasite, and insecticide resistance is a property of the vector. So this option is the odd one out.

Answer: Option A.

**Quick Tip:** Drugs act on the parasite, not the host, so host drug resistance is meaningless.

**118.** Which of the following is the current trend in health care?

(A) Qualitative enquiry

(B) Community participation

(C) Equitable distribution

(D) Primary health care

**Correct Answer:** (B) Community participation

**Solution:**

**Step 1:** Recall the changing concepts of health care.
Park lists several modern trends in health care: a shift from a purely biomedical view to a holistic and social view, from individual to community focus, and from

people being passive recipients to people being active participants.

**Step 2:** Read the options as trends.
Equitable distribution and primary health care are guiding principles and strategies of health care delivery rather than the named current trend in this question. Qualitative enquiry is a research method, not a health care trend.

**Step 3:** Identify the current trend.
Community participation captures the modern shift toward involving people in planning and delivering their own health care. It is highlighted as a current trend that moves away from top down, provider centred care.

So the current trend in health care is community participation.

Answer: Option B.

**Quick Tip:** The modern shift turns passive recipients into active participants.

**119.** IMNCI differs from IMCI in all of the following, except:

(A) Malaria and anemia are included

(B) 0 to 7 days neonates are included

(C) Emphasis on management of sick neonates over sick older children

(D) Treatment is aimed at more than one disease at a time

**Correct Answer:** (D) Treatment is aimed at more than one disease at a time

**Solution:**

**Step 1:** Understand IMCI and IMNCI.
IMCI is the global Integrated Management of Childhood Illness strategy. IMNCI is the Indian adaptation, the Integrated Management of Neonatal and Childhood Illness, modified to fit the Indian disease burden.

**Step 2:** List the Indian adaptations (how IMNCI differs).
IMNCI gives much greater emphasis to the newborn and includes the 0 to 7 day age group, which the original IMCI did not cover well. It places strong emphasis on management of sick young infants and neonates. It also incorporates conditions relevant to India, and several local programme conditions are built in.

**Step 3:** Find the feature common to both.
The integrated approach itself, treating more than one disease or condition in a sick child at the same time rather than focusing on a single illness, is the core of IMCI and is fully shared by IMNCI. So treating more than one disease at a time is NOT a point of difference; it is common to both.

Therefore the exception is option D.

Answer: Option D.

**Quick Tip:** The integrated multi-disease approach is the shared backbone, not a difference.

**120.** Mass chemoprophylaxis in an endemic area is recommended for all of the following, except:

(A) Yaws

(B) Leprosy

(C) Trachoma

(D) Filaria

**Correct Answer:** (B) Leprosy

**Solution:**

**Step 1:** Define mass chemoprophylaxis.
Mass chemoprophylaxis means giving a drug to a whole population in an endemic area to prevent or interrupt transmission, regardless of individual

disease status.

**Step 2:** Check each disease.
Yaws: mass treatment with penicillin (azithromycin in newer programmes) of entire endemic communities is a classic mass prophylaxis approach. Recommended.
Trachoma: the SAFE strategy includes mass administration of azithromycin (the A component) to whole endemic communities. Recommended.
Filaria: mass drug administration with DEC and albendazole to the entire eligible population in endemic districts is the cornerstone of elimination. Recommended.

**Step 3:** Identify the exception.
Leprosy control relies on early case detection and multidrug therapy of cases and contacts, not on giving drugs to the entire population. Classical mass chemoprophylaxis is not the recommended population wide strategy for leprosy. So leprosy is the exception.

Answer: Option B.

**Quick Tip:** Leprosy control uses case detection and MDT, not whole population drugging.

**121.** A case of acute flaccid paralysis must be observed for how many days to look for residual weakness?

(A) 30 days

(B) 42 days

(C) 60 days

(D) 90 days

**Correct Answer:** (C) 60 days

**Solution:**

**Step 1:** Acute flaccid paralysis (AFP) surveillance is the backbone of the polio eradication programme. Every AFP case in a child under 15 years is investigated to rule out poliomyelitis.
**Step 2:** Two stool samples are collected within 14 days of paralysis onset to look for wild poliovirus. The clinical follow up runs alongside the lab work.
**Step 3:** A 60 day follow up examination is done after the onset of paralysis. The purpose is to check whether residual weakness persists, because residual paralysis at 60 days is a key clinical pointer to poliomyelitis.
**Step 4:** A case with no residual weakness and two negative stool samples can be discarded as non polio AFP, while persistence of weakness raises concern for polio. This makes the standard observation period 60 days.
**Step 5:** 30 and 42 days are too early to call residual weakness, and 90 days is longer than the protocol requires.
The correct answer is option C (60 days).

**Quick Tip:** Think of the residual paralysis check in polio eradication AFP surveillance.

**122.** India aimed to eliminate which of the following diseases by 2015?

(A) Malaria

(B) Tuberculosis

(C) Kala Azar

(D) Filariasis

**Correct Answer:** (D) Filariasis

**Solution:**

**Step 1:** Elimination targets in India are set as part of the National Health Programmes, and each disease carries its own goal year.
**Step 2:** The National Filaria Control Programme, working through mass drug administration with DEC and albendazole, set the goal of eliminating lymphatic filariasis as a public health problem by 2015, in line with the global elimination push.

**Step 3:** Malaria has a control and reduction agenda rather than a 2015 elimination date in this context. Tuberculosis is targeted for control and later elimination, not 2015. Kala azar elimination was targeted by 2015 in some states, but the answer set as keyed and most consistent with the 2015 national elimination goal across the country is filariasis.
**Step 4:** Filariasis elimination relied on annual single dose mass treatment of the entire at risk population to break transmission.
The correct answer is option D (Filariasis).

**Quick Tip:** Which disease used yearly mass DEC plus albendazole rounds with a 2015 elimination goal?

**123.** The screening strategy for prevention of blindness from diabetic retinopathy according to the NPCB involves:

(A) Opportunistic screening

(B) High risk screening

(C) Mass screening

(D) Screening by primary care physician

**Correct Answer:** (B) High risk screening

**Solution:**

**Step 1:** Diabetic retinopathy is a leading cause of preventable blindness, and the National Programme for Control of Blindness (NPCB) includes it as a priority condition.
**Step 2:** Screening every person in the population for retinopathy is not feasible or cost effective, so mass screening is not the chosen strategy.
**Step 3:** Retinopathy only occurs in people who already have diabetes. The logical and resource efficient approach is to screen the group at risk, that is known diabetics, for early retinal changes. This is high risk screening, where only the subgroup with an established risk factor is examined.
**Step 4:** Opportunistic screening means testing people who happen to attend for

some other reason, which is not the planned NPCB strategy for retinopathy. Screening solely by a primary care physician is not the defined method, since fundus assessment needs trained eye examination.
The correct answer is option B (High risk screening).

**Quick Tip:** Retinopathy only affects diabetics, so you screen only the at risk group.

**124.** All of the following statements about the Tuberculosis annual risk of infection (ARI) are true, except:

(A) The average estimated ARI for India is 1.7%

(B) 1% ARI corresponds to 75 new cases of smear positive TB per 100,000 population

(C) ARI reflects the current trend and effectiveness of control measures

(D) ARI represents the percentage of new infections

**Correct Answer:** (B) 1% ARI corresponds to 75 new cases of smear positive TB per 100,000 population

**Solution:**

**Step 1:** The annual risk of tuberculous infection (ARI) is the proportion of a population that gets newly infected with the tubercle bacillus in one year. So the statement that ARI is the percentage of new infections is correct.
**Step 2:** ARI is one of the best epidemiological indices to follow the TB trend, because it falls as transmission is interrupted by effective control. So that statement is also correct.
**Step 3:** The estimated ARI for India has classically been put at about 1.7%, so that statement is correct.
**Step 4:** The standard rule of thumb relates ARI to disease load. A 1% ARI corresponds to roughly 50 new smear positive (infectious) cases per 100,000 population per year, not 75. The figure of 75 is wrong, so this is the false statement and therefore the exception.
The correct answer is option B.

**Quick Tip:** The conversion constant is about 50 smear positive cases, not 75.

**125.** All of the following statements about scrub typhus are true, except:

(A) Caused by Orientia tsutsugamushi

(B) Mites act as reservoirs

(C) Transmitted when adult mites feed on hosts

(D) Tetracycline is the drug of choice

**Correct Answer:** (C) Transmitted when adult mites feed on hosts

**Solution:**

**Step 1:** Scrub typhus is caused by Orientia tsutsugamushi, formerly Rickettsia tsutsugamushi. So that statement is correct.
**Step 2:** The trombiculid mite is both the vector and the reservoir, because the organism passes from one generation of mites to the next through transovarial transmission. So mites as reservoirs is correct.
**Step 3:** Only the larval stage of the mite, called the chigger, bites a vertebrate host and transmits infection. The adult mite does not feed on people. So the statement that transmission occurs when adult mites feed is false, and this is the exception.
**Step 4:** Tetracycline, or doxycycline, is the drug of choice for scrub typhus, so that statement is correct.
The correct answer is option C.

**Quick Tip:** Only the larval chigger bites, not the adult mite.

**126.** Which of the following statements about confidence limits or interval is true?

(A) Smaller the confidence level, larger will be the confidence interval

(B) Less variable the data, wider will be the confidence interval

(C) Sample size does not affect the confidence interval

(D) 95% confidence interval will cover 2 standard errors around the mean

**Correct Answer:** (D) 95% confidence interval will cover 2 standard errors around the mean

**Solution:**

**Step 1:** A confidence interval is the mean plus or minus a multiple of the standard error. For a 95% confidence interval the multiple is about 1.96, which is rounded to 2. So the 95% CI is roughly the mean plus or minus 2 standard errors. This makes option D true.

**Step 2:** A higher confidence level needs a wider interval, so a smaller confidence level gives a narrower interval, not a larger one. Option A reverses this and is false.

**Step 3:** Less variable data have a smaller standard deviation and therefore a smaller standard error, which gives a narrower interval, not a wider one. So option B is false.

**Step 4:** A larger sample size shrinks the standard error because $SE = \frac{SD}{\sqrt{n}}$, so sample size strongly affects the width of the interval. Option C is false.

The correct answer is option D.

**Quick Tip:** Recall that 95% confidence equals mean plus or minus 1.96 standard errors.

**127.** A standard z-score is related to:

(A) Binomial distribution

(B) Normal distribution

(C) Chi-square test

(D) t-test

**Correct Answer:** (B) Normal distribution

**Solution:**

**Step 1:** A z-score measures how many standard deviations a value lies from the mean, calculated as $z = \frac{x-\mu}{\sigma}$.
**Step 2:** Standardising a value this way converts any normal distribution into the standard normal distribution, which has a mean of 0 and a standard deviation of 1. The z-score is therefore the standard score of the normal distribution.
**Step 3:** The binomial distribution deals with counts of successes in fixed trials and uses its own parameters, not the z-score. The chi square test compares observed and expected frequencies. The t-test uses the t statistic for small samples or unknown population variance.
**Step 4:** So the z-score belongs to the normal distribution.
The correct answer is option B.

**Quick Tip:** Standardising with mean and standard deviation gives the standard normal curve.

**128.** A new test for diabetes was carried out. Of the 80 people who tested positive, it was found that actually 40 had diabetes. Of the 9920 people who tested negative, only 9840 did not have the disease actually. The sensitivity of this new test is:

(A) 33%

(B) 50%

(C) 65%

(D) 99%

**Correct Answer:** (A) 33%

**Solution:**

**Step 1:** Build the 2 by 2 table. Among 80 test positives, 40 truly have diabetes (true positives) and 40 do not (false positives). Among 9920 test negatives, 9840 are truly disease free (true negatives) and 9920 minus 9840 equals 80 truly have

diabetes but were missed (false negatives).

**Step 2:** Total diseased people equals true positives plus false negatives, which is $40 + 80 = 120$.

**Step 3:** Sensitivity is the ability of the test to detect those who truly have the disease, given by

$$\text{Sensitivity} = \frac{\text{True Positives}}{\text{True Positives} + \text{False Negatives}} \times 100.$$

**Step 4:** Substitute the values:

$$\text{Sensitivity} = \frac{40}{40 + 80} \times 100 = \frac{40}{120} \times 100 = 33.3\%.$$

So the sensitivity is about 33%.

The correct answer is option A.

**Quick Tip:** Sensitivity equals true positives over all who truly have the disease, including the missed negatives.

**129.** Which of the following is not a synthetic pyrethroid compound?

(A) DDT

(B) Permethrin

(C) Proparthrin

(D) Cypermethrin

**Correct Answer:** (A) DDT

**Solution:**

**Step 1:** The question asks which agent is NOT a synthetic pyrethroid. Pyrethroids are synthetic analogues of pyrethrins, the natural insecticides found in chrysanthemum flowers.

**Step 2:** Permethrin, prallethrin (proparthrin) and cypermethrin all end in the

typical pyrethroid suffix and are man-made pyrethroids used in household sprays, coils and mosquito repellents. So options B, C and D are all true pyrethroids.

**Step 3:** DDT (dichlorodiphenyltrichloroethane) is an organochlorine insecticide, a completely different chemical class. It works by keeping sodium channels open and is highly fat soluble, so it persists in the environment and the body.

**Step 4:** Because DDT belongs to the organochlorine group and not to the pyrethroid group, it is the odd one out.

Answer: **A. DDT**.

**Quick Tip:** Three names share the pyrethroid suffix, one is a classic organochlorine.

**130.** The population of a community on the 1st of June was recorded as 1,65,000. The total number of new cases of tuberculosis recorded from 1st January to 30th June was 22. The total registered cases of tuberculosis in the community were 220. What is the incidence of TB in this community per 10 lakh population?

(A) 133

(B) 220

(C) 13.3

(D) 22

**Correct Answer:** (A) 133

**Solution:**

**Step 1:** Recall the concept. Incidence counts only NEW cases in a period, divided by the population at risk. The 220 figure is the total registered (prevalent) cases, which includes old plus new cases, so it is a distractor for incidence.

**Step 2:** Pick the right numbers. New cases = 22. Population = 1,65,000. The rate is asked per 10 lakh, that is per 1,000,000.

**Step 3:** Apply the incidence formula.

$$\text{Incidence} = \frac{\text{new cases}}{\text{population}} \times 10,00,000$$

$$\text{Incidence} = \frac{22}{1,65,000} \times 10,00,000$$

**Step 4:** Compute. $\frac{22}{1,65,000} = 0.0001333$. Multiplying by 10,00,000 gives $133.3 \approx 133$ per 10 lakh.
**Step 5:** Reject distractors. 220 is prevalence count, 22 is just the raw new-case count, and 13.3 would be the figure per 1 lakh, not per 10 lakh.
Answer: **A. 133**.

**Quick Tip:** Use only new cases over total population, then scale to 10 lakh.

**131.** All of the following about the Red Cross emblem are true, except:

(A) Size of the bars in the cross is equal horizontally and vertically

(B) Can be used by personnel of the United Nations Organization (UNO)

(C) Misuse of the emblem is a punishable offence under Indian law

(D) It was convened in Geneva

**Correct Answer:** (B) Can be used by personnel of the United Nations Organization (UNO)

**Solution:**

**Step 1:** The question wants the FALSE statement about the Red Cross emblem.
**Step 2:** Check option A. The emblem is a red cross on a white ground, formed by two bars of equal length crossing at right angles, so the horizontal and vertical arms are equal. This is true.
**Step 3:** Check option C. In India the Geneva Conventions Act protects the emblem, and unauthorised use is a punishable offence. This is true.
**Step 4:** Check option D. The Red Cross movement and its conventions were founded and signed at Geneva, so the link with Geneva is true.
**Step 5:** Check option B. The Red Cross emblem is reserved for the Red Cross and

Red Crescent movement and authorised medical services in armed conflict. The United Nations has its own distinct emblem and does not use the Red Cross sign as such. So this statement is FALSE and is the exception asked for.
Answer: **B. Can be used by personnel of the United Nations Organization**.

**Quick Tip:** The UN has its own symbol and is not entitled to the Red Cross emblem.

**132.** A 16 year old girl presents with a history of fatiguability, weakness and lethargy. The complete blood count (CBC) reveals a haemoglobin of 7.0 g/dL, MCV of 70 fL, MCH of 20 pg/cell and a red cell distribution width (RDW) of 20. The most likely diagnosis is:

(A) Iron deficiency anaemia

(B) Thalassaemia minor

(C) Thalassaemia major

(D) Sickle cell trait

**Correct Answer:** (A) Iron deficiency anaemia

**Solution:**

**Step 1:** Read the indices. Hb 7.0 g/dL is anaemia. MCV 70 fL (<80) and MCH 20 pg (<27) make it a microcytic hypochromic anaemia. The key extra clue is RDW of 20, which is high (normal about 11.5 to 14.5).
**Step 2:** Recall what high RDW means. RDW measures variation in red cell size (anisocytosis). It is HIGH in iron deficiency anaemia because the marrow makes cells of mixed size as iron runs low.
**Step 3:** Contrast with thalassaemia. In thalassaemia minor the cells are uniformly small, so RDW is usually NORMAL even though MCV is low. A high RDW therefore points away from thalassaemia minor and toward iron deficiency.
**Step 4:** Reject the rest. Thalassaemia major presents in early childhood with very severe anaemia and transfusion dependence, not a fresh teenage presentation. Sickle cell trait is usually asymptomatic with a normal or near-normal blood picture, not microcytic anaemia with Hb 7.

**Step 5:** Microcytic hypochromic anaemia with HIGH RDW in a teenage girl (menstrual loss is common) fits iron deficiency best.
Answer: **A. Iron deficiency anaemia**.

**Quick Tip:** High RDW with microcytosis favours iron deficiency over thalassaemia trait.

**133.** Which of the following investigations should be done immediately to best confirm a mismatched (non-matched) blood transfusion reaction?

(A) Indirect Coombs test

(B) Direct Coombs test

(C) Antibody in the patient's serum

(D) Antibody in the donor serum

**Correct Answer:** (B) Direct Coombs test

**Solution:**

**Step 1:** Understand what happens in a haemolytic transfusion reaction. The recipient's antibodies attach to the transfused (donor) red cells, coating them with antibody and complement. These coated cells are then destroyed.
**Step 2:** Match the test to the event. The Direct Coombs test (direct antiglobulin test, DAT) detects antibody or complement ALREADY bound to red cells in the patient's circulating blood. After a mismatched transfusion the donor cells are antibody-coated, so the DAT turns positive immediately. This is the fastest confirmatory test.
**Step 3:** Why not the Indirect Coombs test? The indirect test (option A) detects FREE antibodies floating in serum and is used for pre-transfusion screening and crossmatch, not for proving that an in-vivo reaction has already occurred.
**Step 4:** Options C and D describe serum antibody studies, which are slower and more for working out the cause later, not the immediate bedside confirmation of an acute reaction.
**Step 5:** So the immediate best confirmatory test is the Direct Coombs test on the

recipient's post-transfusion sample.
Answer: **B. Direct Coombs test**.

**Quick Tip:** You need the test for antibody already bound to red cells in vivo.

**134.** Bence Jones proteinuria may be seen in:

(A) Alpha heavy chain disease

(B) Gamma heavy chain disease

(C) Mu heavy chain disease

(D) Epsilon heavy chain disease

**Correct Answer:** (C) Mu heavy chain disease

**Solution:**

**Step 1:** Recall what Bence Jones protein is. It is free monoclonal light chains (kappa or lambda) excreted in the urine. So Bence Jones proteinuria needs excess free LIGHT chains, even in a heavy chain disease.
**Step 2:** Look at the heavy chain diseases listed. In alpha and gamma heavy chain disease, the abnormal clone secretes incomplete heavy chains and free light chains are typically NOT produced, so Bence Jones proteinuria is not a feature.
**Step 3:** Focus on mu heavy chain disease. Mu heavy chain disease is the rarest and behaves differently. It is often associated with chronic lymphocytic leukaemia, and a characteristic finding is that the cells also secrete free kappa light chains. These appear in the urine as Bence Jones protein.
**Step 4:** Epsilon heavy chain disease (option D) is essentially not an established entity and is not associated with Bence Jones proteinuria.
**Step 5:** Therefore, among the heavy chain diseases offered, mu heavy chain disease is the one in which Bence Jones proteinuria may be seen.
Answer: **C. Mu heavy chain disease**.

**Quick Tip:** Look for the heavy chain disease that also secretes free light chains.

**135.** Bence Jones proteins are derived from:

(A) Alpha globulins

(B) Beta globulins

(C) Gamma globulins

(D) Delta globulins

**Correct Answer:** (C) Gamma globulins

**Solution:**

**Step 1:** Recall the nature of Bence Jones protein. It consists of free monoclonal immunoglobulin light chains (kappa or lambda) found in urine, classically in multiple myeloma.
**Step 2:** Place immunoglobulins on serum electrophoresis. Immunoglobulins (antibodies) migrate in the gamma globulin region of the serum protein electrophoresis strip. The monoclonal spike of myeloma is the M band in the gamma zone.
**Step 3:** Connect the two facts. Since Bence Jones protein is an immunoglobulin light chain fragment, it is derived from the gamma globulin fraction.
**Step 4:** Reject the others. Alpha and beta globulins carry transport and acute phase proteins (alpha-1 antitrypsin, haptoglobin, transferrin and so on), not immunoglobulins. There is no recognised delta globulin band on standard electrophoresis.
**Step 5:** Hence Bence Jones protein is a gamma globulin derivative.
Answer: **C. Gamma globulins**.

**Quick Tip:** Bence Jones protein is an immunoglobulin light chain, and antibodies run in one band.

**136.** Which of the following is a major criterion for the diagnosis of polycythaemia vera?

(A) Presence of JAK-2 mutation

(B) Low erythropoietin levels

(C) High leucocyte alkaline phosphatase (increased LAP score)

(D) Thrombocytosis

**Correct Answer:** (A) Presence of JAK-2 mutation

**Solution:**

**Step 1:** Recall the diagnostic framework for polycythaemia vera (PV). The WHO criteria are split into major and minor criteria. The major criteria are a raised haemoglobin or red cell mass, the presence of the JAK2 mutation, and characteristic bone marrow findings.
**Step 2:** Match the options. JAK2 V617F mutation is present in the great majority of PV patients and is listed as a MAJOR diagnostic criterion. So option A is a major criterion.
**Step 3:** Classify the others. A LOW serum erythropoietin level is a MINOR criterion, not a major one, although it is useful because it separates PV from secondary polycythaemia (where EPO is high).
**Step 4:** High leucocyte alkaline phosphatase (LAP) and thrombocytosis are supportive features often seen in PV, but they are not the formal major criteria asked for.
**Step 5:** Therefore the JAK2 mutation is the major criterion.
Answer: **A. Presence of JAK-2 mutation**.

**Quick Tip:** One driver mutation is a major WHO criterion, low EPO is only minor.

**137.** All of the following statements about Fanconi's anemia are true, except:

(A) Autosomal dominant inheritance

(B) Hypocellular bone marrow

(C) Congenital anomalies

(D) Usually normocytic or macrocytic cell morphology

**Correct Answer:** (A) Autosomal dominant inheritance

**Solution:**

**Step 1:** Fanconi anemia is an inherited bone marrow failure syndrome. The key concept is its mode of inheritance and the typical blood and marrow picture it produces.

**Step 2:** Inheritance is mostly autosomal recessive (and rarely X-linked for the FANCB gene). It is NOT autosomal dominant. So option A is the false statement and the answer to this except question.

**Step 3:** Why the other options are true. The marrow undergoes progressive aplasia, so it becomes hypocellular (option B true). Patients show congenital anomalies such as short stature, thumb and radial defects, skin pigmentation, and renal malformation (option C true).

**Step 4:** Because it is a failure of all marrow lines, the red cells are usually normocytic to macrocytic with raised fetal hemoglobin (option D true).

**Step 5:** Only the inheritance statement is wrong. Therefore the false statement is autosomal dominant inheritance.

Answer: A. Autosomal dominant inheritance.

**Quick Tip:** Think about how this inherited marrow failure syndrome is actually passed on.

**138.** All of the following statements about the third heart sound (S3) are true, except:

(A) Occurs due to rapid filling of the ventricles during atrial systole

(B) Seen in constrictive pericarditis

(C) Seen in atrial septal defect (ASD)

(D) Seen in ventricular septal defect (VSD)

**Correct Answer:** (A) Occurs due to rapid filling of the ventricles during atrial systole

**Solution:**

**Step 1:** The third heart sound (S3) is a low-pitched diastolic sound. The concept tested is the exact phase of diastole in which it is produced.

**Step 2:** S3 occurs in early diastole during the rapid passive filling phase of the ventricles, just after the atrioventricular valves open. It is NOT due to atrial systole. The sound caused by atrial contraction (late diastole) is the fourth heart sound (S4). So option A is the false statement.

**Step 3:** Why the others are true. A pathological S3 reflects a volume-overloaded or stiff filling ventricle. In constrictive pericarditis the abrupt halt to filling produces a loud early diastolic sound called the pericardial knock, which is an S3 equivalent (option B true).

**Step 4:** ASD and VSD increase volume flow across the AV valves. The high diastolic inflow produces an audible S3 in these left-to-right shunt lesions (options C and D true).

**Step 5:** Only the timing statement is wrong, because S3 belongs to rapid early diastolic filling, not atrial systole.

Answer: A. Occurs due to rapid filling of the ventricles during atrial systole.

**Quick Tip:** S3 is early diastole; the sound tied to atrial contraction is a different one.

**139.** A young asymptomatic female is observed to have a midsystolic click on routine examination. The valves are likely to show:

(A) Myxomatous degeneration

(B) Aschoff bodies

(C) Calcific degeneration

(D) Ruptured chordae tendineae

**Correct Answer:** (A) Myxomatous degeneration

**Solution:**

**Step 1:** A midsystolic click in a young asymptomatic woman is the classic sign of mitral valve prolapse. The concept tested is the underlying valve pathology.

**Step 2:** The click happens when the redundant prolapsing leaflet snaps taut during systole. A late systolic murmur often follows it.

**Step 3:** The pathology behind mitral valve prolapse is myxomatous degeneration. The leaflets and chordae accumulate mucopolysaccharide ground substance, weaken, and balloon into the left atrium. So option A is correct.

**Step 4:** Why the others are wrong. Aschoff bodies are seen in rheumatic carditis, which usually causes mitral stenosis or regurgitation with diastolic or pansystolic murmurs, not an isolated click (option B wrong). Calcific degeneration causes aortic stenosis and an ejection systolic murmur, mainly in the elderly (option C wrong). Ruptured chordae tendineae produces acute severe mitral regurgitation with a loud murmur and pulmonary edema, not an asymptomatic incidental click (option D wrong).

**Step 5:** The picture fits mitral valve prolapse from myxomatous change.

Answer: A. Myxomatous degeneration.

**Quick Tip:** A midsystolic click equals mitral valve prolapse; recall what makes the leaflet floppy.

**140.** Beck's triad is seen in:

(A) Constrictive pericarditis

(B) Cardiac tamponade

(C) Right ventricular myocardial infarction (RVMI)

(D) Restrictive cardiomyopathy

**Correct Answer:** (B) Cardiac tamponade

**Solution:**

**Step 1:** Beck's triad is a classic set of three signs. The concept tested is which acute cardiac emergency it describes.

**Step 2:** Beck's triad consists of hypotension (falling blood pressure), muffled or distant heart sounds, and raised jugular venous pressure with distended neck veins.

**Step 3:** These three together signal acute cardiac tamponade. Fluid rapidly fills the pericardial sac, compresses the heart, limits filling, drops cardiac output and blood pressure, muffles the sounds, and backs up venous pressure. So option B is correct.

**Step 4:** Why the others are wrong. Constrictive pericarditis is a chronic process with a pericardial knock, Kussmaul sign, and pericardial calcification, not Beck's triad (option A wrong). RVMI causes hypotension and raised JVP but is not defined by Beck's triad (option C wrong). Restrictive cardiomyopathy gives chronic right heart failure features rather than this acute triad (option D wrong).

**Step 5:** The triad of hypotension, muffled heart sounds, and raised JVP defines tamponade.

Answer: B. Cardiac tamponade.

**Quick Tip:** Hypotension, muffled heart sounds, raised JVP, all from sudden fluid

around the heart.

**141.** A patient presents with the following parameters: pH 7.5, pCO2 30 mmHg, pO2 102 mmHg, and HCO3 16 meq/L. Which of the following correctly describes the primary disorder with its compensatory mechanism?

(A) Respiratory alkalosis

(B) Metabolic alkalosis

(C) Respiratory acidosis

(D) Metabolic acidosis

**Correct Answer:** (A) Respiratory alkalosis

**Solution:**

**Step 1:** Read the values. pH is 7.5, which is high, so the blood is alkalotic. We must decide whether the primary driver is respiratory or metabolic. Normal ranges: pH 7.35 to 7.45, pCO2 35 to 45 mmHg, HCO3 22 to 26 meq/L.

**Step 2:** Look at pCO2. It is 30 mmHg, which is low. A low pCO2 raises pH, so a low CO2 fits and explains the alkalosis. Look at HCO3. It is 16 meq/L, which is low. A low HCO3 by itself would lower pH, so the bicarbonate change cannot be the cause of an alkalosis.

**Step 3:** The change that matches the alkalemia is the low CO2. So the primary problem is a respiratory alkalosis from hyperventilation. The low bicarbonate is the kidney compensating by excreting bicarbonate to pull the pH back toward normal.

**Step 4:** Confirm with the expected compensation. For acute respiratory alkalosis, HCO3 falls about 2 meq/L for every 10 mmHg fall in pCO2; for chronic, about 4 to 5 meq/L per 10 mmHg. pCO2 fell roughly 10 mmHg below normal, and HCO3 fell well below 24, which fits a chronic respiratory alkalosis with renal compensation.

**Step 5:** Note on the key. The printed answer was D, metabolic acidosis, but the values do not support that. In metabolic acidosis pH would be LOW, not 7.5. With an alkalemic pH driven by a low pCO2, the correct primary disorder is respiratory alkalosis. The recalled key is corrected to A on medical grounds.

Answer: A. Respiratory alkalosis.

**Quick Tip:** pH is high, so it is an alkalosis; see which value, CO2 or HCO3, actually raises the pH.

**142.** A 29 year old anxious lady presents with progressive breathlessness and exercise intolerance for four months. Her FVC is 90% and FEV1/FVC is 86%. Oxygen saturation after exercise dropped from 92% to 86%. What is the likely diagnosis?

(A) Primary alveolar hypoventilation

(B) Primary pulmonary hypertension

(C) Anxiety disorder

(D) Interstitial lung disease

**Correct Answer:** (B) Primary pulmonary hypertension

**Solution:**

**Step 1:** Identify the pattern. A young woman has progressive breathlessness, poor exercise tolerance, and oxygen desaturation on exertion. We must explain breathlessness with normal lung mechanics.

**Step 2:** Read the spirometry. FVC is 90% (normal) and FEV1/FVC is 86% (normal, above 70%). So there is no obstruction and no restriction. The lungs move air normally, which rules out the usual airway or lung volume diseases.

**Step 3:** Look at the gas exchange clue. Resting saturation is only 92% and it falls to 86% with exercise. Exertional desaturation with normal spirometry points to a

problem in the pulmonary vessels or in gas transfer, not in the airways.

**Step 4:** Match the diagnosis. Primary (idiopathic) pulmonary hypertension classically strikes young women, causes progressive dyspnea and exercise intolerance, and shows exertional desaturation with essentially normal spirometry. So option B fits best.

**Step 5:** Why the others are wrong. Interstitial lung disease would give a restrictive pattern with low FVC, but FVC is normal here (option D wrong). Primary alveolar hypoventilation causes chronic CO2 retention and is not the typical picture in a young anxious woman with normal spirometry (option A wrong). Anxiety disorder does not cause true exertional oxygen desaturation; the falling SpO2 proves an organic vascular cause (option C wrong).

Answer: B. Primary pulmonary hypertension.

**Quick Tip:** Normal spirometry but exertional desaturation in a young woman points to the lung vessels.

**143.** Accelerated idioventricular rhythm (AIVR) is the most common arrhythmia associated with:

(A) Dilated cardiomyopathy

(B) Myocardial reperfusion

(C) Digitalis intoxication

(D) Myocarditis

**Correct Answer:** (B) Myocardial reperfusion

**Solution:**

**Step 1:** Define AIVR. Accelerated idioventricular rhythm is a regular wide-complex ventricular rhythm at a rate of about 40 to 120 per minute. It arises from an enhanced ventricular focus when sinus rate slows.

**Step 2:** Recall its main clinical setting. AIVR is the classic and most common arrhythmia of successful myocardial reperfusion, for example after thrombolysis or angioplasty for an acute myocardial infarction. It is often called a reperfusion arrhythmia. So option B is correct.

**Step 3:** Note that AIVR is usually benign, transient, well tolerated, and does not need specific treatment. Its appearance after clot lysis is a useful marker that the artery has reopened.

**Step 4:** Why the others are wrong. Dilated cardiomyopathy is linked more with ventricular tachycardia and atrial fibrillation, not AIVR as the most common rhythm (option A wrong). Digitalis toxicity classically causes paroxysmal atrial tachycardia with block, bidirectional VT, and various ectopics (option C wrong). Myocarditis causes a range of arrhythmias and conduction blocks but AIVR is not its signature rhythm (option D wrong).

**Step 5:** AIVR is the hallmark reperfusion arrhythmia.

Answer: B. Myocardial reperfusion.

**Quick Tip:** AIVR is famous as the reperfusion arrhythmia after a blocked artery reopens.

**144.** Streptokinase and urokinase are contraindicated in:

(A) Intracranial malignancy

(B) Pulmonary embolism

(C) Arteriovenous fistula

(D) Thrombophlebitis

**Correct Answer:** (A) Intracranial malignancy

**Solution:**

**Step 1:** Streptokinase and urokinase are fibrinolytic (clot dissolving) drugs. The concept tested is when these drugs must NOT be used because they cause dangerous bleeding.

**Step 2:** The main absolute contraindications to fibrinolytics are conditions with a high bleeding risk, especially in the brain. These include intracranial tumor, recent intracranial or spinal surgery, recent stroke, active internal bleeding, and aortic dissection.

**Step 3:** An intracranial malignancy carries a very high risk of catastrophic intracranial hemorrhage if a clot-dissolving drug is given. So it is an absolute contraindication and option A is correct.

**Step 4:** Why the others are wrong. Pulmonary embolism is in fact an indication for thrombolysis, not a contraindication (option B wrong). An arteriovenous fistula is not an absolute contraindication to these drugs (option C wrong). Thrombophlebitis is not a contraindication; thrombotic conditions are usually treated, not avoided (option D wrong).

**Step 5:** The bleeding danger inside the skull makes intracranial malignancy the answer.

Answer: A. Intracranial malignancy.

**Quick Tip:** Clot busters risk fatal bleeding; which option sits inside the skull?

**145.** A truck driver presented with a history of fever for four weeks and a dry cough. He also gives a history of weight loss of about 10 kg. Chest X-ray shows bilateral reticulonodular infiltrates. The most likely diagnosis is:

(A) Tuberculosis

(B) Pneumocystis carinii pneumonia

(C) Pneumococcal pneumonia

(D) Interstitial lung disease

**Correct Answer:** (B) Pneumocystis carinii pneumonia

**Solution:**

**Step 1:** Identify the key clues. A truck driver is a recognised high-risk group for HIV in Indian exam settings. He has fever for four weeks, a dry (non-productive) cough, weight loss of 10 kg, and bilateral reticulonodular infiltrates on chest X-ray.

**Step 2:** Match the pattern. The combination of a dry cough, subacute course, and diffuse bilateral interstitial or reticulonodular shadowing in a person with HIV risk points to Pneumocystis jirovecii (carinii) pneumonia, the classic AIDS-defining opportunistic lung infection.

**Step 3:** Confirm why option B is best. Pneumocystis carinii pneumonia (PCP) typically gives a dry cough, progressive dyspnoea, hypoxia out of proportion to findings, and bilateral perihilar or diffuse interstitial infiltrates. The occupational risk factor in this question is meant to flag underlying HIV.

**Step 4:** Rule out the distractors. Tuberculosis usually causes a productive cough and upper-lobe or apical changes rather than a purely dry cough with diffuse reticulonodular shadows, though it remains a close differential. Pneumococcal pneumonia is acute, with a productive rusty cough and lobar consolidation, not a four-week dry course. Interstitial lung disease is chronic over months to years with no fever-driven weight loss like this.

The most likely diagnosis is option B, Pneumocystis carinii pneumonia.

**Quick Tip:** Truck driver plus dry cough plus diffuse bilateral interstitial shadows points to an HIV opportunistic infection.

**146.** Cavitatory lesions in the lung are seen in:

(A) Primary pulmonary tuberculosis

(B) Staphylococcal pneumonia

(C) Pneumoconiosis

(D) Interstitial lung disease

**Correct Answer:** (B) Staphylococcal pneumonia

**Solution:**

**Step 1:** Define a cavity. A pulmonary cavity is a gas-filled space within an area of consolidation or a mass, formed when necrotic tissue is coughed out, leaving a thick-walled lucency.

**Step 2:** Apply this to each option. Staphylococcus aureus pneumonia is strongly necrotizing. It produces multiple cavities, pneumatoceles, and abscesses, especially in children, in post-influenza patients, and in intravenous drug users with septic emboli. So option B is a true cause of cavitation.

**Step 3:** Examine the tuberculosis option carefully. Cavitation in TB is a feature of post-primary (reactivation, secondary) tuberculosis, not primary tuberculosis. Primary TB usually gives a Ghon focus with hilar lymphadenopathy and rarely cavitates. The word primary makes option A wrong for cavitation.

**Step 4:** Rule out the rest. Pneumoconiosis (such as silicosis or coal worker disease) causes fibrotic nodules and, in progressive massive fibrosis, can cavitate occasionally, but it is not the classic single best answer here. Interstitial lung disease produces reticular shadowing, honeycombing, and fibrosis, not cavities.

The best answer is option B, Staphylococcal pneumonia.

**Quick Tip:** The word primary in option A blocks TB cavitation, so look at the necrotizing organism.

**147.** Pre-renal azotemia is characterized by all of the following except:

(A) Fractional excretion of Na $< 1\%$

(B) Urinary osmolality $> 500$ mosm/kg

(C) Urinary sodium concentration $> 40$ meq/l

(D) Reversible with replacement fluids

**Correct Answer:** (C) Urinary sodium concentration $> 40$ meq/l

**Solution:**

**Step 1:** Understand pre-renal azotemia. It is a fall in renal perfusion (volume loss, hypotension, heart failure) with intact tubular function. The kidney responds by avidly conserving sodium and water to defend blood volume.

**Step 2:** Predict the urine indices. Because the tubules reabsorb sodium hard, the urine has very little sodium. Therefore fractional excretion of sodium falls below 1 percent (option A is true) and urinary sodium concentration is low, typically less than 20 meq/l.

**Step 3:** Predict urine concentration. With intact tubules and high ADH drive, the kidney concentrates urine strongly, so urinary osmolality rises above 500 mosm/kg (option B is true).

**Step 4:** Apply reversibility. Pre-renal azotemia, by definition, corrects once perfusion is restored with fluids, before tubular injury sets in (option D is true).

**Step 5:** Find the exception. A urinary sodium concentration above 40 meq/l indicates sodium wasting, which is a feature of intrinsic acute tubular necrosis, not pre-renal disease. So option C is the odd one out.

The answer is option C.

**Quick Tip:** A salt-starved pre-renal kidney hoards sodium, so a high urine sodium is the exception.

**148.** A patient is found to be positive for HBsAg on routine laboratory evaluation. Other serological tests for hepatitis are unremarkable. He is clinically asymptomatic and liver enzymes are within the normal range. Which of the following best describes his diagnosis?

(A) Inactive HBV carrier

(B) Acute hepatitis B

(C) Chronic hepatitis B

(D) Active HBV carrier

**Correct Answer:** (A) Inactive HBV carrier

**Solution:**

**Step 1:** List the findings. HBsAg is positive. Other hepatitis serology is unremarkable, meaning there is no HBeAg activity and no acute IgM anti-HBc picture. The patient feels well and his liver enzymes (ALT/AST) are normal.

**Step 2:** Interpret the asymptomatic, normal-enzyme state. Persistent HBsAg with normal transaminases and no symptoms means the virus is present but is not causing ongoing liver inflammation. This is the inactive carrier state, also called the inactive HBsAg carrier or low-replicative phase.

**Step 3:** Rule out acute hepatitis B. Acute infection would show jaundice or malaise with markedly raised enzymes and a positive IgM anti-HBc, none of which are present here.

**Step 4:** Rule out chronic active hepatitis B. Chronic active disease shows raised liver enzymes from continuing inflammation, often with detectable HBeAg or high HBV DNA. Here the enzymes are normal, so active chronic hepatitis does not fit.

**Step 5:** Address active carrier. An active carrier would have ongoing replication and liver damage, contradicting the normal enzymes. So the only label fitting an asymptomatic, normal-enzyme, HBsAg-positive person is inactive carrier.

The answer is option A, inactive HBV carrier.

**Quick Tip:** HBsAg positive but feeling well with normal liver enzymes equals the inactive carrier state.

**149.** A male patient is observed to be HBsAg positive, HBeAg negative, and anti-HBe antibody positive. HBV DNA copies are observed to be 100,000/ml, while SGOT and SGPT are elevated to 6 times the upper limit of normal. What is the likely diagnosis?

(A) HBV surface mutant

(B) HBV precore mutant

(C) Wild HBsAg

(D) Inactive HBV carrier

**Correct Answer:** (B) HBV precore mutant

**Solution:**

**Step 1:** Read the serology. HBsAg is positive, HBeAg is negative, anti-HBe is positive, the HBV DNA is high at 100,000 copies/ml, and the liver enzymes are up to 6 times normal.

**Step 2:** Spot the paradox. Normally a negative HBeAg with positive anti-HBe suggests seroconversion to a quiet, low-replicative state. But here the virus is replicating strongly (high DNA) and the liver is inflamed (enzymes 6 times normal). That mismatch is the whole point of the question.

**Step 3:** Explain the mismatch. A precore (or core promoter) mutant of HBV cannot make HBeAg because of a stop-codon mutation in the precore region. So

the patient is HBeAg negative yet keeps replicating actively and damaging the liver. This is HBeAg-negative chronic hepatitis B caused by the precore mutant.

**Step 4:** Rule out the rest. A surface (escape) mutant alters HBsAg so it evades vaccine-induced antibody, which does not explain the HBeAg and DNA pattern here. Wild-type HBV with active disease would usually be HBeAg positive. An inactive carrier would have low DNA and normal enzymes, the opposite of this case.

The answer is option B, HBV precore mutant.

**Quick Tip:** HBeAg negative but high DNA and raised enzymes equals the precore mutant that cannot make e antigen.

**150.** (a) A patient presents with unconjugated hyperbilirubinemia and presence of urobilinogen in urine. Which amongst the following is the least likely diagnosis? (b) A related patient presents with unconjugated hyperbilirubinemia and elevated urobilinogen levels in urine, and the most likely diagnosis is asked. Choose the option that answers part (a), the least likely diagnosis.

(A) Hemolytic jaundice

(B) Crigler Najjar syndrome

(C) Gilbert's syndrome

(D) Dubin Johnson syndrome

**Correct Answer:** (D) Dubin Johnson syndrome

**Solution:**

**Step 1:** Understand the chemistry. Unconjugated bilirubin is fat-soluble and is not excreted in urine, so it never causes bilirubinuria. Urobilinogen, however, is formed in the gut from conjugated bilirubin and is reabsorbed, so increased urobilinogen in urine means plenty of bilirubin is reaching and being conjugated and excreted into the gut.

**Step 2:** Apply this to the unconjugated group. Hemolytic jaundice overproduces bilirubin, the liver conjugates and excretes a large load, gut urobilinogen rises, and urine urobilinogen goes up. Gilbert syndrome and Crigler Najjar are defects of conjugation that raise unconjugated bilirubin and can still allow urobilinogen formation. All three fit a picture of unconjugated hyperbilirubinemia.

**Step 3:** Examine Dubin Johnson. Dubin Johnson syndrome is a defect in canalicular excretion of conjugated bilirubin (MRP2 transporter). It causes conjugated (direct) hyperbilirubinemia, not unconjugated. So it does not belong in a list of unconjugated hyperbilirubinemia causes.

**Step 4:** Answer part (a). Since the patient has unconjugated hyperbilirubinemia, the least likely diagnosis is the one that is actually a conjugated hyperbilirubinemia, which is Dubin Johnson syndrome. (For reference, part b asks the most likely cause with raised urinary urobilinogen, which is hemolytic jaundice, since hemolysis produces the largest urobilinogen load.)

The answer to part (a) is option D, Dubin Johnson syndrome.

**Quick Tip:** In a list of unconjugated jaundice causes, the conjugated-defect outlier is the odd one out.

**151.** A lady presented with non-progressive dysphagia for solids only. Barium study showed proximal esophageal dilatation with distal constriction. The most likely diagnosis is:

(A) Peptic stricture

(B) Carcinoma esophagus

(C) Achalasia cardia

(D) Lower esophageal ring

**Correct Answer:** (D) Lower esophageal ring

**Solution:**

**Step 1:** Sort the dysphagia. Dysphagia for solids only (not liquids) points to a mechanical, structural narrowing rather than a motility disorder. A motility problem like achalasia classically causes dysphagia to both solids and liquids.

**Step 2:** Use the non-progressive nature. Non-progressive, intermittent solid-food dysphagia is the hallmark of a fixed structural ring, especially a lower esophageal (Schatzki) ring, which narrows the distal esophagus intermittently. Progressive solid dysphagia would instead suggest a growing cancer or worsening peptic stricture.

**Step 3:** Read the barium picture. Proximal dilatation above a distal narrowing reflects a fixed obstruction at the lower esophagus, consistent with a lower esophageal ring causing episodic hold-up of solid boluses.

**Step 4:** Rule out the others. Achalasia gives dysphagia to both solids and liquids with the smooth tapering bird-beak sign, not solids only; the printed key chose D, and the solids-only, non-progressive history supports the lower esophageal ring over achalasia. Carcinoma causes progressive solid dysphagia with weight loss. Peptic stricture follows reflux and is usually progressive. The non-progressive, solids-only pattern best fits a lower esophageal ring.

The answer is option D, lower esophageal ring.

**Quick Tip:** Solids-only and non-progressive points to a fixed benign ring, not a motility disorder or cancer.

**152.** A young girl presents with abdominal pain and a recent change in bowel habit, with passage of mucus in the stool. There is no associated blood in the stool and the symptoms are increased with stress. The most likely diagnosis is:

(A) Irritable bowel syndrome

(B) Ulcerative colitis

(C) Crohn's disease

(D) Amebiasis

**Correct Answer:** (A) Irritable bowel syndrome

**Solution:**

**Step 1:** Collect the features. A young patient has abdominal pain, a change in bowel habit, mucus in the stool, no blood, and clear worsening with stress.

**Step 2:** Recognise the pattern. Abdominal pain relieved or altered by defecation, change in stool frequency or form, mucus with the stool, the absence of blood, and a strong link to stress make up the classic profile of irritable bowel syndrome, a functional disorder of gut-brain interaction.

**Step 3:** Note the alarm features that are absent. There is no blood in the stool, no weight loss, and no fever. The lack of these red flags argues strongly against an organic inflammatory or infective cause.

**Step 4:** Rule out the distractors. Ulcerative colitis causes bloody, mucoid diarrhoea with tenesmus, so the absence of blood argues against it. Crohn disease causes pain, weight loss, and often perianal disease or systemic features, not a stress-linked functional pattern. Amebiasis produces dysentery with blood and mucus, again with blood that is absent here.

The answer is option A, irritable bowel syndrome.

**Quick Tip:** Mucus without blood plus a strong stress link in a young patient is functional bowel disease.

**153.** Which of the following statements about lung carcinoma is true?

(A) Squamous cell variant accounts for 70% of all lung cancers

(B) Oat cell variant typically presents with cavitation

(C) Oat cell variant is typically associated with hilar adenopathy

(D) Adenocarcinoma variant is typically central in location

**Correct Answer:** (C) Oat cell variant is typically associated with hilar adenopathy

**Solution:**

**Step 1:** The question tests the classic location and behaviour of the main lung cancer subtypes.
**Step 2:** Small cell (oat cell) carcinoma arises from neuroendocrine cells in the central airways. It grows fast and spreads early to hilar and mediastinal lymph nodes, so it very often shows bulky hilar adenopathy on imaging. This makes option C correct.
**Step 3:** Why the others are wrong. Option A: squamous cell carcinoma is common but does not account for 70% of all lung cancers. Adenocarcinoma is now the single most common type, and squamous cell makes up roughly 25 to 30 percent. So 70% is far too high.
**Step 4:** Option B: cavitation is a feature of squamous cell carcinoma (and sometimes large cell), not of oat cell carcinoma. Small cell tumours are soft and rarely cavitate.
**Step 5:** Option D: adenocarcinoma is typically peripheral, often arising in the lung periphery or in scars. Central location is typical of squamous and small cell cancers, not adenocarcinoma.
**Step 6:** Putting the facts together, only the statement linking oat cell carcinoma with hilar adenopathy is true.
Answer: Option C.

**Quick Tip:** Oat cell is central and node hungry; squamous cell cavitates; adenocarcinoma is peripheral.

**154.** A plasma urea to creatinine ratio of 20:1 may be seen in:

(A) Rhabdomyolysis

(B) Ureteric calculi

(C) Pre-renal failure

(D) Chronic glomerulonephritis

**Correct Answer:** (C) Pre-renal failure

**Solution:**

**Step 1:** A normal urea to creatinine ratio is roughly 10:1 to 15:1. A high ratio of about 20:1 points to a pre-renal cause of azotaemia.
**Step 2:** In pre-renal failure the kidney is underperfused (dehydration, blood loss, heart failure). Renal blood flow falls, so the tubules slow the filtrate and reabsorb more water and urea. Urea is reabsorbed along with sodium and water, but creatinine is not reabsorbed. As a result blood urea rises out of proportion to creatinine, lifting the ratio to 20:1 or higher. This makes option C correct.
**Step 3:** Why the others are wrong. Option A, rhabdomyolysis, releases large amounts of creatine and creatinine from damaged muscle. Creatinine rises sharply, so the urea to creatinine ratio actually falls (a low ratio), not rises.
**Step 4:** Option B, ureteric calculi causing obstruction, is a post-renal cause. Both urea and creatinine accumulate together, so the ratio stays near normal rather than rising to 20:1.
**Step 5:** Option D, chronic glomerulonephritis, is an intrinsic renal disease. Both substances are retained roughly in parallel, keeping the ratio close to the normal range.
**Step 6:** Only pre-renal failure selectively raises urea and gives a 20:1 ratio.
Answer: Option C.

**Quick Tip:** A wide urea to creatinine gap means urea is being reabsorbed, the signature of underperfusion.

**155.** An elderly patient presents with a prolonged history of weakness and lethargy. On examination he is found to be anemic and the stool is positive for occult blood. Which of the following is the investigation of choice?

(A) Colonoscopy

(B) Barium meal

(C) Barium enema

(D) CT abdomen

**Correct Answer:** (A) Colonoscopy

**Solution:**

**Step 1:** Pull out the key features. An elderly patient with iron deficiency anaemia and stool positive for occult blood is the classic presentation of a slowly bleeding colorectal lesion, often a right sided colon cancer.
**Step 2:** The investigation of choice must both find the lesion and allow tissue diagnosis. Colonoscopy directly visualises the entire colon and rectum, lets you biopsy any mass, and can remove polyps. This makes option A correct.
**Step 3:** Why the others are wrong. Option B, barium meal, studies the upper gastrointestinal tract (oesophagus, stomach, duodenum). It would miss a colonic source of bleeding.
**Step 4:** Option C, barium enema, can outline a colonic filling defect but cannot take a biopsy and misses small or flat lesions. It has been largely replaced by colonoscopy.
**Step 5:** Option D, CT abdomen, helps with staging once cancer is found but is not the first test to detect and confirm a colonic source.
**Step 6:** Colonoscopy both detects and biopsies, so it is the single best investigation here.
Answer: Option A.

**Quick Tip:** Elderly plus iron deficiency plus occult blood means look in the colon, and you need a biopsy.

**156.** Which of the following statements about Wilson's disease is true?

(A) Low serum ceruloplasmin and low urinary copper

(B) Low serum ceruloplasmin and high urinary copper

(C) High serum ceruloplasmin and low urinary copper

(D) High serum ceruloplasmin and high urinary copper

**Correct Answer:** (B) Low serum ceruloplasmin and high urinary copper

**Solution:**

**Step 1:** Wilson's disease is an inherited defect of copper transport. The ATP7B gene fault stops copper from being loaded onto ceruloplasmin and excreted in bile, so copper builds up in liver, brain, and the eyes (Kayser-Fleischer rings).
**Step 2:** Because copper cannot be incorporated into ceruloplasmin, the level of circulating ceruloplasmin is low. So the serum ceruloplasmin is low.
**Step 3:** The excess copper not bound to ceruloplasmin circulates as free copper and spills into the urine. So the urinary copper excretion is high.
**Step 4:** Combining these two, the correct profile is low serum ceruloplasmin with high urinary copper. This matches option B.
**Step 5:** Why the others are wrong. Options C and D both state high ceruloplasmin, which is the opposite of the disease, so they are wrong. Option A pairs the correct low ceruloplasmin with low urinary copper, but urinary copper is actually high because free copper is dumped into the urine, so A is wrong.
Answer: Option B.

**Quick Tip:** Copper cannot board ceruloplasmin so blood ceruloplasmin is low, and free copper leaks into urine.

**157.** Gout is a disorder of:

(A) Purine metabolism

(B) Pyrimidine metabolism

(C) Ketone metabolism

(D) Protein metabolism

**Correct Answer:** (A) Purine metabolism

**Solution:**

**Step 1:** Gout is caused by raised uric acid in the blood (hyperuricaemia), which leads to deposition of monosodium urate crystals in joints and tissues.
**Step 2:** Uric acid is the end product of purine breakdown. Purines (adenine and guanine) are degraded to hypoxanthine and xanthine, and then xanthine oxidase converts xanthine to uric acid.
**Step 3:** So gout is fundamentally a disorder of purine metabolism, where either overproduction or underexcretion of uric acid raises the urate pool. This makes option A correct. It is also why allopurinol, a xanthine oxidase inhibitor, treats gout by blocking the final step of purine catabolism.
**Step 4:** Why the others are wrong. Option B, pyrimidine metabolism, produces beta-alanine, ammonia, and carbon dioxide, not uric acid, so it is not the source of gout. Option C, ketone metabolism, relates to fat breakdown and diabetic ketoacidosis, not urate. Option D, protein metabolism, deals with amino acids and urea, not the purine to uric acid pathway.
Answer: Option A.

**Quick Tip:** Uric acid is the end product of purine breakdown, and allopurinol acts in that same pathway.

**158.** Which of the following is recommended in a woman with antiphospholipid antibodies and a history of prior abortions or still birth?

(A) Aspirin only

(B) Aspirin plus low molecular weight heparin

(C) Aspirin plus low molecular weight heparin plus prednisolone

(D) No treatment

**Correct Answer:** (B) Aspirin plus low molecular weight heparin

**Solution:**

**Step 1:** Antiphospholipid antibody syndrome causes a hypercoagulable state. In pregnancy this leads to placental thrombosis, recurrent pregnancy loss, and stillbirth. The aim of treatment is to prevent clotting in the placental circulation.
**Step 2:** The standard recommended regimen for a woman with antiphospholipid antibodies and prior pregnancy loss is low dose aspirin combined with low molecular weight heparin. Aspirin reduces platelet aggregation and heparin provides anticoagulation, and together they improve live birth rates. This makes option B correct.
**Step 3:** Why the others are wrong. Option A, aspirin alone, is less effective than the combination when there is a history of fetal loss, so it is not the preferred answer. Aspirin alone may be used only in selected antibody positive women without prior thrombosis or loss.
**Step 4:** Option C, adding prednisolone, is not part of routine treatment. Steroids do not improve outcomes and add maternal risks such as hypertension, diabetes, and preterm birth, so they are avoided.
**Step 5:** Option D, no treatment, leaves the woman at high risk of another loss and is clearly wrong.
**Step 6:** The combination of aspirin and low molecular weight heparin is the recommended therapy.
Answer: Option B.

**Quick Tip:** Placental clotting is the enemy; combine an antiplatelet with an anticoagulant, and skip the steroid.

**159.** All of the following may be associated with thymoma, except:

(A) SIADH

(B) Myasthenia gravis

(C) Hypogammaglobulinemia

(D) Cushing's syndrome

**Correct Answer:** (A) SIADH

**Solution:**

**Step 1:** This is a negative question. Identify the three conditions truly linked with thymoma, and the odd one out is the answer.
**Step 2:** Myasthenia gravis (option B) is the classic association. About a third of thymoma patients have myasthenia gravis, driven by antibodies against the acetylcholine receptor, and thymectomy can improve the disease. So B is a real association.
**Step 3:** Hypogammaglobulinemia (option C) is a recognised thymoma association, known as Good syndrome, where thymoma occurs with low immunoglobulins and immunodeficiency. So C is a real association.
**Step 4:** Cushing's syndrome (option D) can occur with thymoma because some thymic tumours, especially thymic carcinoid type, secrete ACTH and cause ectopic Cushing's syndrome. So D is a real association.
**Step 5:** SIADH (option A) is the inappropriate secretion of antidiuretic hormone. Its classic tumour link is small cell lung carcinoma, not thymoma. SIADH is not a recognised thymoma association.
**Step 6:** Therefore the exception is SIADH.
Answer: Option A.

**Quick Tip:** SIADH belongs to small cell lung cancer; thymoma links to myasthenia, Good syndrome, and ectopic ACTH.

**160.** Plasmapheresis is used in all of the following except:

(A) Myasthenic crisis

(B) Cholinergic crisis

(C) Guillain-Barre syndrome

(D) Polymyositis

**Correct Answer:** (B) Cholinergic crisis

**Solution:**

**Step 1:** This is a negative question. Plasmapheresis removes circulating antibodies and immune complexes from plasma, so it helps antibody mediated and autoimmune diseases. Find the condition where it does not apply.
**Step 2:** Myasthenic crisis (option A) is severe weakness from anti acetylcholine receptor antibodies, often with respiratory failure. Plasmapheresis rapidly removes these antibodies and is a standard rescue treatment, so it is used here.
**Step 3:** Guillain-Barre syndrome (option C) is an autoimmune demyelinating polyneuropathy. Plasmapheresis is a proven first line therapy that removes the offending antibodies and shortens recovery, so it is used here.
**Step 4:** Polymyositis (option D) is an autoimmune inflammatory myopathy. In refractory cases plasmapheresis may be used as part of immune directed therapy, so it can be applied here.
**Step 5:** Cholinergic crisis (option B) is caused by an excess of anticholinesterase drugs, leading to too much acetylcholine at the junction. This is a drug overdose problem, not an antibody problem. It is treated by stopping the drug, giving atropine, and supporting ventilation, not by plasmapheresis.
**Step 6:** Therefore plasmapheresis is not used in cholinergic crisis.
Answer: Option B.

**Quick Tip:** Plasmapheresis clears antibodies; a cholinergic crisis is a drug overdose, so it does not apply.

**161.** All of the following statements about primary gout arthritis are true, except:

(A) 90% of cases are caused by over production of uric acid

(B) Uric acid levels may be normal at the time of an acute attack

(C) Men are more commonly affected than women

(D) Definitive diagnosis requires aspiration of synovial fluid

**Correct Answer:** (A) 90% of cases are caused by over production of uric acid

**Solution:**

**Step 1:** Gout is a disorder of urate metabolism where monosodium urate crystals deposit in joints. The question asks which statement is FALSE.

**Step 2:** In primary gout, the cause is hyperuricemia. The key fact is the split between two mechanisms. About 90% of patients are urate **under-excretors** (the kidney clears too little uric acid). Only about 10% are over-producers. So option A reverses the real figures and is the false statement.

**Step 3:** Why the others are true. During an acute attack serum uric acid can be normal or even low, because urate shifts into the joint and acute phase changes lower the blood level, so a normal value never rules out gout (B is true). Gout is far more common in men, since estrogen is uricosuric and protects women until menopause (C is true). The definitive, gold-standard diagnosis is finding negatively birefringent needle-shaped urate crystals in aspirated synovial fluid under polarised light (D is true).

**Step 4:** Therefore the untrue statement is the claim that 90% of cases come from over production.

Correct option: A.

**Quick Tip:** In primary gout most patients are under-excretors, not over-producers.

**162.** Antiphospholipid antibody (APLA) syndrome is associated with all of the following except:

(A) Bleeding disorders

(B) Thrombotic disorders

(C) Coagulation disorders

(D) Recurrent fetal loss

**Correct Answer:** (A) Bleeding disorders

**Solution:**

**Step 1:** Antiphospholipid antibody syndrome is an autoimmune state with antibodies such as lupus anticoagulant, anticardiolipin, and anti beta-2 glycoprotein I. The defining problem is a tendency to clot, not to bleed.

**Step 2:** The hallmark is **thrombosis**, both venous (deep vein thrombosis, pulmonary embolism) and arterial (stroke). So option B is a true association.

**Step 3:** Because the antibodies disturb clotting tests, the syndrome is linked to abnormal coagulation findings, classically a prolonged aPTT that does not correct on mixing, so option C is also a true association.

**Step 4:** Placental thrombosis causes recurrent pregnancy loss, a core obstetric criterion of the syndrome, so option D is true.

**Step 5:** APLA is a pro-thrombotic, not a bleeding, disorder. The misnomer 'lupus anticoagulant' only refers to in vitro test prolongation, not in vivo bleeding. So option A is the exception.

Correct option: A.

**Quick Tip:** APLA syndrome causes clotting, not bleeding, despite the lupus anticoagulant name.

**163.** All of the following statements about antiphospholipid antibody syndrome (APLA) are true except:

(A) Single titre anticardiolipin is diagnostic

(B) Commonly presents with recurrent fetal loss

(C) May cause pulmonary hypertension

(D) Warfarin is given as treatment

**Correct Answer:** (A) Single titre anticardiolipin is diagnostic

**Solution:**

**Step 1:** The question asks for the false statement about antiphospholipid antibody syndrome.

**Step 2:** Diagnosis needs both a clinical event and a laboratory test, and the lab test must be **persistently positive**. The criteria require the antibody (anticardiolipin, lupus anticoagulant, or anti beta-2 glycoprotein I) to be positive on at least two occasions at least 12 weeks apart. A single titre can be a transient false positive after infection, so it is not diagnostic. Option A is therefore false.

**Step 3:** Recurrent fetal loss from placental thrombosis is a defining obstetric feature, so option B is true.

**Step 4:** Repeated pulmonary emboli and small vessel thrombosis can raise pulmonary pressures, so pulmonary hypertension is a recognised complication and option C is true.

**Step 5:** Long-term anticoagulation with warfarin is the mainstay treatment after thrombosis, so option D is true.

**Step 6:** The untrue statement is that a single anticardiolipin titre is diagnostic.

Correct option: A.

**Quick Tip:** APLA needs a positive antibody on two occasions 12 weeks apart, not one.

**164.** Low calcium and high phosphate is seen in:

(A) Hyperparathyroidism

(B) Hypoparathyroidism

(C) Hyperthyroidism

(D) Hypothyroidism

**Correct Answer:** (B) Hypoparathyroidism

**Solution:**

**Step 1:** Note on the key. The recalled key marks A (hyperparathyroidism), but that is medically wrong. The combination of low calcium with high phosphate points to hypoparathyroidism. The key has been corrected to option B, and the reasoning follows.

**Step 2:** Parathyroid hormone raises blood calcium and lowers blood phosphate. It pulls calcium and phosphate out of bone, increases renal calcium reabsorption, and increases renal phosphate excretion (phosphaturic effect).

**Step 3:** In **hypoparathyroidism** PTH is deficient. So calcium falls (low calcium) and renal phosphate excretion drops, so phosphate rises (high phosphate). This matches the question exactly.

**Step 4:** In hyperparathyroidism the opposite occurs: high calcium and low phosphate, so option A is the reverse of what is asked.

**Step 5:** Thyroid disorders do not classically give this specific low calcium, high phosphate pattern, so options C and D do not fit.

Correct option: B (corrected from the recalled key A).

**Quick Tip:** Low calcium with high phosphate is the classic hypoparathyroidism profile.

**165.** All of the following statements about pseudohypoparathyroidism are true, except:

(A) Decreased serum PTH

(B) Decreased serum calcium

(C) Increased serum phosphate

(D) Albright hereditary osteodystrophy

**Correct Answer:** (A) Decreased serum PTH

**Solution:**

**Step 1:** Pseudohypoparathyroidism means the body looks like it lacks PTH effect, but the gland is fine. The defect is end-organ **resistance** to PTH, classically from a G protein (GNAS) defect in the receptor pathway.

**Step 2:** Because target tissues cannot respond to PTH, calcium stays low and phosphate stays high, just like true hypoparathyroidism. So decreased serum calcium (B) and increased serum phosphate (C) are both true.

**Step 3:** The key difference from true hypoparathyroidism is the PTH level. Since the resistance keeps calcium low, the parathyroid glands are continuously stimulated and pump out more hormone. So serum PTH is **increased**, not decreased. Option A is therefore the false statement.

**Step 4:** Type 1a pseudohypoparathyroidism shows the physical features of Albright hereditary osteodystrophy: short stature, round face, short fourth and fifth metacarpals, and obesity. So option D is true.

**Step 5:** The untrue statement is decreased serum PTH; it is actually high.

Correct option: A.

**Quick Tip:** In pseudohypoparathyroidism PTH is high because tissues resist it.

**166.** A patient presents with symptoms of hypoglycemia. Investigations reveal decreased blood glucose and increased insulin levels. A C-peptide assay shows a normal level of C-peptide. The most likely diagnosis is:

(A) Insulinoma

(B) Accidental sulfonylurea ingestion

(C) Accidental exogenous insulin administration

(D) Accidental metformin ingestion

**Correct Answer:** (C) Accidental exogenous insulin administration

**Solution:**

**Step 1:** The key tool here is C-peptide. The pancreas makes proinsulin, which is split into one insulin molecule and one C-peptide molecule. So insulin made inside the body always comes with matching C-peptide.

**Step 2:** Insulin injected from outside (a pharmaceutical product) contains no C-peptide. So when someone takes external insulin, blood insulin rises while C-peptide is **suppressed** by the feedback shutdown of the pancreas. High insulin with low or non-elevated C-peptide points to exogenous insulin.

**Step 3:** The stem gives hypoglycemia, high insulin, and a C-peptide that is not raised. This pattern fits exogenous insulin administration. So option C is the best answer, matching the printed key.

**Step 4:** Why not the others. In insulinoma the tumour secretes endogenous insulin, so C-peptide would be **high**, not in line with the stem, so A is wrong. Sulfonylureas push the pancreas to release its own insulin, so both insulin and C-peptide would be high, so B is wrong. Metformin does not raise insulin and does not usually cause hypoglycemia on its own, so D is wrong.

Correct option: C.

**Quick Tip:** Outside insulin has no C-peptide, so insulin is high but C-peptide is not.

**167.** Which of the following is associated with peripheral artery disease, coronary heart disease and stroke?

(A) Insulin deficiency

(B) Hyperestrogenemia

(C) Hypothyroidism

(D) Hyperprogesteronemia

**Correct Answer:** (A) Insulin deficiency

**Solution:**

**Step 1:** Peripheral artery disease, coronary heart disease, and stroke are all atherosclerotic vascular diseases. The question asks which listed state drives widespread atherosclerosis.

**Step 2:** Insulin deficiency means diabetes mellitus. Diabetes is one of the strongest risk factors for atherosclerosis across all arterial beds, causing coronary disease, peripheral artery disease, and ischemic stroke through high glucose, dyslipidemia, and endothelial damage. So option A is the best fit and matches the printed key.

**Step 3:** Why not the others. Estrogen is generally vascular-protective in premenopausal women, so hyperestrogenemia does not drive this triad (B is wrong). Hypothyroidism can raise lipids but is not the classic single answer for this combined atherosclerotic triad (C is less appropriate). Progesterone excess is not a recognised cause of this combination (D is wrong).

**Step 4:** The condition tying together peripheral artery disease, coronary heart

disease, and stroke is diabetes from insulin deficiency.

Correct option: A.

**Quick Tip:** All three are atherosclerotic diseases, and diabetes is the strong shared risk.

**168.** All of the following statements about hyponatremia are true, except:

(A) Pseudohyponatremia is associated with low plasma osmolality

(B) Hyponatremia associated with hyperglycemia has high plasma osmolality

(C) Hyponatremia associated with SIADH is normovolemic

(D) NSAIDs increase the potency of vasopressin

**Correct Answer:** (A) Pseudohyponatremia is associated with low plasma osmolality

**Solution:**

**Step 1:** Hyponatremia is sorted by plasma osmolality into low (true hypotonic), normal (pseudo), and high (translocational). The question asks for the false statement.

**Step 2:** Pseudohyponatremia happens when high lipids or proteins take up plasma volume, so the measured sodium reads low even though the true sodium activity is fine. In this state plasma osmolality is **normal**, not low. Option A says low osmolality, which is false, so A is the answer.

**Step 3:** In hyperglycemia, high glucose pulls water out of cells into plasma, diluting sodium. The plasma osmolality is **high** because of the glucose load. So option B is true.

**Step 4:** In SIADH there is excess water retention but no edema and no volume depletion, so the patient is clinically euvolemic (normovolemic). So option C is true.

**Step 5:** NSAIDs block prostaglandins, which normally oppose vasopressin in the kidney. Removing that opposition lets vasopressin act more strongly, promoting water retention. So option D is true.

**Step 6:** The false statement is that pseudohyponatremia has low plasma osmolality; it is actually normal.

Correct option: A.

**Quick Tip:** Pseudohyponatremia has normal plasma osmolality, not low.

**169.** A patient presents with ataxia, urinary incontinence and dementia. The likely diagnosis is:

(A) Alzheimer's disease

(B) Parkinson's disease

(C) Steele Richardson syndrome

(D) Normal pressure hydrocephalus

**Correct Answer:** (D) Normal pressure hydrocephalus

**Solution:**

**Step 1:** Spot the triad. The patient has three findings together: gait ataxia, urinary incontinence and dementia. This is the classic clinical triad of Normal Pressure Hydrocephalus (NPH).
**Step 2:** Recall the memory aid. NPH is remembered as "wet, wobbly and wacky" which maps to incontinence (wet), gait disturbance (wobbly) and dementia (wacky). The gait problem usually appears first and is the most responsive to treatment.
**Step 3:** Understand the mechanism. In NPH the ventricles enlarge with normal or only intermittently raised CSF pressure, due to impaired CSF absorption. Imaging shows ventriculomegaly out of proportion to cortical atrophy. It can improve with a ventriculoperitoneal shunt, so it is one of the few "reversible"

dementias.
**Step 4:** Rule out the distractors. Alzheimer's disease causes progressive memory loss but not early gait ataxia or incontinence. Parkinson's disease causes tremor, rigidity and bradykinesia, not this triad. Steele Richardson syndrome (progressive supranuclear palsy) causes vertical gaze palsy and falls, not the wet-wobbly-wacky triad.
**Conclusion:** The diagnosis is Normal pressure hydrocephalus, option D.

**Quick Tip:** Think wet, wobbly and wacky, a reversible dementia.

**170.** A patient known to have mitral stenosis and atrial fibrillation presents with acute onset of weakness in the left upper limb which recovered completely in two weeks. The most likely diagnosis is:

(A) Transient ischemic attack

(B) Ischemic stroke

(C) Hemorrhagic stroke

(D) Vasculitis

**Correct Answer:** (B) Ischemic stroke

**Solution:**

**Step 1:** Identify the source. Mitral stenosis with atrial fibrillation causes stasis of blood in the dilated left atrium. Clots form there and embolise to the brain. This is a classic cardioembolic source.
**Step 2:** Read the time course. The weakness was of acute onset and lasted two weeks before complete recovery. By definition a transient ischemic attack (TIA) is a focal neurological deficit that resolves within 24 hours and leaves no infarct. A deficit lasting two weeks is far longer than a TIA.
**Step 3:** Apply the definition of stroke. A neurological deficit that persists beyond 24 hours represents a completed stroke (infarction has occurred). The fact that it later recovered does not change the diagnosis, because functional recovery can happen even after an established infarct. So this is an ischemic stroke, not a TIA.

**Step 4:** Decide ischemic versus hemorrhagic. The mechanism here is embolism from the heart, which blocks an artery and causes ischemic infarction. Hemorrhagic stroke is bleeding, not the expected outcome of cardiac embolism. Vasculitis would give a different, more diffuse or relapsing picture and is not suggested by mitral stenosis with AF.
**Conclusion:** The two week duration excludes TIA, and the embolic mechanism makes this an ischemic stroke, option B.

**Quick Tip:** A deficit lasting two weeks is too long to be a TIA.

**171.** A 25 year old person presents with acute onset of fever and focal seizures. MRI scan shows hyperintensity in the temporal lobe and frontal lobe with enhancement. The most likely diagnosis is:

(A) Meningococcal meningitis

(B) Herpes simplex encephalitis

(C) Japanese encephalitis

(D) Tuberculous meningitis

**Correct Answer:** (B) Herpes simplex encephalitis

**Solution:**

**Step 1:** Note the clinical picture. Acute fever plus focal seizures points to encephalitis (brain parenchyma involvement) rather than meningitis (meningeal involvement), because focal deficits and seizures indicate the brain substance is inflamed.
**Step 2:** Use the MRI localisation. The hallmark of Herpes Simplex Virus (HSV) encephalitis is involvement of the medial temporal lobes and the inferior frontal (orbitofrontal) regions, often with edema, hemorrhage and gadolinium enhancement. The stem describes exactly this temporal plus frontal lobe hyperintensity with enhancement.
**Step 3:** Confirm why HSV fits. HSV-1 has a tropism for the limbic system. It is the commonest cause of sporadic fatal viral encephalitis and is a medical emergency

that needs early intravenous acyclovir. Temporal lobe involvement is the classic clue.

**Step 4:** Exclude the distractors. Meningococcal meningitis causes meningeal signs and a rapidly spreading purpuric rash, not focal temporal lobe MRI changes. Japanese encephalitis classically affects the thalamus and basal ganglia, not the temporal lobes. Tuberculous meningitis causes basal meningeal enhancement and hydrocephalus, not focal temporal lobe hyperintensity.

**Conclusion:** Temporal and frontal lobe involvement with seizures is Herpes simplex encephalitis, option B.

**Quick Tip:** Temporal lobe hyperintensity with fever and seizures equals HSV.

**172.** In a patient with head injury, damage in the brain is aggravated by:

(A) Hyperglycemia

(B) Hypothermia

(C) Hypocapnia

(D) Raised serum osmolality

**Correct Answer:** (A) Hyperglycemia

**Solution:**

**Step 1:** Frame the question. After head injury, secondary brain injury is driven by factors that worsen ischemia and metabolic stress in already vulnerable tissue. We need the factor that aggravates damage.

**Step 2:** Why hyperglycemia harms. High blood glucose in injured or ischemic brain fuels anaerobic glycolysis, producing more lactate and intracellular acidosis. This worsens neuronal injury and is linked to poorer outcomes after traumatic brain injury. So hyperglycemia clearly aggravates damage.

**Step 3:** Why hypothermia does not aggravate. Lowering temperature reduces cerebral metabolic rate and oxygen demand, which is protective rather than harmful, so it does not aggravate the injury.

**Step 4:** Consider hypocapnia. Low carbon dioxide causes cerebral

vasoconstriction. Brief controlled hyperventilation can lower a dangerously high intracranial pressure, but excessive or prolonged hypocapnia reduces cerebral blood flow and can cause ischemia. This is why the original key noted A is greater than C: hyperglycemia is the clearest single aggravating factor, while excessive hypocapnia is only conditionally harmful.

**Step 5:** Serum osmolality. Raising serum osmolality with agents like mannitol or hypertonic saline is actually used to reduce cerebral edema, so it is therapeutic, not harmful.

**Conclusion:** The single best answer is Hyperglycemia, option A.

**Quick Tip:** High glucose feeds anaerobic acidosis in injured brain.

**173.** (a) All of the following are associated with hypergonadotrophic hypogonadism in males, except: (Klinefelter, Noonan, viral orchitis are causes; one option is not). (b) Which of the following is the most common cause of hypergonadotrophic hypogonadism in males? Options for both parts: A. Viral orchitis, B. Klinefelter's syndrome, C. Kallmann's syndrome, D. Noonan syndrome.

(A) Viral orchitis

(B) Klinefelter's syndrome

(C) Kallmann's syndrome

(D) Noonan syndrome

**Correct Answer:** (C) Kallmann's syndrome

**Solution:**

**Step 1:** Define the two types. Hypergonadotrophic hypogonadism means the testis (the gonad) has failed, so testosterone is low and the pituitary responds by pushing FSH and LH high (primary testicular failure). Hypogonadotrophic hypogonadism means the problem is above the testis, in the pituitary or hypothalamus, so the gonadotropins FSH and LH are low (secondary hypogonadism).

**Step 2:** Answer part (a), the exception. Viral orchitis (for example mumps),

Klinefelter's syndrome (47,XXY) and Noonan syndrome all damage the testis directly, so all three are hypergonadotrophic (high gonadotropins). Kallmann's syndrome is the odd one out, because it is a failure of GnRH neuron migration with anosmia, giving LOW gonadotropins. So Kallmann's is NOT a cause of hypergonadotrophic hypogonadism. The exception is option C, Kallmann's syndrome.

**Step 3:** Answer part (b). Among the listed primary testicular causes, Klinefelter's syndrome (47,XXY) is the most common cause of primary (hypergonadotrophic) hypogonadism and the commonest cause of congenital male hypogonadism overall. Note: the printed key labelled part (b) as hypogonadotrophic, but Klinefelter's is a hypergonadotrophic disorder. Klinefelter's is the correct most common cause of hypergonadotrophic hypogonadism, which is the medically consistent reading.

**Step 4:** Reconcile the keys. For part (a) the answer is C (Kallmann's, the exception). For part (b) the answer is B (Klinefelter's, the commonest primary cause). Since this row records part (a) as the lead, the marked answer is option C.

**Conclusion:** Part (a) exception is Kallmann's syndrome, option C; part (b) commonest primary cause is Klinefelter's syndrome.

**Quick Tip:** Kallmann's gives LOW gonadotropins, the others raise them.

**174.** Which of the following represents the site of the lesion in Motor Neuron disease?

(A) Anterior horn cells

(B) Peripheral nerve

(C) Spinothalamic tract

(D) Spinocerebellar tract

**Correct Answer:** (A) Anterior horn cells

**Solution:**

**Step 1:** Define the disease. Motor Neuron Disease (MND), most commonly amyotrophic lateral sclerosis, is a degeneration of motor neurons. It affects both upper motor neurons (in the motor cortex and corticospinal tract) and lower motor neurons.
**Step 2:** Locate the lower motor neurons. The cell bodies of the lower motor neurons sit in the anterior (ventral) horn of the spinal cord. Their degeneration produces wasting, fasciculations and weakness. Among the options, the anterior horn cells are the spinal site of MND.
**Step 3:** Note what is spared. A key feature of MND is that the sensory pathways are spared. There is no sensory loss.
**Step 4:** Exclude the distractors. The peripheral nerve is affected in peripheral neuropathies, not primarily in MND. The spinothalamic tract carries pain and temperature (a sensory pathway) and is spared. The spinocerebellar tract carries unconscious proprioception (also sensory) and is spared.
**Conclusion:** The lesion sits in the anterior horn cells, option A.

**Quick Tip:** Lower motor neuron cell bodies live in the anterior horn.

**175.** All of the following are true about Guillain Barre Syndrome (GBS), except:

(A) Ascending paralysis

(B) Flaccid paralysis

(C) Sensory level

(D) Albumino-cytological dissociation

**Correct Answer:** (C) Sensory level

**Solution:**

**Step 1:** Recall what GBS is. Guillain Barre Syndrome is an acute, immune mediated, demyelinating polyradiculoneuropathy, often following an infection. It attacks peripheral nerves and roots.
**Step 2:** Verify the true statements. Ascending paralysis is true: weakness classically starts in the legs and climbs upward. Flaccid paralysis is true: because

it is a lower motor neuron, peripheral nerve process, the paralysis is flaccid with loss of reflexes (areflexia). Albumino-cytological dissociation is true: the CSF shows raised protein with a normal cell count, a hallmark finding.
**Step 3:** Find the false statement. A sensory level is a feature of a SPINAL CORD lesion (myelopathy), where there is a clear horizontal level below which sensation is lost. GBS is a peripheral nerve disease and does NOT produce a sensory level. So the statement "sensory level" is false for GBS.
**Step 4:** Confirm the exception. The question asks for the statement that is NOT true. That is the sensory level, option C.
**Conclusion:** A sensory level is not a feature of GBS, so the exception is option C.

**Quick Tip:** A sensory level points to the cord, not a peripheral neuropathy.

**176.** Kayser Fleischer rings (KF rings) are seen in:

(A) Pterygium

(B) Hemochromatosis

(C) Wilson's disease

(D) Menkes kinky hair syndrome

**Correct Answer:** (C) Wilson's disease

**Solution:**

**Step 1:** Identify the sign. A Kayser Fleischer ring is a golden brown to greenish ring at the periphery of the cornea, caused by copper deposition in Descemet's membrane.
**Step 2:** Link copper to disease. Wilson's disease is a disorder of copper metabolism (defect in the ATP7B gene) where copper accumulates in the liver, brain and cornea. The corneal copper produces the KF ring, so KF rings are a classic sign of Wilson's disease.
**Step 3:** Recall the wider picture. Wilson's disease presents with liver disease, neuropsychiatric features (tremor, dystonia, behavioural change) and KF rings. Low serum ceruloplasmin and high urinary copper support the diagnosis.

**Step 4:** Exclude the distractors. Pterygium is a fleshy conjunctival growth, unrelated to copper. Hemochromatosis is iron overload, not copper, and does not cause KF rings. Menkes kinky hair syndrome is a copper deficiency disorder (failure to absorb copper) with kinky hair and is the opposite metabolic problem, not associated with KF rings.
**Conclusion:** Kayser Fleischer rings are seen in Wilson's disease, option C.

**Quick Tip:** Copper ring in the cornea means a copper overload disease.

**177.** Lord's plication is done for:

(A) Inguinal hernia

(B) Testicular cancer

(C) Hydrocele

(D) Testicular varices

**Correct Answer:** (C) Hydrocele

**Solution:**

**Step 1:** Identify the procedure. Lord's plication is an operation for a hydrocele, which is an abnormal collection of serous fluid within the tunica vaginalis surrounding the testis.

**Step 2:** Understand the technique. In Lord's plication the sac is opened through a small scrotal incision and a series of interrupted sutures (plication stitches) are placed radially from the edge of the opened sac toward the testis. Tying these sutures bunches the redundant tunica into a ruff around the testis, so the secreting surface is obliterated and fluid no longer collects.

**Step 3:** Note when it is preferred. Lord's plication suits a thin-walled hydrocele sac because there is minimal dissection, little dead space, and therefore a low risk of haematoma compared with the Jaboulay procedure (eversion of the sac), which is better for a thick-walled sac.

**Step 4:** Reject the distractors. Inguinal hernia is repaired by herniotomy or mesh repair, not plication. Testicular cancer is managed by radical inguinal orchidectomy. Testicular varices (varicocele) are treated by varicocele ligation or embolisation.

**Answer: Hydrocele (Option C).**

**Quick Tip:** Think of the operation that pleats the tunica vaginalis to stop fluid collecting around the testis.

**178.** A 50 year old female is admitted with abdominal pain and anuria. Radiological studies revealed bilateral impacted ureteric stones with hydronephrosis. Urine analysis showed RBCs with pus cells in urine. Serum creatinine level was 16 mg/dl and urea level was 200 mmol/l. Which of the following should be the immediate treatment?

(A) Hemodialysis

(B) 'J' stent drainage

(C) Lithotripsy

(D) Ureteroscopic removal of stones

**Correct Answer:** (B) 'J' stent drainage

**Solution:**

**Step 1:** Frame the emergency. The patient has bilateral impacted ureteric stones causing complete obstruction. Anuria with a serum creatinine of **16** mg/dl indicates obstructive (post-renal) acute kidney injury. The pus cells suggest associated infection sitting behind an obstructed system, which is a urological emergency.

**Step 2:** Decide the priority. In obstructive AKI the kidney cannot recover until the obstruction is relieved and the urine drains. Therefore the immediate goal is urgent decompression of the collecting system, not definitive stone removal.

**Step 3:** Choose the drainage method. A double 'J' stent (or alternatively percutaneous nephrostomy) bypasses the impacted stone, restores urine flow, drains infected urine, and lets the obstructed kidneys recover. It is quick and can be done even in a sick, septic patient.

**Step 4:** Reject the distractors. Lithotripsy (ESWL) and ureteroscopic stone removal are definitive treatments that should not be done in the acute setting on an obstructed, infected, uraemic patient, because manipulating an infected obstructed system risks pushing infection into the circulation. Hemodialysis corrects the biochemistry but does nothing for the mechanical obstruction; it is added only if life-threatening uraemia, hyperkalaemia or fluid overload persists, and even then drainage is still needed.

**Answer: 'J' stent drainage (Option B).**

**Quick Tip:** In obstructed plus infected kidneys, drain first and remove the stone later.

**179.** Which of the following is a complication of PCNL done through the 11th intercostal space, EXCEPT:

(A) Hydrothorax

(B) Hematuria

(C) Damage to colon

(D) Remnant fragments

**Correct Answer:** (A) Hydrothorax

**Solution:**

**Step 1:** Understand the question. This is a negative (EXCEPT) question. Three options are genuine complications of supracostal percutaneous nephrolithotomy (PCNL); one is the odd answer that is NOT typically counted among them.

**Step 2:** Recall the supracostal access risk. When the tract is made above the 11th rib (11th intercostal space), the puncture crosses the pleural reflection. This is exactly why thoracic complications such as hydrothorax, pleural effusion, hydropneumothorax and pneumothorax are the classic and feared risks of this approach.

**Step 3:** Resolve the wording. Because hydrothorax is in fact the hallmark complication of the supracostal route, this appears to be a recall/transcription issue: as written, hydrothorax is being singled out as the answer (printed key A) even though it is a true complication. The intended teaching point is that the supracostal tract endangers the pleura. The printed key is retained as given because that is the recorded answer for this recall.

**Step 4:** Note the genuine complications. Haematuria is common to any PCNL because the tract passes through vascular renal parenchyma. Colonic injury can occur with a retrorenal colon, particularly on the left. Residual (remnant) stone fragments are a recognised problem after any stone-clearance procedure.

**Answer: Hydrothorax (Option A), as per the recorded key.**

**Quick Tip:** Going above the 11th rib brings the needle near the pleura, so think chest complications.

**180.** Which of the following stones is hard to break by ESWL?

(A) Calcium oxalate monohydrate

(B) Calcium oxalate dihydrate

(C) Uric acid

(D) Struvite

**Correct Answer:** (A) Calcium oxalate monohydrate

**Solution:**

**Step 1:** Understand ESWL fragmentation. Extracorporeal shock wave lithotripsy breaks a stone by focusing acoustic shock waves on it; how well a stone fragments depends on its hardness (composition) and density.

**Step 2:** Rank stone fragility. Soft, easily shattered stones include uric acid and struvite (and dihydrate calcium oxalate is relatively fragile). Hard, shock-resistant stones include calcium oxalate monohydrate (whewellite), brushite (calcium hydrogen phosphate) and cystine.

**Step 3:** Apply to options. Calcium oxalate monohydrate is dense and mechanically tough, so it resists fragmentation by shock waves and often needs more shocks, higher energy, or an alternative such as ureteroscopy with laser lithotripsy.

**Step 4:** Reject the distractors. Calcium oxalate dihydrate (weddellite) is more brittle and breaks more readily. Uric acid and struvite stones are soft and respond well to ESWL.

**Answer: Calcium oxalate monohydrate (Option A).**

**Quick Tip:** The dense monohydrate crystal resists shock waves; soft uric acid and struvite break easily.

**181.** Which of the following is the most common renal vascular anomaly?

(A) Supernumerary renal arteries

(B) Supernumerary renal veins

(C) Double renal arteries

(D) Double renal veins

**Correct Answer:** (A) Supernumerary renal arteries

**Solution:**

**Step 1:** Recall renal vascular embryology. During development the kidney ascends and receives a series of transient lateral splanchnic arteries; normally all but one regress, leaving a single renal artery. If extra branches persist, accessory or supernumerary renal arteries result.

**Step 2:** Identify the commonest anomaly. Persistence of these extra vessels is frequent, so supernumerary (accessory/aberrant) renal arteries are the most common renal vascular variation, present in roughly a quarter to a third of kidneys in anatomical series.

**Step 3:** Note clinical relevance. An accessory lower-pole artery can cross and compress the pelviureteric junction and cause hydronephrosis; these vessels are also important during renal transplantation and partial nephrectomy because they are end arteries with no collateral supply.

**Step 4:** Reject the distractors. Supernumerary and double renal veins exist but are less common than arterial variants, and the term commonly tested as the single most frequent renal vascular anomaly is supernumerary renal arteries.

**Answer: Supernumerary renal arteries (Option A).**

**Quick Tip:** Extra embryonic arteries that fail to regress are the commonest renal vessel variant.

**182.** The first autologous renal transplantation was done by:

(A) Hardy

(B) Kavosis

(C) Higgins

(D) Studor

**Correct Answer:** (A) Hardy

**Solution:**

**Step 1:** Define the term. Autologous (autotransplant) renal transplantation means removing a patient's own kidney and re-implanting it elsewhere in the same patient, typically into the iliac fossa, for example to reconstruct a long ureteric defect or treat complex renovascular disease.

**Step 2:** Recall the historical attribution. James D. Hardy is the name linked in surgical history to renal autotransplantation; Hardy is credited with the first kidney autotransplant. Hardy is also famous as a pioneering transplant surgeon who performed the first human lung transplant.

**Step 3:** Match to options. Among the names given, Hardy is the recognised pioneer of autologous renal transplantation, so option A is correct.

**Step 4:** Reject the distractors. The other names listed are not the figures credited with the first autologous renal transplant in the standard surgical record, so they are incorrect.

**Answer: Hardy (Option A).**

**Quick Tip:** The same pioneering surgeon famous for the first human lung transplant.

**183.** The best time for surgery of an undescended testis is:

(A) Just after birth

(B) 6 months of age

(C) 12 months of age

(D) 21 months of age

**Correct Answer:** (C) 12 months of age

**Solution:**

**Step 1:** Recall natural descent. A testis may still descend spontaneously during the first few months of life, so surgery is not done immediately at birth. Spontaneous descent rarely occurs after about 6 months of (corrected) age.

**Step 2:** Recall the timing rule. Orchidopexy is recommended once spontaneous descent is no longer expected, traditionally completed by about 1 year (12 months) of age, with current guidance favouring between 6 and 12 months. The question's expected answer is 12 months.

**Step 3:** Justify why early surgery matters. Bringing the testis into the cooler scrotum early helps preserve germ-cell development and fertility and improves later examination for malignancy, since a retained intra-abdominal testis carries a higher cancer risk and impaired spermatogenesis.

**Step 4:** Reject the distractors. 'Just after birth' is too early because spontaneous descent may still happen. 21 months is too late, since waiting beyond the first year risks irreversible damage to the germinal epithelium.

**Answer: 12 months of age (Option C).**

**Quick Tip:** Wait for spontaneous descent to finish, but finish surgery by the first birthday.

**184.** The Grayhack shunt is established between:

(A) Corpora cavernosa and corpus spongiosum

(B) Corpora cavernosa and saphenous vein

(C) Corpora cavernosa and dorsal vein

(D) Corpora cavernosa and glans

**Correct Answer:** (B) Corpora cavernosa and saphenous vein

**Solution:**

**Step 1:** Set the context. Shunt operations are used to treat refractory low-flow (ischaemic) priapism. They drain trapped blood out of the engorged corpora cavernosa so the persistent erection resolves and the cavernosal tissue is saved.

**Step 2:** Classify the shunts. Distal (glanular) shunts connect the corpora cavernosa to the glans, for example Winter, Ebbehoj and Al-Ghorab. Proximal shunts connect the corpora cavernosa to the corpus spongiosum (Quackels/Sacher). Venous shunts connect the corpora cavernosa to a vein.

**Step 3:** Identify Grayhack. The Grayhack shunt is a venous (caverno-venous) shunt that anastomoses the corpus cavernosum to the saphenous vein, diverting the trapped blood into the venous system to relieve the priapism.

**Step 4:** Reject the distractors. Corpora cavernosa to corpus spongiosum is the Quackels shunt. Corpora cavernosa to dorsal vein is the Barry shunt (a different venous shunt). Corpora cavernosa to glans describes the distal glanular shunts (Winter/Al-Ghorab), not Grayhack.

**Answer: Corpora cavernosa and saphenous vein (Option B).**

**Quick Tip:** It is a venous shunt for priapism that drains into a leg vein.

**185.** Most common site of urethral carcinoma in men is:

(A) Bulbomembranous urethra

(B) Penile urethra

(C) Prostatic urethra

(D) Fossa navicularis

**Correct Answer:** (A) Bulbomembranous urethra

**Solution:**

**Step 1:** Male urethral carcinoma is rare. It is divided by site into anterior (penile and bulbomembranous) and posterior (prostatic) urethra.
**Step 2:** By far the most common site is the bulbomembranous urethra, which accounts for roughly half of all cases. The penile urethra is next, and prostatic urethra is least common.
**Step 3:** The dominant tumour type at the bulbomembranous and penile urethra is squamous cell carcinoma, linked to chronic inflammation and stricture. Site matters because more proximal (bulbomembranous) tumours present late and carry a worse prognosis.
**Step 4:** Penile urethra (option B) is a frequent but second-ranked site. Prostatic urethra (option C) is uncommon and tends to be urothelial. Fossa navicularis (option D) is a distal anterior site that is not the most common.
**Conclusion:** The correct answer is **A. Bulbomembranous urethra**.

**Quick Tip:** Think of the segment where strictures and chronic inflammation cluster.

**186.** An adult presented with hematemesis and upper abdominal pain. Endoscopy revealed a growth at the pyloric antrum of the stomach. CT scan showed growth involving the pyloric antrum without infiltration or invasion into surrounding structures and no evidence of distant metastasis. At laparotomy the neoplastic growth was observed to involve the posterior wall of the stomach and the pancreas, extending 6 cm up to the tail of the pancreas. What will be the most appropriate surgical management?

(A) Closure of the abdomen

(B) Antrectomy and vagotomy

(C) Partial gastrectomy and distal pancreatectomy

(D) Partial gastrectomy, distal pancreatectomy and splenectomy

**Correct Answer:** (C) Partial gastrectomy and distal pancreatectomy

**Solution:**

**Step 1:** This is a gastric carcinoma of the antrum that, at laparotomy, is found to directly invade an adjacent organ (the pancreas, up to the tail). There is no distant metastasis. Direct invasion of a contiguous organ is a T4b lesion, not a contraindication to surgery, provided an en bloc resection can clear it.
**Step 2:** The principle for locally advanced gastric cancer invading a neighbouring organ is en bloc resection: remove the involved stomach together with the directly invaded organ in one specimen to achieve R0 margins. Here that means partial (subtotal) gastrectomy plus distal pancreatectomy to take the tumour and the invaded pancreatic body and tail together.
**Step 3:** Closure of the abdomen (option A) is wrong because there is no unresectable distant disease or peritoneal spread; the lesion is resectable.
**Step 4:** Antrectomy and vagotomy (option B) is an operation for peptic ulcer disease, not for an invasive cancer, and it would leave the pancreatic disease behind.
**Step 5:** Adding splenectomy (option D) is not automatically required just because a distal pancreatectomy is done. Splenectomy is added only if the spleen or splenic hilum nodes are involved; here the disease stops at the pancreatic tail without stated splenic involvement, so routine splenectomy is overtreatment in this stem.
**Conclusion:** The correct answer is **C. Partial gastrectomy and distal pancreatectomy**.

**Quick Tip:** Resectable cancer invading a neighbour means en bloc removal of both, no more than needed.

**187.** All of the following about gastrointestinal carcinoid tumors are true, except:

(A) Small intestine and appendix account for almost 60% of all gastrointestinal carcinoids

(B) Rectum is spared

(C) 5 year survival for carcinoid tumors is >60%

(D) Appendiceal carcinoids are more common in females than males

**Correct Answer:** (B) Rectum is spared

**Solution:**

**Step 1:** This is a true/except question, so identify the one false statement. Carcinoid (well-differentiated neuroendocrine) tumours arise throughout the gut, with the GI tract being the commonest overall site.
**Step 2:** Option A is true. The small intestine and appendix together account for a large share, around 60%, of GI carcinoids, the small bowel being the leading single site.
**Step 3:** Option B says the rectum is spared, which is false. The rectum is actually one of the most common sites of GI carcinoids and is frequently picked up incidentally at endoscopy. Saying it is spared is medically wrong, so this is the EXCEPT answer.
**Step 4:** Option C is true. Carcinoids are generally indolent and slow growing, and overall 5 year survival exceeds 60%.
**Step 5:** Option D is true. Appendiceal carcinoids show a female preponderance, partly reflecting more appendicectomies and incidental detection in women.
**Conclusion:** The false statement, and therefore the correct answer, is **B. Rectum is spared**.

**Quick Tip:** Which option claims a common carcinoid site is left out entirely?

**188.** Treatment of choice for annular pancreas is:

(A) Division of pancreas

(B) Duodenoduodenostomy

(C) Duodenojejunostomy

(D) Roux-en-Y loop

**Correct Answer:** (C) Duodenojejunostomy

**Solution:**

**Step 1:** Annular pancreas is a congenital anomaly where a ring of pancreatic tissue encircles the second part of the duodenum and can cause duodenal obstruction. The problem is the obstruction, not the pancreatic ring itself.
**Step 2:** The surgical principle is to bypass the obstruction rather than divide the pancreatic ring. Dividing the annulus risks pancreatitis, a pancreatic fistula and duodenal injury, so it is avoided.
**Step 3:** A bypass procedure is therefore chosen. The classic operations are duodenoduodenostomy or duodenojejunostomy, which restore duodenal continuity around the obstructing ring. As printed, this question takes duodenojejunostomy as the answer of choice for relieving the obstruction.
**Step 4:** Division of the pancreas (option A) is contraindicated for the reasons above. A Roux-en-Y loop (option D) is a reconstruction technique used in other settings but is not the standard named bypass here.
**Conclusion:** The correct answer is **C. Duodenojejunostomy**. Note that duodenoduodenostomy is an equally valid bypass; the operative aim in either case is to bypass the ring, never to cut it.

**Quick Tip:** Bypass the ring, never cut it.

**189.** A lady presented with recurrent attacks of giddiness and abdominal pain for three months. Endoscopy was normal. Her fasting blood glucose was 40 mg% and insulin levels were elevated. CT abdomen showed a well defined 8 mm enhancing lesion in the head of the pancreas, with no other abnormal findings. What should be the treatment plan for this patient?

(A) Whipple's operation

(B) Enucleation

(C) Enucleation with radiotherapy

(D) Administration of streptozotocin

**Correct Answer:** (B) Enucleation

**Solution:**

**Step 1:** Fasting hypoglycaemia (glucose 40 mg%) with inappropriately high insulin and a small enhancing pancreatic lesion is the picture of an insulinoma. The symptoms fit Whipple's triad: symptoms of hypoglycaemia, low measured glucose, and relief with glucose.
**Step 2:** Insulinomas are usually small (often under 2 cm), solitary and benign (about 90%). This one is only 8 mm and well defined, so it behaves as a typical benign lesion.
**Step 3:** For a small, benign, well-localised insulinoma, the treatment of choice is simple surgical enucleation. This removes the tumour while preserving maximal pancreatic tissue and exocrine and endocrine function.
**Step 4:** Whipple's operation (option A) is an extensive pancreaticoduodenectomy reserved for large, deep, or malignant pancreatic head tumours; it is excessive for an 8 mm benign nodule.
**Step 5:** Radiotherapy (option C) has no role for a benign, resectable insulinoma. Streptozotocin (option D) is chemotherapy used for unresectable or metastatic malignant islet cell tumours, not for a small benign curable lesion.
**Conclusion:** The correct answer is **B. Enucleation**.

**Quick Tip:** Small benign insulinoma wants the most tissue-sparing operation.

**190.** A young male patient presents with complete rectal prolapse. The surgery of choice is:

(A) Abdominal rectopexy

(B) Delorme's procedure

(C) Anterior resection

(D) Goodsall's procedure

**Correct Answer:** (A) Abdominal rectopexy

**Solution:**

**Step 1:** Complete rectal prolapse (procidentia) is full-thickness protrusion of the rectum through the anus. Definitive treatment is surgical, and the operative route

is chosen partly by the patient's fitness.
**Step 2:** In a young, fit patient, the abdominal approach is preferred because it gives the most durable repair with the lowest recurrence. Abdominal rectopexy fixes the mobilised rectum to the sacral promontory and corrects the prolapse.
**Step 3:** Delorme's procedure (option B) is a perineal operation reserved mainly for elderly or unfit patients or short prolapses; it has higher recurrence and is not first choice in a young fit man.
**Step 4:** Anterior resection (option C) alone is not the standard primary operation for prolapse; resection rectopexy may be used in selected cases but plain anterior resection is not the answer here.
**Step 5:** Goodsall's rule/procedure (option D) relates to anal fistula tracks and has nothing to do with rectal prolapse, so it is a distractor.
**Conclusion:** The correct answer is **A. Abdominal rectopexy**.

**Quick Tip:** Young and fit favors the durable abdominal route.

**191.** According to the Bismuth / Strasberg classification, 'cystic duct blow out' (cystic duct stump leak) is classified as:

(A) Type A

(B) Type B

(C) Type C

(D) Type D

**Correct Answer:** (A) Type A

**Solution:**

**Step 1:** The Strasberg (Bismuth-Strasberg) classification grades bile duct injuries after cholecystectomy from A to E.
**Step 2:** Type A is a bile leak from a minor duct still in continuity with the main duct, classically the cystic duct stump leak or a leak from a small duct in the gallbladder bed (duct of Luschka). A cystic duct stump blow out is the prototype Type A injury.

**Step 3:** Type B is occlusion of an aberrant right hepatic duct, and Type C is a transected aberrant right hepatic duct that leaks. Type D is a lateral injury to the main bile duct. Type E covers circumferential injuries of the main duct (further subdivided E1 to E5).
**Step 4:** Since the cystic duct stump is a minor duct in continuity, its leak fits Type A, not B, C or D.
**Conclusion:** The correct answer is **A. Type A**.

**Quick Tip:** A leak from a minor duct still in continuity is the lowest grade.

**192.** In orthotopic liver transplantation, which is the best way to get bile drainage in the donor liver?

(A) Donor bile duct to recipient bile duct (duct-to-duct), or Roux-en-Y choledochojejunostomy

(B) Donor bile duct to duodenum of recipient

(C) Donor bile duct to jejunum of recipient

(D) External drainage for a few days followed by choledochojejunostomy

**Correct Answer:** (A) Donor bile duct to recipient bile duct (duct-to-duct), or Roux-en-Y choledochojejunostomy

**Solution:**

**Step 1:** After implanting the donor liver in orthotopic liver transplantation, biliary continuity must be restored so bile drains into the gut.
**Step 2:** The preferred reconstruction is a direct duct-to-duct anastomosis (donor common bile duct to recipient common bile duct, choledochocholedochostomy). This is physiological, preserves the sphincter of Oddi, and keeps endoscopic access to the biliary tree for later stenting if needed.
**Step 3:** When duct-to-duct is not feasible, for example with a diseased or absent recipient duct (as in primary sclerosing cholangitis) or a large size mismatch, a Roux-en-Y choledochojejunostomy (hepaticojejunostomy) is used instead.
**Step 4:** Option A captures both the first choice and its standard backup, so it is

the best answer. Anastomosis to the duodenum (option B) is not standard. A plain loop jejunal anastomosis without a Roux limb (option C) risks reflux cholangitis and is inferior. Routine external drainage then later surgery (option D) is not the preferred primary plan.
**Conclusion:** The correct answer is **A. Donor duct to recipient duct or Roux-en-Y choledochojejunostomy**.

**Quick Tip:** Reconnect duct to duct when you can, Roux-en-Y when you cannot.

**193.** Most common cysts of the spleen are:

(A) Hydatid cyst

(B) Dermoid cyst

(C) Pseudocyst

(D) Lymphangioma

**Correct Answer:** (A) Hydatid cyst

**Solution:**

**Step 1:** Splenic cysts are divided into two broad groups, true (primary) cysts that have an epithelial or parasitic lining, and false (secondary) cysts that lack a lining.
**Step 2:** Among true cysts, the parasitic group is by far the largest. Worldwide the most common splenic cyst overall is the hydatid (echinococcal) cyst, caused by *Echinococcus granulosus*. In endemic regions it accounts for the majority of all splenic cysts.
**Step 3:** A pseudocyst (option C) is the most common type of false cyst and usually follows trauma. In Western, non-endemic populations pseudocysts may be the commonest seen locally, but for the worldwide most common splenic cyst the hydatid cyst is the standard answer expected in this exam.
**Step 4:** Dermoid cysts (option B) and lymphangiomas (option D) are rare congenital true cysts and are not the most common.

**Step 5:** Therefore the most common cyst of the spleen is the hydatid cyst.
Answer: A. Hydatid cyst.

**Quick Tip:** Think parasitic true cyst, the same organism that famously affects the liver.

**194.** All of the following are primary restrictive operations for morbid obesity, except:

(A) Vertical banded gastroplasty

(B) Duodenal switch operation

(C) Roux-en-Y gastric bypass

(D) Laparoscopic adjustable gastric banding

**Correct Answer:** (B) Duodenal switch operation

**Solution:**

**Step 1:** Bariatric operations are grouped as restrictive (reduce stomach volume so the patient eats less), malabsorptive (bypass small bowel so less is absorbed), or combined.
**Step 2:** Vertical banded gastroplasty (option A) creates a small upper gastric pouch with a banded outlet, so it is purely restrictive.
**Step 3:** Laparoscopic adjustable gastric banding (option D) places an inflatable band around the upper stomach to limit intake, so it is purely restrictive.
**Step 4:** The duodenal switch (option B) combines a sleeve gastrectomy with a biliopancreatic diversion that bypasses most of the small intestine. It is predominantly a malabsorptive (combined) procedure, not a primary restrictive one. This is the exception.
**Step 5:** Roux-en-Y gastric bypass (option C) is classically taught as a combined procedure, but it is dominated by gastric restriction from the small pouch, so among the given choices it is treated as the restrictive component and the printed key marks the duodenal switch as the odd one out.
Answer: B. Duodenal switch operation.

**Quick Tip:** Which listed operation works mainly by bypassing small bowel rather than shrinking the stomach?

**195.** The Trauma and Injury Severity Score (TRISS) includes:

(A) GCS + BP + RR

(B) RTS + ISS + Age

(C) RTS + ISS + GCS

(D) RTS + GCS + BP

**Correct Answer:** (B) RTS + ISS + Age

**Solution:**

**Step 1:** TRISS is a method used to predict the probability of survival after trauma by combining a physiological measure of injury, an anatomical measure of injury, and the patient's age.
**Step 2:** The physiological component is the Revised Trauma Score (RTS), which itself is built from the Glasgow Coma Scale, systolic blood pressure, and respiratory rate.
**Step 3:** The anatomical component is the Injury Severity Score (ISS), derived from the Abbreviated Injury Scale for the three most severely injured body regions.
**Step 4:** The third component is patient age, which adjusts the estimate because survival worsens with increasing age for the same injury. So TRISS = RTS + ISS + Age.
**Step 5:** Options C and D wrongly add GCS or BP separately; those values are already captured inside the RTS, so they are not listed as independent TRISS inputs. Option A lists only physiological variables and omits the anatomical score and age.
Answer: B. RTS + ISS + Age.

**Quick Tip:** One physiological score, one anatomical score, and a patient factor that

worsens with years.

**196.** A 27-year-old patient presented with left-sided abdominal pain to the emergency room 6 hours after a road traffic accident. He was haemodynamically stable and FAST positive. A contrast-enhanced CT (CECT) scan showed a grade III splenic laceration. (a) With no contrast blush, what is the most appropriate treatment? (b) If the same CECT instead shows a contrast blush along with the grade III laceration, what is the most appropriate management? (Choose the answer for part b.)

(A) Splenectomy

(B) Splenorrhaphy

(C) Splenic artery embolization

(D) Conservative management

**Correct Answer:** (C) Splenic artery embolization

**Solution:**

**Step 1:** This is a two-part question. The patient is young, haemodynamically stable, and FAST positive with a grade III splenic laceration on CECT. The deciding factor is whether the CT shows a contrast blush, which signals active arterial extravasation or a pseudoaneurysm.

**Step 2:** Part (a): no contrast blush. A stable patient with a low-to-moderate grade injury and no blush is the ideal candidate for non-operative (conservative) management with close monitoring. So part (a) answer is D, conservative management.

**Step 3:** Part (b): a contrast blush is now present. A blush means ongoing bleeding even though the patient is currently stable, so pure observation is unsafe and splenic salvage is still desirable in a stable patient.

**Step 4:** In a stable patient with a blush, splenic artery embolization controls the bleeding while preserving the spleen, avoiding the immune cost of splenectomy. This is the answer for part (b).

**Step 5:** Splenectomy and splenorrhaphy are operative and are reserved for haemodynamic instability or failed non-operative management, not for a stable

patient who can be managed by angioembolization.
Answer for part (b): C. Splenic artery embolization.

**Quick Tip:** Stable patient, but the contrast blush means active bleeding, so save the spleen without open surgery.

**197.** A lady presented to the emergency department with a stab injury to the left side of the abdomen. She was haemodynamically stable and a contrast-enhanced CT scan revealed a laceration in the spleen. Laparoscopy was planned, but the patient's pO2 suddenly dropped as soon as the pneumoperitoneum was created. What is the most likely cause?

(A) Gas embolism through splenic vessels

(B) Injury to the left lobe of the diaphragm

(C) Inferior vena cava compression

(D) Injury to the colon

**Correct Answer:** (A) Gas embolism through splenic vessels

**Solution:**

**Step 1:** The key event is an abrupt fall in pO2 at the exact moment carbon dioxide pneumoperitoneum is created, in a patient who has an open laceration of a vascular organ, the spleen.
**Step 2:** Pneumoperitoneum is made by insufflating CO2 into the abdomen under positive pressure. The lacerated splenic vessels provide an open, low-pressure venous channel directly exposed to that pressurised gas.
**Step 3:** The high intra-abdominal CO2 pressure can be forced into the opened splenic veins, producing a gas (CO2) embolism. The gas travels to the right heart and pulmonary circulation, blocking gas exchange and causing a sudden drop in pO2 and oxygen saturation.
**Step 4:** A diaphragmatic injury (option B) can cause respiratory compromise but would not classically produce an instantaneous desaturation precisely synchronised with insufflation. IVC compression (option C) lowers venous return

and blood pressure rather than causing an acute hypoxic drop. Colonic injury (option D) does not cause acute hypoxia.
**Step 5:** The temporal link between creating pneumoperitoneum and the sudden hypoxia in a patient with open splenic vessels points to CO2 gas embolism.
Answer: A. Gas embolism through splenic vessels.

**Quick Tip:** Open vein plus pressurised CO2 the moment you insufflate equals gas in the circulation.

**198.** A patient presents with fever for 3 weeks. On examination he has splenomegaly. Ultrasonography reveals a hypoechoic shadow in the spleen near the hilum. Gram-negative bacilli are isolated on blood culture. Which of the following is the most likely causative organism?

(A) Cytomegalovirus

(B) Toxoplasma

(C) Salmonella

(D) Lymphoma virus

**Correct Answer:** (C) Salmonella

**Solution:**

**Step 1:** Identify the syndrome. Prolonged fever (3 weeks), splenomegaly, a hypoechoic splenic lesion (suggesting a splenic abscess), and crucially a Gram-negative bacillus on blood culture.
**Step 2:** The blood culture result is the discriminator. The organism must be a Gram-negative bacillus that grows on routine blood culture.
**Step 3:** Among the options, only *Salmonella* (typically *Salmonella typhi* causing enteric fever) is a Gram-negative bacillus. Enteric fever classically gives prolonged fever and splenomegaly, and the spleen is the commonest visceral site for a typhoid abscess.
**Step 4:** The other options do not fit. Cytomegalovirus and the Epstein-Barr type lymphoma-associated virus are viruses, not Gram-negative bacilli. *Toxoplasma* is a

protozoan parasite. None would be reported as a Gram-negative bacillus on a bacterial blood culture.
**Step 5:** The clinical picture of enteric fever with a splenic abscess and a Gram-negative bacillus on culture points to Salmonella.
Answer: C. Salmonella.

**Quick Tip:** Only one of the four options is a Gram-negative bacillus, and it causes enteric fever.

**199.** A patient with ITP has a platelet count of 50,000 and is being planned for splenectomy. What is the best time for platelet infusion in this patient?

(A) 2 hours before surgery

(B) At the time of skin incision

(C) After ligating the splenic artery

(D) Immediately after removal of the spleen

**Correct Answer:** (C) After ligating the splenic artery

**Solution:**

**Step 1:** In immune thrombocytopenic purpura (ITP), antiplatelet antibodies coat platelets, and the spleen is the major organ that destroys these antibody-coated platelets and also produces the antibody.
**Step 2:** If platelets are transfused early, before the spleen is dealt with, the spleen rapidly sequesters and destroys the transfused platelets, so the benefit is lost.
**Step 3:** The splenic artery is the main inflow to the spleen. Ligating it cuts off blood flow so the spleen can no longer trap and destroy circulating platelets effectively.
**Step 4:** Therefore the ideal time to give platelets is after the splenic artery has been ligated. The transfused platelets then survive and help control intraoperative bleeding when they are most needed.
**Step 5:** Giving platelets 2 hours before surgery or at skin incision (options A and B) wastes them on the still-active spleen. Waiting until after the spleen is

removed (option D) is later than needed; once the artery is tied the spleen is functionally out, so platelets given at that point are already protected and can act during the rest of the operation.
Answer: C. After ligating the splenic artery.

**Quick Tip:** Give platelets only once the spleen can no longer destroy them, that is the moment its artery is tied off.

**200.** The most common cause of abdominal aortic aneurysm is:

(A) Atherosclerosis

(B) Trauma

(C) Syphilis

(D) Vasculitis

**Correct Answer:** (A) Atherosclerosis

**Solution:**

**Step 1:** An abdominal aortic aneurysm (AAA) is a permanent localised dilatation of the infrarenal aorta, occurring mostly in older men with cardiovascular risk factors.
**Step 2:** The dominant underlying process is degeneration of the aortic wall driven by atherosclerosis, with loss of elastin and smooth muscle and increased wall stress. Atherosclerosis is therefore the most common cause.
**Step 3:** Trauma (option B) causes pseudoaneurysms and is uncommon as a cause of a true AAA. Syphilis (option C) classically affects the ascending thoracic aorta, not the abdominal aorta. Vasculitis (option D) is a rare cause.
**Step 4:** The location (infrarenal abdominal aorta) and the typical risk profile (smoking, hypertension, hyperlipidaemia, age, male sex) all align with atherosclerotic degeneration.
**Step 5:** Hence the most common cause is atherosclerosis.
Answer: A. Atherosclerosis.

**Quick Tip:** Think older smoker with an infrarenal aorta worn down by the same plaque that clogs arteries.

**201.** Which of the following grading methods is used to evaluate the prognosis or outcome after subarachnoid hemorrhage?

(A) Glasgow coma scale

(B) Hunt and Hess scale

(C) Glasgow-Blatchford bleeding score

(D) Intracerebral hemorrhage score

**Correct Answer:** (B) Hunt and Hess scale

**Solution:**

**Step 1:** Subarachnoid hemorrhage (SAH) means bleeding into the space between the arachnoid and pia mater, most often from a ruptured berry aneurysm. The clinical state on arrival predicts how the patient will do, so a dedicated grading scale is used.

**Step 2:** The Hunt and Hess scale grades SAH from I to V based on the clinical picture. Grade I is mild headache, Grade V is deep coma with decerebrate posturing. A higher grade means worse prognosis and higher surgical risk. This makes it the standard tool to predict outcome after SAH.

**Step 3:** Why the others are wrong. The Glasgow coma scale measures the depth of unconsciousness in general head injury and is not SAH specific, though it feeds into other scores. The Glasgow-Blatchford bleeding score is for upper gastrointestinal bleeding, not brain bleeds. The intracerebral hemorrhage (ICH) score predicts mortality in intraparenchymal brain bleeds, not subarachnoid bleeds.

**Step 4:** The dedicated prognostic grading scale for SAH is the Hunt and Hess scale.

Answer: Option B, Hunt and Hess scale.

**Quick Tip:** Think of the SAH specific clinical grade that runs I to V.

**202.** Health status of a child under 5 years of age will be adversely affected by all of the following, except:

(A) Malnutrition

(B) Low birth weight

(C) Maternal Hb of 11 gm%

(D) Infections

**Correct Answer:** (C) Maternal Hb of 11 gm%

**Solution:**

**Step 1:** The question asks for the one factor that does NOT harm the health of an under-5 child. So we look for the normal or protective value among the choices.
**Step 2:** A maternal hemoglobin of 11 gm% is the normal lower cut-off for pregnancy. The WHO defines anemia in pregnancy as Hb less than 11 gm%. So an Hb of 11 gm% means the mother is not anemic, which is good for the baby. This is the exception.
**Step 3:** Why the others are harmful. Malnutrition directly stunts growth and weakens immunity, raising death risk. Low birth weight is one of the strongest predictors of infant illness and death. Infections such as pneumonia and diarrhea are leading causes of under-5 mortality.
**Step 4:** The only factor that does not adversely affect the child is a maternal Hb of 11 gm%, which is normal.
Answer: Option C, Maternal Hb of 11 gm%.

**Quick Tip:** Which option is actually a normal value, not a risk?

**203.** A seven year old asymptomatic girl is found to have persistent hypertension. There is no significant history and urine examination is normal. Which of the following is the most likely cause?

(A) Essential hypertension

(B) Renal parenchymal disease

(C) Polycystic kidney disease

(D) Coarctation of aorta

**Correct Answer:** (B) Renal parenchymal disease

**Solution:**

**Step 1:** In children, unlike adults, hypertension is usually secondary, meaning it has an underlying cause. The younger the child and the higher the blood pressure, the more likely there is a secondary cause.
**Step 2:** The single most common cause of secondary hypertension in children is renal disease. Of the renal causes, renal parenchymal disease (such as chronic glomerulonephritis, reflux nephropathy or scarring) is the leading group, followed by renovascular disease.
**Step 3:** The normal urine examination does not rule out renal parenchymal disease, because scarred or chronically damaged kidneys can have bland urine. So this choice still fits best.
**Step 4:** Why the others are less likely. Essential hypertension is the rule in adults and adolescents but is uncommon at age seven. Polycystic kidney disease is one specific cause but is far less common than parenchymal disease as a group and often gives a palpable mass or family history. Coarctation of the aorta would usually show a blood pressure difference between arms and legs with weak femoral pulses, none of which is described.
**Step 5:** The most likely cause is renal parenchymal disease.
Answer: Option B, Renal parenchymal disease.

**Quick Tip:** In young children most hypertension is secondary, and the commonest group is renal.

**204.** A child presented with intermittent episodes of left sided flank pain. Ultrasonography reveals a large hydronephrosis with dilated renal pelvis and cortical thinning with a normal ureter. Kidney differential function was observed to be 19 percent. Which of the following is the best management?

(A) Nephrectomy

(B) Pyeloplasty

(C) External drainage

(D) Endopyelotomy

**Correct Answer:** (B) Pyeloplasty

**Solution:**

**Step 1:** The picture is hydronephrosis with a dilated renal pelvis and a normal (non-dilated) ureter. Obstruction at the junction where the pelvis meets the ureter, with a normal ureter below it, is the hallmark of pelviureteric junction (PUJ) obstruction.

**Step 2:** The aim is to relieve the obstruction and save kidney function. The standard, definitive operation for PUJ obstruction is a pyeloplasty (Anderson-Hynes dismembered pyeloplasty), which removes the narrow segment and creates a wide, dependent join between pelvis and ureter.

**Step 3:** The differential function of 19 percent is reduced but the kidney is not dead. As a rule of thumb a function above roughly 10 to 15 percent is salvageable, so the kidney is worth preserving rather than removing. Pyeloplasty can recover or stabilize function.

**Step 4:** Why the others are wrong. Nephrectomy is reserved for a poorly functioning kidney, usually with function below about 10 percent, which is not the case here. External drainage (nephrostomy) is only a temporary measure, for example in infection, not the definitive cure. Endopyelotomy has lower success rates in children, especially with a very dilated system and crossing vessels, so it is not the best choice.

**Step 5:** The best management is pyeloplasty.

Answer: Option B, Pyeloplasty.

**Quick Tip:** Dilated pelvis with a normal ureter equals PUJ obstruction, and 19 percent function is still worth saving.

**205.** A neonate presented with fever, lethargy, abdominal distension, vomiting and constipation. Clinically he was diagnosed as volvulus neonatorum with suspected perforation. Best investigation would be:

(A) Plain X-ray

(B) Barium enema

(C) Upper GI endoscopy

(D) Barium meal follow through

**Correct Answer:** (A) Plain X-ray

**Solution:**

**Step 1:** The key phrase is suspected perforation. Once perforation is suspected, the first concern is to look for free air in the abdomen and to avoid any contrast that could leak into the peritoneum.
**Step 2:** A plain X-ray of the abdomen, taken erect or as a lateral decubitus film, shows free gas under the diaphragm or along the abdominal wall when there is a perforation. It is quick, safe and needs no contrast, so it is the best first investigation in a sick neonate with suspected perforation.
**Step 3:** Why the contrast studies are wrong here. Barium is strongly contraindicated when perforation is suspected, because barium leaking into the peritoneum causes a severe, often fatal chemical peritonitis. This rules out both barium enema and barium meal follow through. If contrast is truly needed, a water-soluble agent is used, not barium.
**Step 4:** Upper GI endoscopy has no role in diagnosing a neonatal perforation or volvulus and is unsafe in this setting.
**Step 5:** The best investigation is a plain X-ray to detect free air.
Answer: Option A, Plain X-ray.

**Quick Tip:** Suspected perforation means look for free air and never give barium.

**206.** A ten year old boy presents to the pediatric emergency unit with seizures. Blood pressure in the upper extremity is measured as 200/140 mm Hg. Femoral pulses are not palpable. The most likely diagnosis amongst the following is:

(A) Takayasu aortoarteritis

(B) Renal parenchymal disease

(C) Grand mal seizures

(D) Coarctation of aorta

**Correct Answer:** (D) Coarctation of aorta

**Solution:**

**Step 1:** Pick out the two strong clues. There is very high blood pressure in the arms (200/140) and the femoral pulses are absent. This combination of upper-limb hypertension with weak or absent lower-limb pulses points to a fixed narrowing of the aorta beyond the arm vessels.
**Step 2:** Coarctation of the aorta is a congenital narrowing just past the left subclavian artery. Blood pressure is high in the arms but low in the legs, so femoral pulses are weak, delayed or absent. A hypertensive crisis can cause the seizure. This fits the picture exactly.
**Step 3:** Why the others are less likely. Takayasu arteritis can also cause pulseless limbs and hypertension, but it usually affects young adult women and many vessels, and it is far less likely than coarctation as the classic exam answer for a child with this exact triad. Renal parenchymal disease causes hypertension but does not by itself abolish the femoral pulses. Grand mal seizure is just a description of the fit, not a cause of the absent femoral pulses or the marked blood pressure gradient.
**Step 4:** The diagnosis that ties together arm hypertension, absent femoral pulses and seizures in a child is coarctation of the aorta.
Answer: Option D, Coarctation of aorta.

**Quick Tip:** High arm BP plus absent femoral pulses equals a narrowed aorta.

**207.** A child presents with hepatomegaly and hypoglycemia. There is no improvement in blood sugar even after administration of epinephrine. What is the likely diagnosis?

(A) Von Gierke's disease

(B) Andersen's disease

(C) Pompe's disease

(D) McArdle's disease

**Correct Answer:** (A) Von Gierke's disease

**Solution:**

**Step 1:** The features are a big liver, low blood sugar, and crucially, the blood sugar does not rise after epinephrine. Epinephrine and glucagon raise sugar by breaking down liver glycogen and by gluconeogenesis, with the final step being glucose-6-phosphatase converting glucose-6-phosphate to free glucose for release.

**Step 2:** If the sugar will not rise after epinephrine, the block must be at the last common step, glucose-6-phosphatase. A deficiency of this enzyme is Von Gierke's disease, glycogen storage disease type I.

**Step 3:** Von Gierke's disease gives exactly this picture: massive hepatomegaly, severe fasting hypoglycemia, and a failure to respond to epinephrine or glucagon, along with lactic acidosis, hyperuricemia and hyperlipidemia.

**Step 4:** Why the others are wrong. Andersen's disease (type IV, branching enzyme defect) causes cirrhosis and liver failure, not this hypoglycemia pattern. Pompe's disease (type II, acid maltase) is a lysosomal disorder affecting the heart and muscles with cardiomegaly, not fasting hypoglycemia. McArdle's disease (type V, muscle phosphorylase) causes exercise-induced muscle cramps, not liver disease or hypoglycemia.

**Step 5:** The diagnosis is Von Gierke's disease.

Answer: Option A, Von Gierke's disease.

**Quick Tip:** No sugar rise after epinephrine points to a glucose-6-phosphatase block.

**208.** All of the following factors are associated with a substantially greater risk of developing epilepsy after febrile seizures, except:

(A) Complex febrile seizures

(B) Early age of onset

(C) Developmental abnormalities

(D) Positive family history of epilepsy

**Correct Answer:** (B) Early age of onset

**Solution:**

**Step 1:** The question separates two different risks. One is the risk that febrile seizures will simply recur, and the other is the risk of going on to develop true epilepsy (afebrile seizures). This question is about the risk of later epilepsy.
**Step 2:** The established risk factors for developing epilepsy after a febrile seizure are: complex (atypical) febrile seizures, a family history of epilepsy, and neurodevelopmental abnormalities present before the seizure. These three are the textbook predictors.
**Step 3:** Early age of onset is a risk factor for recurrence of febrile seizures, not for developing later epilepsy. So it is the exception in this question, and the printed key of B is correct.
**Step 4:** Confirming the others. Complex febrile seizures (prolonged, focal, or more than one in 24 hours) raise the epilepsy risk. A positive family history of epilepsy raises it. Pre-existing developmental abnormalities raise it. All three belong on the list, so they are not the exception.
**Step 5:** The factor not linked to a substantially greater risk of epilepsy is early age of onset.
Answer: Option B, Early age of onset.

**Quick Tip:** Separate the risk of recurrence from the risk of true epilepsy; early onset

predicts recurrence.

**209.** A child presents with short episodes of vacant stare several times a day. The vacant episode begins abruptly and the child remains unresponsive during the episode. There is no associated history of aura or postictal confusion and the child is otherwise normal. The likely diagnosis is:

(A) Grand mal seizures

(B) Absence seizures

(C) Complex partial seizures

(D) Day dreaming

**Correct Answer:** (B) Absence seizures

**Solution:**

**Step 1:** Pick out the key clinical features. The episodes are brief, occur many times a day, start abruptly, and involve a vacant stare with loss of responsiveness. There is no aura before and no confusion after the spell. The child is normal otherwise.

**Step 2:** Match this picture to a seizure type. Brief (5 to 10 second), frequent staring spells with sudden onset and offset and no postictal phase are the hallmark of typical absence seizures (childhood absence epilepsy). The EEG shows a classic 3 Hz generalized spike and wave pattern, and hyperventilation often triggers an attack.

**Step 3:** Rule out the distractors.
Grand mal (generalized tonic clonic) seizures cause loss of consciousness with falling, limb stiffening and jerking, and clear postictal drowsiness or confusion. None of that is present here.
Complex partial (focal impaired awareness) seizures usually last longer, often have an aura, frequently show automatisms like lip smacking, and are followed by postictal confusion. The absence of aura and postictal confusion argues

against this.
Day dreaming can be interrupted by calling the child and lacks the abrupt automatic onset and the very high daily frequency; it is a diagnosis of exclusion and does not fit a true ictal stare.

**Step 4:** The brief, abrupt, frequent staring spell with no aura and no postictal confusion is classic for absence seizures.

The correct answer is option B, Absence seizures.

**Quick Tip:** Brief, frequent staring with sudden onset and offset and no aura or postictal confusion equals 3 Hz spike and wave.

**210.** Which of the following is the most common cause of meningoencephalitis in children?

(A) Mumps

(B) Arbovirus

(C) HSV

(D) Enterovirus

**Correct Answer:** (D) Enterovirus

**Solution:**

**Step 1:** Note that the question asks for the single most common cause of viral meningoencephalitis (aseptic meningitis with brain involvement) in children, not the most dangerous one.

**Step 2:** Recall the epidemiology. Non polio enteroviruses (coxsackieviruses and echoviruses) are by far the leading viral cause of meningitis and meningoencephalitis worldwide, especially in children, with a clear summer to autumn peak and faeco oral spread. They account for the large majority of identified aseptic meningitis cases.

**Step 3:** Compare the distractors.
HSV is the most common cause of sporadic fatal viral encephalitis and the most clinically important to treat with acyclovir, but in terms of sheer frequency it is far less common than enterovirus.
Arboviruses (for example Japanese encephalitis) cause important epidemic encephalitis in endemic regions but are not the overall most common cause.
Mumps was a notable cause in the pre vaccination era but has become uncommon where MMR vaccination is widespread.

**Step 4:** By overall frequency the leading agent is enterovirus.

The correct answer is option D, Enterovirus.

**Quick Tip:** Most common by frequency is enterovirus; HSV is the most common cause of fatal sporadic encephalitis.

**211.** Which of the following agents is most commonly associated with recurrent meningitis due to CSF leaks?

(A) Meningococci

(B) Pneumococci

(C) Hemophilus influenzae

(D) E. coli

**Correct Answer:** (B) Pneumococci

**Solution:**

**Step 1:** Understand the mechanism. A CSF leak (for example after a skull base fracture or with a congenital dural defect) creates a direct route between the nasopharynx and the meninges. Organisms that colonize the nasopharynx can then repeatedly seed the CSF, causing recurrent bacterial meningitis.

**Step 2:** Identify which nasopharyngeal coloniser dominates this setting.
Streptococcus pneumoniae (pneumococcus) is the classic and most common organism in recurrent meningitis associated with CSF rhinorrhoea or otorrhoea and with anatomical or post traumatic dural defects.

**Step 3:** Address the distractors.
Meningococci cause epidemic and sporadic meningitis but are not the typical organism in recurrent CSF leak meningitis.
Haemophilus influenzae type b was an important cause of childhood meningitis before conjugate vaccination, but it is not the characteristic agent linked to CSF leaks.
E. coli is a leading cause of neonatal meningitis (acquired during birth) and is not the organism implicated in recurrent CSF leak meningitis.

**Step 4:** The classic association is with pneumococcus.

The correct answer is option B, Pneumococci.

**Quick Tip:** CSF leak plus recurrent meningitis equals pneumococcus, the dominant nasopharyngeal coloniser.

**212.** A two year old child with a long history of purulent nasal discharge and fever now presents with conjunctival congestion and edema. His fever is 102 to 103 F and WBC count is 12,000. The culture of eye discharge was negative. X-rays show opacification of the ethmoid sinus. Which of the following should be the next step in evaluating this patient?

(A) CT scan

(B) Urine culture

(C) Blood culture

(D) Repeat culture of eye discharge

**Correct Answer:** (A) CT scan

**Solution:**

**Step 1:** Recognise the clinical picture. A young child with chronic purulent nasal discharge, fever, and opacified ethmoid sinus who now develops periorbital and conjunctival swelling has ethmoiditis complicated by orbital involvement. The combination of proptosis or eyelid and conjunctival oedema with sinusitis raises strong concern for orbital cellulitis or a subperiosteal/orbital abscess spreading from the ethmoid sinus.

**Step 2:** Decide what changes management. The crucial question is whether the infection is preseptal (anterior to the orbital septum) or has crossed into the orbit (postseptal), and whether an abscess has formed. That distinction drives the need for surgical drainage and aggressive intravenous therapy.

**Step 3:** Select the investigation. A contrast enhanced CT scan of the orbit and paranasal sinuses is the imaging of choice. It defines the extent of sinus disease, detects subperiosteal or orbital abscess, and guides surgical decision making. This is the appropriate next step.

**Step 4:** Why the others are wrong.
Urine culture has no role; this is not a urinary source.
Blood culture may be drawn as part of the workup, but it is frequently negative in localized orbital infection and does not define the anatomy or the need for surgery, so it is not the key next step.
Repeating the eye discharge culture adds little when the first was negative and the problem is deep orbital extension, not a superficial conjunctival infection.

**Step 5:** Imaging to assess orbital spread is the priority.

The correct answer is option A, CT scan.

**Quick Tip:** Sinusitis with a swollen eye needs CT to tell preseptal from orbital spread and find an abscess.

**213.** A boy presented with weakness in the lower limbs, calf hypertrophy, positive Gower's sign and an elevated CPK value of 10,000. The most likely diagnosis is:

(A) Duchenne muscular dystrophy

(B) Spinal muscular atrophy

(C) Myotonia congenita

(D) Myotonic dystrophy

**Correct Answer:** (A) Duchenne muscular dystrophy

**Solution:**

**Step 1:** List the findings. A boy with proximal lower limb weakness, pseudohypertrophy of the calves, a positive Gower's sign (climbing up his own legs to stand because of proximal weakness), and a markedly raised creatine phosphokinase of about 10,000.

**Step 2:** Match the syndrome. This tetrad is the textbook presentation of Duchenne muscular dystrophy (DMD), an X linked recessive disorder of the dystrophin gene affecting boys, with onset in early childhood, calf pseudohypertrophy, and CPK levels in the thousands to tens of thousands.

**Step 3:** Eliminate the alternatives.
Spinal muscular atrophy is a lower motor neuron disorder with hypotonia, weakness, fasciculations, and tongue fasciculations, but it does not cause calf pseudohypertrophy, and CPK is normal or only mildly raised.
Myotonia congenita features muscle stiffness and difficulty relaxing (myotonia) often with muscle hypertrophy, but it lacks progressive proximal weakness and the very high CPK; it is a channelopathy.
Myotonic dystrophy shows myotonia, distal weakness, facial weakness, cataracts, and frontal balding, with only mild CPK elevation, and the classic age and pattern do not fit a young boy with calf pseudohypertrophy and CPK 10,000.

**Step 4:** The complete picture is Duchenne muscular dystrophy.

The correct answer is option A, Duchenne muscular dystrophy.

**Quick Tip:** Boy plus calf pseudohypertrophy plus Gower's sign plus very high CPK equals Duchenne.

**214.** The primary metabolic bone disorder in scurvy is:

(A) Decreased mineralization

(B) Decreased osteoid matrix formation

(C) Increased bone resorption

(D) Decreased bone mass with normal mineralization and osteoid formation

**Correct Answer:** (B) Decreased osteoid matrix formation

**Solution:**

**Step 1:** Recall the biochemistry of scurvy. Scurvy is vitamin C (ascorbic acid) deficiency. Vitamin C is an essential cofactor for prolyl and lysyl hydroxylase, the enzymes that hydroxylate proline and lysine during collagen synthesis. Without it, collagen cannot be properly cross linked and stabilised.

**Step 2:** Apply this to bone. Bone osteoid (the organic matrix laid down by osteoblasts) is mostly type I collagen. In scurvy, defective collagen synthesis means the osteoblasts cannot form normal osteoid matrix. So the primary defect is impaired osteoid (collagen) matrix formation, not a mineral problem.

**Step 3:** Contrast with rickets and osteomalacia. There the matrix (osteoid) is laid down normally but fails to mineralize because of vitamin D or phosphate problems, giving decreased mineralization (option A). That is the opposite of scurvy, where the matrix itself is deficient.

**Step 4:** Address the remaining distractors.
Increased bone resorption is the dominant feature of hyperparathyroidism, not the primary defect of scurvy.
Decreased bone mass with normal mineralization and osteoid (option D) describes osteoporosis, where both matrix and mineral are normal in quality but

reduced in quantity; scurvy specifically impairs matrix quality.

**Step 5:** The core lesion of scurvy is failure of osteoid (collagen) matrix formation.

The correct answer is option B, Decreased osteoid matrix formation.

**Quick Tip:** Vitamin C builds collagen, so scurvy fails to form osteoid matrix; rickets instead fails to mineralize.

**215.** Which of the following drugs is not used in Juvenile Myoclonic epilepsy (JME)?

(A) Topiramate

(B) Zonisamide

(C) Carbamazepine

(D) Valproate

**Correct Answer:** (C) Carbamazepine

**Solution:**

**Step 1:** Recognise that this is a negative lead question; we must find the drug that is NOT used, and in fact may worsen JME.

**Step 2:** Recall what JME is. Juvenile myoclonic epilepsy is an idiopathic generalized epilepsy of adolescence with early morning myoclonic jerks, generalized tonic clonic seizures, and sometimes absences. The key prescribing rule is that narrow spectrum (sodium channel blocking) drugs that suit focal epilepsy can aggravate generalized seizures, especially myoclonus.

**Step 3:** Evaluate each drug.
Valproate is the classic broad spectrum drug of choice for JME.
Topiramate and zonisamide are broad spectrum agents that are effective and used in JME.

Carbamazepine is a sodium channel blocker that is well known to worsen myoclonic and absence seizures and is therefore avoided in JME (along with phenytoin, oxcarbazepine, gabapentin, vigabatrin, tiagabine).

**Step 4:** The agent that is not used and can exacerbate JME is carbamazepine.

The correct answer is option C, Carbamazepine.

**Quick Tip:** Sodium channel blockers like carbamazepine worsen myoclonus; valproate is the JME drug of choice.

**216.** A child presents to the clinic with a history of seizures and mental retardation. Clinical examination reveals multiple hypopigmented macules. What is the likely diagnosis?

(A) Tuberous sclerosis

(B) Neurofibromatosis

(C) Sturge Weber syndrome

(D) Linear epidermal nevus syndrome

**Correct Answer:** (A) Tuberous sclerosis

**Solution:**

**Step 1:** Group the findings. A child with seizures, intellectual disability (mental retardation), and multiple hypopigmented skin macules points to a neurocutaneous syndrome.

**Step 2:** Match the triad. The classic triad of tuberous sclerosis is seizures, intellectual disability, and skin lesions, the earliest of which are hypopigmented ash leaf macules (best seen under a Wood's lamp). Other features include adenoma sebaceum (facial angiofibromas), shagreen patch, periungual fibromas, cortical tubers, and renal angiomyolipomas.

**Step 3:** Rule out the distractors.
Neurofibromatosis is characterised by hyperpigmented cafe au lait macules and neurofibromas, the opposite pigment pattern to the hypopigmented macules described here.
Sturge Weber syndrome features a facial port wine stain (capillary malformation) in the trigeminal distribution with leptomeningeal angiomas and seizures, not hypopigmented macules.
Linear epidermal nevus syndrome shows linear verrucous epidermal nevi, not hypopigmented macules.

**Step 4:** Seizures plus mental retardation plus hypopigmented (ash leaf) macules is tuberous sclerosis.

The correct answer is option A, Tuberous sclerosis.

**Quick Tip:** Seizures plus mental retardation plus hypopigmented ash leaf macules equals tuberous sclerosis.

**217.** Which of the following conditions presents with absence of both Mullerian and Wolffian duct structures?

(A) Antimullerian hormone deficiency

(B) Androgen insensitivity syndrome

(C) FSH receptor defect

(D) Ovotesticular syndrome

**Correct Answer:** (B) Androgen insensitivity syndrome

**Solution:**

**Step 1:** Recall the two internal duct systems. The Mullerian (paramesonephric) ducts form the uterus, fallopian tubes and upper vagina. The Wolffian (mesonephric) ducts form the epididymis, vas deferens and seminal vesicles. For both to be absent, the embryo must have signals that suppress Mullerian ducts

AND a failure of signals that maintain Wolffian ducts.
**Step 2:** In androgen insensitivity syndrome (AIS), the karyotype is 46,XY with functioning testes. The testes secrete anti-Mullerian hormone (AMH), so Mullerian structures regress and are absent. The testes also secrete testosterone, but because the androgen receptor is defective, the Wolffian ducts cannot respond and therefore fail to develop. So both duct systems are missing.
**Step 3:** Why the other options are wrong.
AMH deficiency (option A): testes still make androgens, so Wolffian ducts develop normally and Mullerian ducts persist. Wolffian structures are present, so it does not fit.
FSH receptor defect (option C): affects gonadal follicular function, not duct formation; ducts are not both absent.
Ovotesticular syndrome (option D): both ovarian and testicular tissue coexist, so duct development is mixed, not absent.
**Step 4:** Only AIS removes both Mullerian (via AMH) and Wolffian (via androgen receptor failure) structures. The answer is option B, Androgen insensitivity syndrome.

**Quick Tip:** AMH removes Mullerian ducts; a broken androgen receptor stops Wolffian ducts from forming.

**218.** A lady presented with secondary amenorrhea 6 months after having an abortion. Her FSH level was measured as 6 mIU/ml. What is the most probable diagnosis?

(A) Pituitary failure

(B) Ovarian failure

(C) Fresh pregnancy

(D) Uterine synechiae

**Correct Answer:** (D) Uterine synechiae

**Solution:**

**Step 1:** Note the trigger event. Amenorrhea that starts after a uterine procedure such as an abortion or dilatation and curettage points to intrauterine damage. Curettage can scar the endometrium and stick the uterine walls together, called Asherman syndrome (uterine synechiae).
**Step 2:** Use the hormone value. An FSH of 6 mIU/ml is normal. This is important because it rules out the conditions that change FSH.
Ovarian failure (option B) causes high FSH, usually >40 mIU/ml, because the ovary stops giving feedback. FSH of 6 is not high, so ovarian failure is excluded.
Pituitary failure (option A) causes low FSH, often very low, because the pituitary cannot make it. FSH of 6 is not low, so pituitary failure is excluded.
**Step 3:** Consider fresh pregnancy (option C). Pregnancy would cause amenorrhea, but the question frames this as a diagnostic puzzle after an abortion with a measured FSH; the classic teaching point here is the scarring problem, not a new pregnancy, and a simple beta-hCG would settle that. The examiner wants the post-curettage cause.
**Step 4:** With a normal FSH and a clear history of a uterine procedure, the ovary and pituitary are working but the endometrium or cavity is damaged. This is Asherman syndrome, the uterine synechiae. The answer is option D.

**Quick Tip:** Normal FSH after a curettage points to the cavity, not the ovary or pituitary.

**219.** Which of the following methods for assessment of female fertility during a menstrual cycle can best predict the timing of ovulation?

(A) Basal body temperature (BBT)

(B) Fern test

(C) Spinnbarkeit phenomenon

(D) Hormonal study

**Correct Answer:** (D) Hormonal study

**Solution:**

**Step 1:** Understand the difference between predicting ovulation and confirming it. To predict means to know it is about to happen so the couple can time intercourse; to confirm means to show it already happened.
**Step 2:** Evaluate each clinical method.
Basal body temperature (option A): the temperature rises by about 0.3 to 0.5 degrees Celsius only after ovulation, because of progesterone from the corpus luteum. So BBT confirms past ovulation, it does not predict it.
Fern test (option B): cervical mucus dries in a fern pattern under estrogen before ovulation, but it is qualitative and indirect.
Spinnbarkeit (option C): the stretchability of cervical mucus increases near mid-cycle under estrogen; again indirect and observer dependent.
**Step 3:** Evaluate hormonal study (option D). Measuring the LH surge in serum or urine pinpoints the trigger of ovulation, which follows the surge by roughly 24 to 36 hours. Serial estradiol and LH levels track the cycle precisely. This is the most accurate and direct way to predict the timing of ovulation.
**Step 4:** Since the question asks for the best predictor, the objective hormonal assay (LH surge) beats the indirect mucus tests and the retrospective BBT. The answer is option D, Hormonal study.

**Quick Tip:** BBT only tells you afterward; the LH surge tells you it is coming.

**220.** Primary amenorrhea with absent uterus, normal breasts and scant pubic hair is seen in:

(A) Mayer Rokitansky Kuster Hauser syndrome

(B) Turner's syndrome

(C) Androgen insensitivity syndrome

(D) Noonan syndrome

**Correct Answer:** (C) Androgen insensitivity syndrome

**Solution:**

**Step 1:** Break the stem into three findings: absent uterus, normal breasts, and scant or absent pubic hair. Each finding narrows the diagnosis.
**Step 2:** Absent uterus means the Mullerian structures regressed, which happens when AMH is present, that is, when testes exist (46,XY) or when Mullerian agenesis occurs.
**Step 3:** Normal breasts mean estrogen is active. In androgen insensitivity syndrome (AIS), the testosterone made by the testes is converted to estrogen and, because androgen action is blocked, this estrogen drives normal female breast development.
**Step 4:** Scant pubic hair is the key separator. Pubic and axillary hair depend on androgens. In AIS the androgen receptor is defective, so sexual hair is sparse or absent. This is the hallmark that points to AIS over Mullerian agenesis.
**Step 5:** Compare the distractors.
MRKH (option A): also has absent uterus and normal breasts, but pubic hair is NORMAL because the ovaries and androgen response are intact. So scant hair excludes MRKH.
Turner syndrome (option B): has a uterus present, streak ovaries, poor breast development and short stature; does not fit.
Noonan syndrome (option D): not a cause of this picture.
The combination of absent uterus, normal breasts and scant pubic hair is classic for AIS. The answer is option C.

**Quick Tip:** Normal breasts but scant pubic hair is the androgen insensitivity giveaway.

**221.** Which of the following agents is most commonly associated with carcinoma cervix?

(A) HPV 16

(B) HPV 18

(C) HPV 33

(D) HPV 35

**Correct Answer:** (A) HPV 16

**Solution:**

**Step 1:** Recall the cause of cervical cancer. Persistent infection with high-risk human papillomavirus (HPV) types is the central cause of nearly all cervical cancers.
**Step 2:** Rank the high-risk types. HPV 16 and HPV 18 together account for about 70 percent of cervical cancers. Of these two, HPV 16 is the single most common type, responsible for roughly 50 to 55 percent of cases worldwide.
**Step 3:** The viral oncoproteins E6 and E7 drive cancer. E6 degrades the p53 tumor suppressor and E7 inactivates the retinoblastoma (Rb) protein, removing the brakes on the cell cycle and allowing malignant transformation.
**Step 4:** Compare options. HPV 18 (option B) is the second most common and is linked more with adenocarcinoma, but it is less common overall than HPV 16. HPV 33 and HPV 35 (options C and D) are high-risk types but far less frequent. Therefore the single most commonly associated agent is HPV 16. The answer is option A.

**Quick Tip:** HPV 16 and 18 cause most cases, and 16 is the single most common.

**222.** A 52 year old lady presents with constant leakage of urine and dysuria two weeks after a complicated total abdominal hysterectomy. A diagnosis of vesicovaginal fistula is suspected. The most important test for the diagnosis is:

(A) Triple swab test

(B) Urine culture

(C) Cystoscopy

(D) Intravenous pyelography (IVP)

**Correct Answer:** (A) Triple swab test

**Solution:**

**Step 1:** Understand the clinical picture. Constant urine leakage after a hysterectomy strongly suggests a urinary fistula. The two candidates are

vesicovaginal fistula (bladder to vagina) and ureterovaginal fistula (ureter to vagina). The first diagnostic step is to confirm a fistula exists and to localise it.
**Step 2:** Recall the triple swab test (three swab or dye test). Three cotton swabs are placed in the vagina, then dye such as methylene blue is instilled into the bladder. The pattern of staining tells you the source:
top swab stained with clear urine suggests a ureterovaginal fistula (urine bypasses the bladder dye),
middle or lower swab stained blue suggests a vesicovaginal fistula,
blue staining at the introitus suggests urethral leak or stress incontinence.
**Step 3:** This simple bedside test both confirms the fistula and helps localise it, so it is the most important first diagnostic test.
**Step 4:** Why the others are not the first test.
Urine culture (option B): only checks for infection causing dysuria, does not diagnose a fistula.
Cystoscopy (option C): useful to see the fistula and plan repair, but it is a later, more invasive step.
IVP (option D): used mainly to exclude an associated ureteric injury, not the primary test for vesicovaginal fistula.
So the triple swab test is the answer, option A.

**Quick Tip:** The bedside dye test both confirms and localises the fistula first.

**223.** Which of the following statements about partial mole is false?

(A) Usually associated with triploidy

(B) Rarely causes persistent gestational trophoblastic neoplasia

(C) Usually presents as missed abortion

(D) Can be reliably diagnosed by USG in early gestation

**Correct Answer:** (D) Can be reliably diagnosed by USG in early gestation

**Solution:**

**Step 1:** Recall the features of a partial hydatidiform mole and check each statement, because the question asks which one is FALSE.
**Step 2:** Statement A, usually associated with triploidy. This is TRUE. A partial mole typically has 69 chromosomes (triploid), from one egg fertilised by two sperm or one diploid sperm. So A is correct.
**Step 3:** Statement B, rarely causes persistent gestational trophoblastic neoplasia. This is TRUE. Partial moles have a low risk of progressing to persistent trophoblastic disease, around 1 to 5 percent, far less than complete moles. So B is correct.
**Step 4:** Statement C, usually presents as missed abortion. This is TRUE. Partial moles often present as a missed or incomplete abortion rather than the classic grape-like passage, because there is a fetus or fetal tissue and only focal villous swelling.
**Step 5:** Statement D, can be reliably diagnosed by USG in early gestation. This is FALSE. Partial moles are notoriously hard to detect on early ultrasound, the changes are subtle and often missed, and many are only confirmed on histology after evacuation. So D is the false statement and the answer is option D.

**Quick Tip:** Early ultrasound often misses a partial mole; histology confirms it.

**224.** Conversion of a complete hydatidiform mole into an invasive mole is indicated by all of the following except:

(A) Plateau in hCG

(B) Enlarged uterine size

(C) Persistence of theca lutein cysts

(D) Suburethral nodule

**Correct Answer:** (D) Suburethral nodule

**Solution:**

**Step 1:** Understand the question. After evacuating a complete mole, certain signs warn that the disease has become an invasive mole or persistent trophoblastic

disease. The question asks which finding is NOT such a sign.
**Step 2:** Review the genuine warning signs.
Plateau in hCG (option A): if serial beta-hCG fails to fall and instead stays flat over several weeks, this signals persistent or invasive trophoblastic disease. So A is a true sign of conversion.
Enlarged uterine size (option B): a uterus that re-enlarges or stays bulky after evacuation suggests ongoing invasive trophoblastic tissue. So B is a true sign.
Persistence of theca lutein cysts (option C): these ovarian cysts form from high hCG; if they persist, hCG is still high, again indicating ongoing disease. So C is a true sign.
**Step 3:** Examine option D, suburethral nodule. A suburethral nodule is not a recognised marker of molar conversion. (Vaginal metastatic nodules of trophoblastic disease are typically suburethral or in the lower vagina in choriocarcinoma, but the listed term as a marker of invasive mole conversion is the odd one out here.)
**Step 4:** The three accepted indicators are plateauing hCG, persistent uterine enlargement and persistent theca lutein cysts. The exception is the suburethral nodule. The answer is option D.

**Quick Tip:** Plateauing hCG, a bulky uterus and persistent theca lutein cysts all warn of conversion.

**225.** A 40 year old woman presents with abnormal cervical cytology on PAP smear suggestive of CIN III (HSIL). The next best step in management is:

(A) Hysterectomy

(B) Colposcopy and LEEP

(C) Colposcopy and cryotherapy

(D) Conization

**Correct Answer:** (B) Colposcopy and LEEP

**Solution:**

**Step 1:** Understand the cytology finding. A PAP smear is only a screening test. CIN III on cytology means a high grade squamous intraepithelial lesion (HSIL) is suspected, but cytology cannot tell how deep the lesion goes or whether invasion has already occurred.

**Step 2:** Recall the rule that an abnormal screening smear must be confirmed by a diagnostic step before any treatment. For HSIL the standard next step is colposcopy, which lets you see the abnormal area and take a directed biopsy.

**Step 3:** Choose the procedure that both confirms and treats. In a see-and-treat approach for HSIL, colposcopy is combined with LEEP (loop electrosurgical excision procedure). LEEP removes the transformation zone, gives a tissue specimen for histology to rule out invasion, and treats the lesion in one sitting.

**Step 4:** Rule out the wrong options. Hysterectomy (A) is too aggressive for a pre-invasive lesion and removes the chance of histological assessment. Cryotherapy (C) is an ablative method, so no specimen is obtained and it is not preferred for high grade disease. Conization (D) is reserved when the lesion extends into the canal, colposcopy is unsatisfactory, or microinvasion is suspected, not as the routine first step.

**Answer: Colposcopy and LEEP (Option B).**

**Quick Tip:** A screening smear needs colposcopic confirmation; for HSIL pick the option that also yields a specimen.

**226.** Sentinel lymph node biopsy is most useful for which of the following gynaecological malignancies?

(A) Carcinoma endometrium

(B) Carcinoma cervix

(C) Carcinoma vulva

(D) Carcinoma vagina

**Correct Answer:** (C) Carcinoma vulva

**Solution:**

**Step 1:** Recall what a sentinel lymph node is. It is the first node that drains a tumour. If it is free of cancer, the rest of that nodal basin is very likely free too, so a full lymphadenectomy can be avoided along with its morbidity.

**Step 2:** Identify where this concept is best established in gynaecology. Vulvar carcinoma has a predictable, superficial lymphatic drainage to the inguinofemoral nodes, which can be mapped easily with blue dye and radiotracer. Full inguinofemoral lymphadenectomy causes severe wound breakdown and lifelong leg lymphoedema, so a reliable way to skip it is highly valuable.

**Step 3:** Apply this to early vulvar cancer. In early stage, unifocal vulvar carcinoma the sentinel node technique is validated and is the recommended approach, sparing many women from radical groin dissection.

**Step 4:** Eliminate the other sites. Endometrial cancer drainage is deep and more variable, and sentinel mapping there became standard only later. Cervical and vaginal lymphatic drainage is complex and multidirectional, making sentinel mapping far less reliable at the time and in general.

**Answer: Carcinoma vulva (Option C).**

**Quick Tip:** Think which gynae cancer has simple, superficial, predictable inguinal drainage.

**227.** All of the following are associated with polycystic ovarian syndrome, except:

(A) Ovarian carcinoma

(B) Endometrial carcinoma

(C) Insulin resistance

(D) Osteoporosis

**Correct Answer:** (D) Osteoporosis

**Solution:**

**Step 1:** Recall the core hormonal picture of PCOS. There is chronic anovulation, raised androgens, and a relative excess of unopposed oestrogen because regular progesterone-producing corpora lutea are not formed.

**Step 2:** Link the hormones to risk. Unopposed oestrogen continuously stimulates the endometrium, raising the risk of endometrial hyperplasia and endometrial carcinoma (B). PCOS is also strongly tied to insulin resistance and hyperinsulinaemia, which drives the metabolic syndrome and worsens the hyperandrogenism (C). There is a modest reported association with ovarian carcinoma as well (A).

**Step 3:** Test the bone option. Osteoporosis comes from oestrogen deficiency, as in menopause or hypothalamic amenorrhoea. In PCOS oestrogen levels are normal to high, not low, so the bones are not deprived of oestrogen. Hence osteoporosis is not a feature.

**Step 4:** Conclude. The odd one out is osteoporosis because it requires an oestrogen-deficient state, the opposite of the unopposed-oestrogen environment of PCOS.

**Answer: Osteoporosis (Option D).**

**Quick Tip:** PCOS has unopposed oestrogen, so which listed problem actually needs low oestrogen?

**228.** Which of the following is the most specific marker for neural tube defects?

(A) Acetylcholinesterase

(B) Pseudocholinesterase

(C) Alpha fetoprotein (AFP)

(D) Human chorionic gonadotrophin (HCG)

**Correct Answer:** (A) Acetylcholinesterase

**Solution:**

**Step 1:** Separate screening from confirmation. In an open neural tube defect, fetal proteins leak into the amniotic fluid and then into maternal serum. Maternal serum AFP rises, which is why it is used to screen, but a raised AFP has many other causes.

**Step 2:** List why AFP is sensitive but not specific. Maternal serum AFP also rises with open abdominal wall defects, multiple pregnancy, wrong gestational dating, and fetal demise. So AFP flags the problem but cannot confirm it.

**Step 3:** Bring in the confirmatory marker. When AFP is high, amniotic fluid is tested for acetylcholinesterase (AChE). AChE is a neural enzyme that escapes only when neural tissue is exposed, as in an open neural tube defect. Its presence in amniotic fluid is far more specific for an open NTD than AFP.

**Step 4:** Reject the rest. Pseudocholinesterase is a liver-derived enzyme unrelated to NTD. HCG is a placental marker used in aneuploidy screening, not for NTD.

**Answer: Acetylcholinesterase (Option A).**

**Quick Tip:** AFP screens, but which neural enzyme in amniotic fluid actually confirms an open NTD?

**229.** AFP (alpha fetoprotein) is raised in:

(A) Teratoma

(B) Yolk sac tumour

(C) Choriocarcinoma

(D) Dysgerminoma

**Correct Answer:** (B) Yolk sac tumour

**Solution:**

**Step 1:** Match each germ cell tumour to its tumour marker. AFP is produced by yolk sac elements, while beta-HCG is produced by trophoblastic (syncytiotrophoblast) elements.

**Step 2:** Apply this to the yolk sac tumour. The endodermal sinus tumour, or yolk sac tumour, is the classic AFP-secreting ovarian germ cell tumour. Its Schiller-Duval bodies reflect yolk sac differentiation, and serum AFP is the marker used to diagnose and monitor it.

**Step 3:** Check the distractors. Choriocarcinoma (C) is trophoblastic and secretes beta-HCG, not AFP. Pure dysgerminoma (D) secretes LDH and may show mild placental alkaline phosphatase, but not AFP. A pure mature teratoma (A) is typically marker negative; only if it contains yolk sac elements would AFP rise, so as a single best answer the teratoma is not the AFP tumour.

**Step 4:** Conclude. The tumour most directly and characteristically associated with raised AFP is the yolk sac tumour.

**Answer: Yolk sac tumour (Option B).**

**Quick Tip:** AFP tracks yolk sac differentiation; beta-HCG tracks trophoblast.

**230.** Weight gain in pregnancy depends on all of the following factors, except:

(A) Socioeconomic status

(B) Prepregnancy weight

(C) Smoking

(D) Ethnicity

**Correct Answer:** (C) Smoking

**Solution:**

**Step 1:** Recall how gestational weight gain targets are set. The single biggest determinant is the prepregnancy weight or BMI: a woman who starts underweight is advised to gain more, and one who starts obese is advised to gain less. So prepregnancy weight (B) is a true determinant.

**Step 2:** Account for background factors. Socioeconomic status affects nutrition, food access, and the pattern of weight gain (A), and ethnicity influences body composition and recommended ranges (D). Both genuinely modify gestational weight gain.

**Step 3:** Examine smoking. Smoking is a well known cause of fetal growth restriction and lower birth weight, and it tends to lower, not determine, maternal and fetal weight. It is not listed among the standard factors that the recommended maternal weight gain is based upon. In the context of this question it is the exception.

**Step 4:** Conclude. Prepregnancy weight, socioeconomic status, and ethnicity are the determinants used to plan weight gain, so smoking is the odd one out.

**Answer: Smoking (Option C).**

**Quick Tip:** Three options set the recommended weight gain target; one is just a harmful exposure.

**231.** Which of the following statements about multiple pregnancies is true?

(A) Fetuses of the same gender exclude dichorionicity

(B) Twin peak sign is seen in dichorionicity

(C) Thick separating membrane is a feature of monochorionic twins

(D) Chorionicity can be reliably detected only after 16 weeks of gestation

**Correct Answer:** (B) Twin peak sign is seen in dichorionicity

**Solution:**

**Step 1:** Understand why chorionicity matters and how it is read on ultrasound. The key sign is at the base of the dividing membrane where it meets the placenta.

**Step 2:** Define the twin peak sign. In dichorionic twins, placental tissue extends up into the base of the dividing membrane, creating a triangular wedge called the twin peak or lambda sign. In monochorionic twins the membrane meets the placenta at a right angle, giving the thin T sign. So option B is correct.

**Step 3:** Knock out the false statements. Same gender does not exclude dichorionicity, because dizygotic same sex twins exist and monozygotic twins can be dichorionic, so A is wrong. A thick dividing membrane indicates a dichorionic pregnancy (it contains two amnions and two chorions), not monochorionic, so C is wrong. Chorionicity is read most reliably in the first trimester, roughly 10 to 14 weeks, when the lambda and T signs are clearest, so D is wrong.

**Step 4:** Conclude. The only true statement is that the twin peak sign indicates dichorionicity.

**Answer: Twin peak sign is seen in dichorionicity (Option B).**

**Quick Tip:** Lambda or twin peak sign means a wedge of placenta, which means two chorions.

**232.** Treatment of choice for intrahepatic cholestasis in pregnancy is:

(A) Cholestyramine

(B) Ursodiol (ursodeoxycholic acid)

(C) Corticosteroids (dexamethasone)

(D) Antihistaminics

**Correct Answer:** (B) Ursodiol (ursodeoxycholic acid)

**Solution:**

**Step 1:** Recognise the condition. Intrahepatic cholestasis of pregnancy presents with intense pruritus, especially of the palms and soles, in the late second or third trimester, with raised serum bile acids and often raised transaminases. The danger is to the fetus from high circulating bile acids.

**Step 2:** Decide the goal of treatment. The aim is to lower maternal serum bile acids, relieve itching, and reduce fetal risk. The drug that does this best is ursodeoxycholic acid.

**Step 3:** Explain why ursodiol is first line. Ursodeoxycholic acid improves bile flow, lowers the concentration of toxic hydrophobic bile acids, relieves pruritus, and improves liver enzymes. It is the recommended treatment of choice and is considered safe in pregnancy.

**Step 4:** Reject the alternatives. Cholestyramine (A) binds bile acids in the gut but is less effective for itch, worsens fat malabsorption, and can lower vitamin K and worsen bleeding risk, so it is second line at best. Corticosteroids (C) and antihistamines (D) give only limited or symptomatic benefit and do not address bile acids, so they are not the treatment of choice.

**Answer: Ursodiol (ursodeoxycholic acid) (Option B).**

**Quick Tip:** Pick the drug that actually lowers maternal bile acids, not just the itch.

**233.** All of the following are cardiac contraindications to pregnancy, except:

(A) Eisenmenger's syndrome

(B) Pulmonary hypertension

(C) Coarctation of aorta

(D) WPW syndrome

**Correct Answer:** (D) WPW syndrome

**Solution:**

**Step 1:** The question asks which condition is NOT a contraindication to pregnancy. Pregnancy nearly doubles cardiac output and plasma volume, so lesions that fix or limit cardiac output, or that depend on a delicate pressure balance, become dangerous.

**Step 2:** Eisenmenger's syndrome carries a maternal mortality of about 30 to 50 percent. The fall in systemic vascular resistance during pregnancy worsens the right-to-left shunt and causes hypoxia. It is an absolute contraindication.

**Step 3:** Severe pulmonary hypertension (pulmonary artery pressure > two-thirds systemic) is one of the highest-risk states, with maternal mortality of 30 to 50 percent. The fixed pulmonary circulation cannot accommodate the increased volume load. Absolute contraindication.

**Step 4:** Coarctation of aorta, especially uncorrected with associated aneurysm, risks aortic dissection and rupture during the haemodynamic stress of pregnancy, so it is also regarded as a contraindication.

**Step 5:** Wolff-Parkinson-White (WPW) syndrome is a pre-excitation arrhythmia from an accessory pathway. It is managed with drugs or ablation and does not impose a fixed haemodynamic limit. It is not a contraindication to pregnancy. Hence the exception is WPW syndrome, option D.

**Quick Tip:** Three options fix or strain cardiac output; one is just an arrhythmia.

**234.** Two related recalls. (a) The drug of choice to treat Chlamydia infection in pregnancy, when azithromycin is an option, is: A. Tetracycline, B. Doxycycline, C. Erythromycin, D. Azithromycin (Ans D). (b) When azithromycin is not listed and the choice is between A. Tetracycline, B. Doxycycline, C. Erythromycin, D. Penicillin, the drug of choice is Erythromycin (Ans C). The single combined answer below addresses the version that lists azithromycin. Which is the drug of choice for Chlamydia in pregnancy?

(A) Tetracycline

(B) Doxycycline

(C) Erythromycin

(D) Azithromycin

**Correct Answer:** (D) Azithromycin

**Solution:**

**Step 1:** Chlamydia trachomatis in pregnancy must be treated with a drug that is both effective against the organism and safe for the fetus.
**Step 2:** Tetracycline and doxycycline are tetracyclines. They are contraindicated in pregnancy because they cause permanent staining of fetal teeth and inhibit bone growth. So options A and B are ruled out.
**Step 3:** Erythromycin (a macrolide) is safe in pregnancy and was the traditional drug of choice, but it requires a longer course and is poorly tolerated due to gastrointestinal side effects, which lowers compliance.
**Step 4:** Azithromycin is also a macrolide and is safe in pregnancy. It is given as a single 1 g oral dose, which gives excellent compliance and cure rates. Current CDC guidance makes azithromycin the preferred single-dose drug of choice.
**Step 5:** Therefore, when azithromycin is offered as a choice, it is the answer (option D). In the older version where azithromycin is absent and penicillin is listed, penicillin is not reliable against Chlamydia, so erythromycin becomes the answer for that set.
For this version the answer is azithromycin, option D.

**Quick Tip:** Tetracyclines are banned in pregnancy; pick the single-dose macrolide.

**235.** Which of the following conditions is associated with polyhydramnios?

(A) Posterior urethral valves

(B) Cleft palate

(C) Congenital diaphragmatic hernia

(D) Bladder exstrophy

**Correct Answer:** (B) Cleft palate

**Solution:**

**Step 1:** Amniotic fluid volume reflects a balance between fetal urine production (which adds fluid) and fetal swallowing (which removes fluid). Polyhydramnios (excess fluid) usually means the fetus cannot swallow normally or there is a gut obstruction.
**Step 2:** Posterior urethral valves cause bladder outlet obstruction and reduced urine output, so they classically cause oligohydramnios, not polyhydramnios. Option A is wrong.
**Step 3:** Bladder exstrophy involves an open bladder but does not impair swallowing or reduce overall urine entering the amniotic cavity, so it is not a recognised cause of polyhydramnios. Option D is wrong.
**Step 4:** Congenital diaphragmatic hernia can be associated with polyhydramnios when the herniated stomach kinks and obstructs swallowing, but the more consistent and classic association offered here is cleft palate.
**Step 5:** Cleft palate impairs the fetal ability to swallow amniotic fluid effectively, so fluid accumulates and polyhydramnios results. This is the best answer.
Hence the answer is cleft palate, option B.

**Quick Tip:** Polyhydramnios follows impaired fetal swallowing; oligohydramnios follows poor urine output.

**236.** Increased nuchal translucency at 14 weeks gestation is seen in:

(A) Turner's syndrome

(B) Down's syndrome

(C) Hydrocephalus

(D) Skeletal dysplasia

**Correct Answer:** (B) Down's syndrome

**Solution:**

**Step 1:** Nuchal translucency (NT) is the sonographic measurement of the fluid collection at the back of the fetal neck, taken between 11 and 14 weeks. An increased NT is a marker of chromosomal and structural abnormality.
**Step 2:** Increased NT is most strongly and most commonly linked with Down's syndrome (trisomy 21), where it forms part of the combined first-trimester screen along with maternal age, free beta-hCG and PAPP-A.
**Step 3:** Turner's syndrome (45,X) can also raise NT, but in Turner's the classic finding is a cystic hygroma and a much larger nuchal collection rather than the modest NT increase, and it is not the single best answer the examiner wants here.
**Step 4:** Hydrocephalus and skeletal dysplasia are not first-trimester NT markers in the way trisomy 21 is; they present later with their own specific findings.
**Step 5:** Because Down's syndrome is the classic and most examined association of increased NT, the answer is Down's syndrome.
Hence the answer is Down's syndrome, option B.

**Quick Tip:** Increased nuchal translucency is the classic first-trimester marker of trisomy 21.

**237.** Non-immune hydrops fetalis is caused by:

(A) CMV

(B) Parvovirus

(C) HSV

(D) HIV

**Correct Answer:** (B) Parvovirus

**Solution:**

**Step 1:** Hydrops fetalis is abnormal fluid accumulation in two or more fetal compartments. Non-immune hydrops is the type not caused by red-cell alloimmunisation (not Rh disease).
**Step 2:** Among infective causes, Parvovirus B19 is the classic and most important. The virus infects erythroid progenitor cells in the fetal marrow, halting red cell production and causing severe aplastic anaemia.
**Step 3:** The severe anaemia leads to high-output cardiac failure and tissue hypoxia, which drives the generalised oedema and effusions of hydrops.
**Step 4:** CMV can cause fetal infection and occasionally hydrops, but its hallmark is intracranial calcification, microcephaly and growth restriction rather than the marrow-suppression hydrops of parvovirus. HSV and HIV are not classic causes of non-immune hydrops.
**Step 5:** The best-known infective cause of non-immune hydrops fetalis is Parvovirus B19.
Hence the answer is Parvovirus, option B.

**Quick Tip:** Which virus wipes out fetal red-cell precursors and causes severe anaemia?

**238.** A lady presented with features of threatened abortion at 32 weeks of pregnancy. Which of the following statements with regard to antibiotic usage is not correct?

(A) Antibiotic prophylaxis even with unruptured membranes

(B) Metronidazole if asymptomatic but significant bacterial vaginosis

(C) Antibiotics if asymptomatic but significant bacteriuria

(D) Antibiotics for preterm premature rupture of membranes

**Correct Answer:** (A) Antibiotic prophylaxis even with unruptured membranes

**Solution:**

**Step 1:** The question asks which statement about antibiotic use in threatened preterm labour is incorrect. The evidence base is the ORACLE trials and standard obstetric guidance.
**Step 2:** Routine antibiotic prophylaxis when membranes are intact (unruptured) does not help and may even harm. The ORACLE II trial showed no benefit in preterm labour with intact membranes and signalled possible long-term harm to the child. So giving antibiotics with unruptured membranes is not correct. Option A is the wrong statement and therefore the answer.
**Step 3:** Treating significant bacterial vaginosis with metronidazole even if asymptomatic is reasonable in pregnancy because BV is linked to preterm birth, so option B is a correct statement.
**Step 4:** Asymptomatic bacteriuria in pregnancy must be treated to prevent pyelonephritis and preterm labour, so option C (antibiotics for significant bacteriuria) is correct.
**Step 5:** Preterm premature rupture of membranes is a clear indication for antibiotics (erythromycin), which prolong latency and reduce infection. Option D is correct.
The statement that is not correct is option A, antibiotic prophylaxis with unruptured membranes.

**Quick Tip:** Recall ORACLE II: prophylaxis with intact membranes gives no benefit.

**239.** A woman presents with leakage of fluid per vaginum and meconium stained liquor at 34 weeks of gestation. The most likely organism causing infection would be:

(A) Listeria monocytogenes

(B) Toxoplasmosis

(C) CMV

(D) Herpes

**Correct Answer:** (A) Listeria monocytogenes

**Solution:**

**Step 1:** The clinical picture is preterm rupture of membranes (leakage of fluid) at 34 weeks with meconium-stained liquor. Meconium passage at this early gestation is unusual and is a recognised clue to intrauterine Listeria infection.
**Step 2:** Listeria monocytogenes is acquired from contaminated food (unpasteurised dairy, deli meats). In pregnancy it crosses the placenta and causes chorioamnionitis, preterm labour, and the characteristic early meconium staining of the liquor.
**Step 3:** Meconium-stained liquor before term is the classic examination flag for Listeria, distinguishing it from the other listed organisms.
**Step 4:** Toxoplasma and CMV cause congenital infection syndromes (calcification, chorioretinitis, growth restriction) rather than acute meconium-stained PROM. Herpes causes neonatal skin, eye and CNS disease, typically through birth-canal exposure, not this picture.
**Step 5:** The organism best fitting preterm membrane rupture with meconium-stained liquor is Listeria monocytogenes.
Hence the answer is Listeria monocytogenes, option A.

**Quick Tip:** Early meconium-stained liquor with membrane rupture is the classic Listeria clue.

**240.** Two related recalls on active management of the third stage of labour. (a) Which of the following interventions is not recommended in active management of the third stage of labour? A. Administration of uterotonic within 1 minute of delivery, B. Immediate clamping, cutting and ligation of cord, C. Gentle massage of uterus, D. Controlled cord traction (Ans B). (b) Active management of the third stage of labour includes all of the following except: A. Oxytocin injection, B. Ergometrine injection, C. Controlled cord traction, D. Gentle massage of uterus. The combined answer below addresses part (a). Which intervention is not recommended in active management of the third stage?

(A) Administration of uterotonic within 1 minute of delivery

(B) Immediate clamping, cutting and ligation of cord

(C) Gentle massage of uterus

(D) Controlled cord traction

**Correct Answer:** (B) Immediate clamping, cutting and ligation of cord

**Solution:**

**Step 1:** Active management of the third stage of labour (AMTSL) is the package used to reduce postpartum haemorrhage. Its core components are a uterotonic, controlled cord traction, and uterine assessment/massage.
**Step 2:** A uterotonic (oxytocin) given soon after delivery of the baby is a key, recommended step. So option A is recommended, not the answer.
**Step 3:** Controlled cord traction with counter-pressure on the uterus helps deliver the placenta and is part of AMTSL. Option D is recommended.
**Step 4:** Gentle uterine massage after the placenta is delivered keeps the uterus contracted and reduces bleeding, so option C is recommended.
**Step 5:** Immediate cord clamping is NOT recommended. WHO advises delayed cord clamping (after 1 to 3 minutes) for better neonatal iron stores; early routine clamping is no longer part of good AMTSL. So option B is the intervention not recommended and is the answer for part (a).
For part (b), where the choices are oxytocin, ergometrine, controlled cord traction, and uterine massage, the formal AMTSL components are the uterotonic, controlled cord traction and massage, so ergometrine as a separate routine injection is the relative exception there.
The answer to part (a) is immediate clamping and ligation of cord, option B.

**Quick Tip:** Modern AMTSL favours delayed, not immediate, cord clamping.

**241.** All of the following maneuvers are used in shoulder dystocia, except:

(A) Woods corkscrew maneuver

(B) McRoberts maneuver

(C) Suprapubic pressure

(D) Mauriceau-Smellie-Veit maneuver

**Correct Answer:** (D) Mauriceau-Smellie-Veit maneuver

**Solution:**

**Step 1:** Shoulder dystocia is an obstetric emergency where, after the head delivers, the anterior shoulder gets stuck behind the pubic symphysis and routine traction fails to deliver the baby. The aim of every maneuver is to disimpact that anterior shoulder.
**Step 2:** McRoberts maneuver (hyperflexion of the maternal thighs onto the abdomen) flattens the sacral promontory and rotates the symphysis upward, freeing the shoulder. It is the first-line step.
**Step 3:** Suprapubic pressure pushes the impacted anterior shoulder away from the midline so it slips under the symphysis. It is used along with McRoberts.
**Step 4:** Woods corkscrew maneuver rotates the posterior shoulder by 180 degrees to bring it under the symphysis and free the impaction. It is a recognised internal rotation maneuver.
**Step 5:** The Mauriceau-Smellie-Veit maneuver is used to deliver the AFTER-COMING HEAD in a breech (vaginal breech delivery), not for shoulder dystocia. So it is the exception here.
Hence the answer is **Option D (Mauriceau-Smellie-Veit maneuver)**.

**Quick Tip:** Which listed maneuver actually belongs to vaginal breech delivery of the after-coming head?

**242.** All of the following interventions are recommended to prevent mother-to-child transmission of HIV, except:

(A) Avoid ergometrine in the third stage of labour

(B) Highly active antiretroviral therapy (HAART)

(C) Elective Caesarean section

(D) Intrapartum Zidovudine

**Correct Answer:** (A) Avoid ergometrine in the third stage of labour

**Solution:**

**Step 1:** Prevention of mother-to-child transmission (PMTCT) of HIV works by lowering the maternal viral load and reducing the baby's exposure to infected blood and secretions during birth.
**Step 2:** HAART given to the mother brings the plasma viral load down, ideally to undetectable, which is the single most effective measure to cut vertical transmission. So Option B is recommended.
**Step 3:** Elective (pre-labour) Caesarean section at term reduces transmission when the maternal viral load is high or unknown, because it avoids the baby passing through the birth canal and reduces contact with maternal blood. So Option C is recommended.
**Step 4:** Intrapartum intravenous Zidovudine for the mother, with neonatal prophylaxis afterwards, further lowers the risk during the high-exposure delivery window. So Option D is recommended.
**Step 5:** Ergometrine is a uterotonic used to prevent and treat postpartum haemorrhage. Avoiding it has no role in PMTCT; in fact, controlling blood loss matters. The caution about ergometrine relates to its interaction with protease inhibitors (risk of severe vasoconstriction), not to HIV transmission itself. So routinely "avoiding ergometrine" is NOT a PMTCT intervention.
Hence the answer is **Option A (Avoid ergometrine in the third stage of labour)**.

**Quick Tip:** Three options lower viral load or birth-canal exposure; one is just a uterotonic choice.

**243.** A person with prolonged use of contact lenses presented with irritation of the left eye. After examination, a diagnosis of keratitis was made and corneal scrapings revealed growth of Pseudomonas aeruginosa. The bacteria were observed to be multidrug resistant. Which of the following best explains the mechanism of antimicrobial resistance in these isolated Pseudomonas aeruginosa strains?

(A) Ability to transfer resistance genes from adjacent commensal flora

(B) Improper contact lens hygiene

(C) Frequent and injudicious use of topical antibiotics

(D) Ability of Pseudomonas to produce biofilms

**Correct Answer:** (D) Ability of Pseudomonas to produce biofilms

**Solution:**

**Step 1:** The question asks for the MECHANISM of multidrug resistance in Pseudomonas aeruginosa associated with contact lens keratitis, not the risk factor for getting infected.
**Step 2:** Pseudomonas aeruginosa readily forms biofilms, a matrix of secreted polysaccharide (glycocalyx) in which the bacteria embed themselves, including on the surface of soft contact lenses and the cornea.
**Step 3:** Within a biofilm, antibiotics penetrate poorly, the bacteria grow slowly, and protected sub-populations survive. This makes the organism phenotypically resistant to many drugs at once, explaining the multidrug-resistant behaviour in this clinical setting.
**Step 4:** Option B (poor lens hygiene) and Option C (overuse of topical antibiotics) are risk factors or selection pressures, not the intrinsic mechanism by which this strain resists multiple drugs. Option A (gene transfer from commensals) is a general resistance route but is not the characteristic, lens-associated mechanism for Pseudomonas keratitis.
**Step 5:** The biofilm explains why the contact-lens-related Pseudomonas is hard to eradicate and resists several antibiotics simultaneously.
Hence the answer is **Option D (Ability of Pseudomonas to produce biofilms)**.

**Quick Tip:** Think about the slimy matrix this organism builds on plastic lens surfaces.

**244.** Endophthalmitis involves inflammation of all of the following, except:

(A) Sclera

(B) Uvea

(C) Retina

(D) Vitreous

**Correct Answer:** (A) Sclera

**Solution:**

**Step 1:** Endophthalmitis means inflammation of the INTERNAL contents of the eyeball, mainly the intraocular cavities and tissues, usually due to infection.
**Step 2:** It involves the vitreous and aqueous (the intraocular fluids), the uvea (uveal inflammation), and the adjacent retina. So the uvea, retina, and vitreous are all affected.
**Step 3:** The sclera is the tough outer coat of the eye. When inflammation extends to involve all coats INCLUDING the sclera and orbital tissues, the condition is called panophthalmitis, not endophthalmitis.
**Step 4:** Therefore, by definition, endophthalmitis spares the sclera; involvement of the sclera marks a more extensive process (panophthalmitis).
Hence the answer is **Option A (Sclera)**.

**Quick Tip:** Once the outer white coat is involved, the term changes to panophthalmitis.

**245.** Which of the following is the least common corneal dystrophy?

(A) Macular dystrophy

(B) Lattice type I

(C) Lattice type III

(D) Granular corneal dystrophy

**Correct Answer:** (A) Macular dystrophy

**Solution:**

**Step 1:** Among the classic stromal corneal dystrophies, the three to compare are granular, lattice, and macular dystrophy.
**Step 2:** Granular dystrophy is the most common of the stromal dystrophies, characterised by discrete hyaline deposits that stain with Masson trichrome.
**Step 3:** Lattice dystrophy is the next, with amyloid deposits forming branching lattice lines that stain with Congo red; the type I form is a well-recognised, relatively frequent variant.
**Step 4:** Macular dystrophy is the LEAST common of these stromal dystrophies. It is autosomal recessive, deposits glycosaminoglycans (mucopolysaccharide) that stain with Alcian blue and colloidal iron, involves the cornea diffusely up to the limbus, and is the most severe but the rarest.
**Step 5:** So comparing frequency, granular is commonest, lattice is intermediate, and macular dystrophy is the rarest among the options.
Hence the answer is **Option A (Macular dystrophy)**.

**Quick Tip:** Rank granular, lattice, and macular by frequency; the rarest deposits mucopolysaccharide.

**246.** Cherry-red spot at the macula is seen in all of the following, except:

(A) Niemann-Pick disease

(B) GM1 gangliosidosis

(C) Tay-Sachs disease

(D) Gaucher's disease

**Correct Answer:** (D) Gaucher's disease

**Solution:**

**Step 1:** A cherry-red spot is produced when lipid-laden ganglion cells make the perifoveal retina look pale and opaque, while the thin fovea (which has no

ganglion cells) still shows the normal red choroidal colour underneath, giving a red dot in a pale background.
**Step 2:** Tay-Sachs disease (GM2 gangliosidosis) is the classic cause of a cherry-red spot, so Option C shows it.
**Step 3:** GM1 gangliosidosis also accumulates ganglioside in retinal ganglion cells and produces a cherry-red spot, so Option B shows it.
**Step 4:** Niemann-Pick disease (especially type A) stores sphingomyelin in retinal ganglion cells and produces a cherry-red spot, so Option A shows it.
**Step 5:** Gaucher's disease is a glucocerebrosidase deficiency that mainly affects the liver, spleen, bone marrow, and bones; it does NOT classically produce a macular cherry-red spot. So it is the exception.
**Note on the key:** The recalled key was printed as "None," which is medically incorrect because Gaucher's disease is the clear exception. The corrected answer is Option D (Gaucher's disease).
Hence the answer is **Option D (Gaucher's disease)**.

**Quick Tip:** Which lysosomal storage disorder loads macrophages of spleen and marrow, not retinal ganglion cells?

**247.** Relative afferent pupillary defect (RAPD) is characteristically seen in damage to:

(A) Optic nerve

(B) Optic tract

(C) Lateral geniculate body

(D) Oculomotor nerve

**Correct Answer:** (A) Optic nerve

**Solution:**

**Step 1:** A relative afferent pupillary defect (RAPD), detected by the swinging flashlight test, reflects asymmetry in the AFFERENT (sensory) limb of the pupillary light reflex between the two eyes.
**Step 2:** The afferent limb runs from the retina along the optic nerve; light input

from each eye is then shared (decussated) so both pupils normally constrict equally to light in either eye.
**Step 3:** A unilateral optic nerve lesion (for example optic neuritis) reduces light signal from that eye. When the light swings to the affected eye, both pupils paradoxically dilate because the afferent input is weaker. This is the classic RAPD. So Option A is correct.
**Step 4:** The oculomotor nerve carries the EFFERENT limb. Its damage causes a fixed dilated pupil on that side, not a relative afferent defect, so Option D is wrong.
**Step 5:** Because of the extensive crossing of fibres, lesions of the optic tract or lateral geniculate body produce visual field defects but typically not a clinically obvious RAPD as seen with optic nerve disease; so Options B and C are not the characteristic site.
Hence the answer is **Option A (Optic nerve)**.

**Quick Tip:** RAPD is an input-side defect found with the swinging flashlight test; which structure carries that afferent signal?

**248.** A patient with ptosis presents with retraction of the ptotic eyelid on chewing. This represents:

(A) Marcus Gunn jaw-winking syndrome

(B) Third nerve misdirection syndrome

(C) Abducent palsy

(D) Oculomotor palsy

**Correct Answer:** (A) Marcus Gunn jaw-winking syndrome

**Solution:**

**Step 1:** The key clinical clue is a drooping (ptotic) eyelid that suddenly lifts (retracts) when the patient chews or moves the jaw.
**Step 2:** This is a synkinesis between the trigeminal nerve (motor branch to the pterygoid muscles, which move the jaw) and the oculomotor nerve branch to the

levator palpebrae superioris (which lifts the lid).
**Step 3:** When the patient activates the pterygoids during chewing or lateral jaw movement, the abnormal connection co-fires the levator, so the ptotic lid elevates. This is the Marcus Gunn jaw-winking phenomenon, a congenital trigemino-oculomotor synkinesis.
**Step 4:** Third nerve misdirection (aberrant regeneration) follows oculomotor nerve injury and links eye movements to lid or pupil behaviour, but it is not triggered by jaw movement; so Option B is wrong.
**Step 5:** Abducent palsy (Option C) causes failure of lateral gaze, and a simple oculomotor palsy (Option D) causes ptosis with a fixed dilated eye, but neither shows lid retraction on chewing.
Hence the answer is **Option A (Marcus Gunn jaw-winking syndrome)**.

**Quick Tip:** A trigeminal-oculomotor cross-wiring makes the drooping lid lift during jaw movement.

**249.** Otoacoustic emissions arise from:

(A) Inner hair cells

(B) Outer hair cells

(C) Organ of Corti

(D) Both outer and inner hair cells

**Correct Answer:** (B) Outer hair cells

**Solution:**

**Step 1:** Otoacoustic emissions (OAEs) are low-level sounds produced by the cochlea and recorded in the external ear canal with a sensitive microphone. They reflect the active mechanical process happening inside the cochlea.
**Step 2:** The cochlea has two types of hair cells. Inner hair cells are the true sensory receptors that send signals up the auditory nerve. Outer hair cells are motile and act as a built-in amplifier, the so-called cochlear amplifier.
**Step 3:** When outer hair cells contract and expand in response to sound, they

generate mechanical energy that travels back out through the middle ear. This back-travelling energy is the source of OAEs.
**Step 4:** So OAEs specifically test outer hair cell function. They are absent when outer hair cells are damaged, which is why OAEs are used for newborn hearing screening.
**Step 5:** Inner hair cells (A) generate the afferent nerve signal, not the emission. Organ of Corti (C) is the whole structure, too broad to be the specific source. Option D is wrong because inner hair cells do not produce the emission.
Hence the answer is **Option B: Outer hair cells**.

**Quick Tip:** Think about which cochlear cells are motile and act as the cochlear amplifier, the basis of newborn hearing screening.

**250.** Which of the following statements regarding corneal transplantation is true?

(A) Whole eye needs to be preserved in tissue culture

(B) Donor not accepted if age is more than 60 years

(C) Specular microscopy analysis is used to assess endothelial cell count

(D) HLA matching is mandatory

**Correct Answer:** (C) Specular microscopy analysis is used to assess endothelial cell count

**Solution:**

**Step 1:** The cornea is avascular. Its clarity depends on the corneal endothelium, a single layer of cells that pumps water out of the stroma. The quality of donor tissue is judged mainly by endothelial cell density.
**Step 2:** Specular microscopy projects light onto the donor cornea and images the endothelial mosaic. It counts cells per square millimetre and assesses their shape. A healthy graft usually needs more than about 2000 cells per square millimetre. So option C is correct.
**Step 3:** Option A is wrong. The whole eyeball is not kept in tissue culture. The corneoscleral disc is excised and stored in media such as McCarey-Kaufman

medium or organ culture; the whole globe is only briefly preserved by moist chamber storage.
**Step 4:** Option B is wrong. Older donors are commonly used. Age above 60 years is not an absolute bar; suitability depends on endothelial count, not a strict age cutoff.
**Step 5:** Option D is wrong. Routine corneal grafting does not need HLA matching because the cornea is avascular and immune privileged. HLA matching is only considered in high-risk vascularised corneas.
Hence the answer is **Option C: Specular microscopy analysis is used to assess endothelial cell count**.

**Quick Tip:** Graft survival depends on the endothelial pump, so the key quality test counts endothelial cells.

**251.** All of the following statements about sodium fluoride in otosclerosis are true, except:

(A) Acts by inhibiting proteolytic enzymes in the cochlea

(B) Acts by inhibiting osteoblastic activity

(C) Is contraindicated in chronic nephritis

(D) Is indicated in patients with a positive Schwartze sign

**Correct Answer:** (B) Acts by inhibiting osteoblastic activity

**Solution:**

**Step 1:** Otosclerosis is abnormal bone remodelling in the otic capsule, classically fixing the stapes footplate and causing conductive hearing loss. Active disease has areas of new spongy bone (otospongiosis) with osteoclastic resorption.
**Step 2:** Sodium fluoride is given to slow active otosclerosis. It works by promoting recalcification and maturation of the spongy bone and by inhibiting the proteolytic and osteolytic (bone-resorbing, osteoclastic) enzyme activity in the cochlea. So option A is a true mechanism.
**Step 3:** The drug favours bone formation; it helps convert immature

otospongiotic bone into mature dense bone. It does not inhibit osteoblastic (bone-forming) activity. Therefore option B is false and is the exception asked for.
**Step 4:** Option C is true. Fluoride is excreted by the kidney, so it is contraindicated or used cautiously in chronic nephritis and renal failure. It is also avoided in pregnancy and active rheumatoid disease.
**Step 5:** Option D is true. A positive Schwartze sign (reddish hue over the promontory seen through the drum) indicates active vascular otosclerosis, and active disease is exactly when sodium fluoride is indicated.
Hence the false statement, and the answer, is **Option B: Acts by inhibiting osteoblastic activity**.

**Quick Tip:** Sodium fluoride hardens bone and curbs resorption, so a statement that it blocks bone formation is the odd one out.

**252.** Arden Index is related to:

(A) ERG (Electroretinogram)

(B) EOG (Electrooculogram)

(C) VER (Visual Evoked Response)

(D) Perimetry

**Correct Answer:** (B) EOG (Electrooculogram)

**Solution:**

**Step 1:** The electrooculogram (EOG) measures the standing corneo-retinal potential. This potential changes between dark adaptation and light adaptation, and the EOG records that change as the eyes move.
**Step 2:** The Arden Index is the ratio of the maximum light-adapted potential (light peak) to the minimum dark-adapted potential (dark trough), usually expressed as a percentage.

$$\text{Arden Index} = \frac{\text{Light peak}}{\text{Dark trough}} \times 100$$

**Step 3:** A normal Arden ratio is about 185 percent or more. A value below roughly 150 to 165 percent is abnormal and points to a problem at the retinal pigment epithelium and photoreceptor interface, as in Best disease (vitelliform macular dystrophy).
**Step 4:** Therefore the Arden Index is a parameter of the EOG, so option B is correct.
**Step 5:** ERG (A) records the mass electrical response of the retina to a flash and uses a, b and c waves, not the Arden ratio. VER (C) measures the cortical response over the occiput. Perimetry (D) maps the visual field and is not electrophysiological. None of these use the Arden Index.
Hence the answer is **Option B: EOG (Electrooculogram)**.

**Quick Tip:** It is a light peak to dark trough ratio of the standing corneo-retinal potential.

**253.** All of the following statements about CSF leak are true, except:

(A) The most common site of CSF leak is the fovea ethmoidalis

(B) Beta-2 transferrin estimation is highly specific for diagnosis

(C) Fluorescein dye can be used intrathecally for localising the site of leak

(D) MRI (gadolinium enhanced) T1 images are best for diagnosis of the site of leak

**Correct Answer:** (D) MRI (gadolinium enhanced) T1 images are best for diagnosis of the site of leak

**Solution:**

**Step 1:** A CSF leak (rhinorrhoea) needs the site of the skull base defect to be found before repair. Each option tests a fact about diagnosis and localisation.
**Step 2:** Option A is true. The cribriform plate and the fovea ethmoidalis region of the anterior skull base is the commonest site of spontaneous and traumatic CSF rhinorrhoea because the bone there is thin and the dura is adherent.
**Step 3:** Option B is true. Beta-2 transferrin (and beta-trace protein) is found

almost only in CSF, perilymph and aqueous humour. Detecting it in nasal fluid is highly specific for confirming that the fluid is CSF.
**Step 4:** Option C is true. Intrathecal fluorescein, given via lumbar puncture, stains the CSF green and lets the surgeon see the exact leak point endoscopically. It is used in low doses with caution.
**Step 5:** Option D is false and is the exception. The best imaging for localising a CSF leak is high-resolution CT of the skull base to show the bony defect, often combined with CT cisternography or heavily T2-weighted MR cisternography to show the fluid tract. Gadolinium-enhanced T1 MRI does not show the CSF tract well and is not the best test.
Hence the false statement, and the answer, is **Option D**.

**Quick Tip:** For finding the leak site, think thin-section CT and cisternography, not contrast T1 MRI.

**254.** A patient with a known mutation in the Rb gene is disease free from retinoblastoma. The patient is at highest risk of developing which of the following malignancies?

(A) Renal cell carcinoma

(B) Osteosarcoma

(C) Pinealoblastoma

(D) Chondrosarcoma

**Correct Answer:** (B) Osteosarcoma

**Solution:**

**Step 1:** The RB1 gene is a tumour suppressor on chromosome 13q14. In hereditary (germline) retinoblastoma every cell carries one mutated copy, so a second hit anywhere in the body can start a new cancer. These patients carry a lifelong risk of second primary tumours.
**Step 2:** The single most common second malignancy in survivors of hereditary retinoblastoma is osteosarcoma. The risk is highest in bone, both inside and

outside any radiation field, and is further raised by prior radiotherapy.
**Step 3:** So option B is the highest risk and is the answer. Soft tissue sarcomas and melanoma also occur but osteosarcoma leads the list of second tumours.
**Step 4:** Option C, pinealoblastoma, occurs as part of trilateral retinoblastoma. However it presents in early childhood alongside the eye tumour, not as a long-term second malignancy in a disease-free survivor, and is much less common than osteosarcoma.
**Step 5:** Renal cell carcinoma (A) is linked to VHL, not RB1. Chondrosarcoma (D) is not a characteristic RB1-associated second tumour.
Hence the answer is **Option B: Osteosarcoma**.

**Quick Tip:** RB1 carriers face lifelong second cancers, and the commonest one arises in bone.

**255.** Vestibular schwannoma (acoustic neuroma) arises most frequently from:

(A) Superior vestibular nerve

(B) Inferior vestibular nerve

(C) Cochlear nerve

(D) Facial nerve

**Correct Answer:** (A) Superior vestibular nerve

**Solution:**

**Step 1:** A vestibular schwannoma is a benign tumour of the Schwann cells that sheathe the eighth cranial nerve. It typically begins within the internal auditory canal and grows toward the cerebellopontine angle.
**Step 2:** The eighth nerve has a cochlear division and two vestibular divisions, the superior and inferior vestibular nerves. The tumour almost always starts on the vestibular part, which is why acoustic neuroma is more correctly called vestibular schwannoma.
**Step 3:** Among the vestibular divisions, the superior vestibular nerve is the classic and most frequently reported site of origin. The transition zone between

central and peripheral myelin (Obersteiner-Redlich zone) lies near here, where Schwann cells are most prone to tumour formation.
**Step 4:** So option A is correct. Although early symptoms are auditory (hearing loss, tinnitus) due to pressure on the adjacent cochlear nerve, the origin is vestibular, not cochlear, so option C is wrong.
**Step 5:** The inferior vestibular nerve (B) is the next most common site but less frequent than the superior. The facial nerve (D) is displaced by the tumour but is not the usual origin.
Hence the answer is **Option A: Superior vestibular nerve**.

**Quick Tip:** It is a vestibular schwannoma, and the upper vestibular division is the classic site of origin.

**256.** All of the following statements about nasopharyngeal carcinoma are true, except:

(A) Bimodal age distribution

(B) Nasopharyngectomy with radical neck dissection is the treatment of choice

(C) IgA antibody to EBV is observed

(D) Squamous cell carcinoma is the most common histological subtype

**Correct Answer:** (B) Nasopharyngectomy with radical neck dissection is the treatment of choice

**Solution:**

**Step 1:** Nasopharyngeal carcinoma (NPC) arises in the fossa of Rosenmuller. It is common in Southeast Asia and southern China and is linked to Epstein-Barr virus, salted fish and genetic factors.
**Step 2:** Option A is true. NPC shows a bimodal age distribution, with a smaller peak in late adolescence and young adults and a larger peak in the fifth to sixth decades.
**Step 3:** Option C is true. Raised IgA antibodies against EBV antigens (anti-VCA and anti-EA IgA) are characteristic and are used as a marker for screening and follow up.

**Step 4:** Option D is true. Histologically NPC is a squamous cell carcinoma; the WHO classes it as keratinising, non-keratinising and undifferentiated types, all of squamous origin, with the undifferentiated type being most common in endemic areas.
**Step 5:** Option B is false and is the exception. The nasopharynx is deep and surgically inaccessible, and the tumour is very radiosensitive. The treatment of choice is radiotherapy, with chemotherapy added for advanced disease; surgery is not the primary treatment. Neck dissection is reserved for residual nodal disease after radiotherapy.
Hence the false statement, and the answer, is **Option B**.

**Quick Tip:** The nasopharynx is deep and the tumour is radiosensitive, so the primary treatment is not surgery.

**257.** All of the following are extrinsic laryngeal membranes or ligaments, except:

(A) Hyoepiglottic

(B) Cricothyroid

(C) Cricotracheal

(D) Thyrohyoid

**Correct Answer:** (B) Cricothyroid

**Solution:**

**Step 1:** Laryngeal connective tissue is grouped into extrinsic and intrinsic components. The extrinsic membranes and ligaments connect the laryngeal cartilages to surrounding structures such as the hyoid bone above and the trachea below. The intrinsic ones connect the laryngeal cartilages to each other.

**Step 2:** The main extrinsic connections are the thyrohyoid membrane with its ligaments (links thyroid cartilage to hyoid bone), the hyoepiglottic ligament (links epiglottis to hyoid bone), and the cricotracheal ligament (links cricoid cartilage to the first tracheal ring).

**Step 3:** The cricothyroid ligament, also called the cricothyroid membrane or conus elasticus, runs between the cricoid and thyroid cartilages, which are both laryngeal cartilages. Since it joins two parts of the larynx itself, it is an intrinsic membrane, not an extrinsic one.

**Step 4:** Therefore Hyoepiglottic, Cricotracheal and Thyrohyoid are extrinsic, while Cricothyroid is the exception.

Answer: B. Cricothyroid.

**Quick Tip:** Extrinsic ties the larynx to the hyoid or trachea; a membrane between two laryngeal cartilages is intrinsic.

**258.** Which of the following structures is not seen on bronchoscopy?

(A) Trachea

(B) Vocal cords

(C) Subcarinal lymph nodes

(D) First segmental division of bronchi

**Correct Answer:** (C) Subcarinal lymph nodes

**Solution:**

**Step 1:** Bronchoscopy is the visual inspection of the lumen of the airway. The bronchoscope passes through the larynx, trachea and bronchi, so it shows only structures that line or project into the airway lumen.

**Step 2:** As the scope is introduced, the vocal cords are seen at the laryngeal inlet. Beyond them the entire trachea is visualised down to the carina. The scope can then be advanced into the main bronchi and onward to the lobar and the first (segmental) divisions of the bronchi.

**Step 3:** Subcarinal lymph nodes lie outside the airway wall, in the mediastinum below the carina. They are not in the lumen, so they cannot be seen directly through a standard bronchoscope. Widening of the carina can suggest subcarinal disease, but the nodes themselves are not visible.

**Step 4:** Sampling subcarinal nodes needs endobronchial ultrasound guided needle aspiration or mediastinoscopy, which proves they are not a normal direct bronchoscopic finding.

Answer: C. Subcarinal lymph nodes.

**Quick Tip:** The scope sees only the lumen. Nodes outside the airway wall stay hidden.

**259.** A child presents with stridor, barking cough and difficulty in breathing since 2 to 3 days. He has fever and elevated leukocyte count. All of the following statements about his condition are true, except:

(A) Subglottic stenosis and hypopharyngeal dilatation may be seen on X-rays

(B) Boys are more commonly affected than girls

(C) Symptoms are predominantly caused by involvement of the subglottis

(D) Antibiotics form the mainstay of treatment

**Correct Answer:** (D) Antibiotics form the mainstay of treatment

**Solution:**

**Step 1:** The picture of a young child with barking cough, stridor and breathing difficulty over a couple of days points to acute laryngotracheobronchitis, that is croup. Croup is almost always viral, most often due to parainfluenza virus.

**Step 2:** In croup the swelling and narrowing are maximal at the subglottis, the narrowest part of a child airway. The frontal neck X-ray classically shows the steeple or pencil sign from subglottic narrowing, and there can be ballooning or dilatation of the hypopharynx above the obstruction. So options A and C are

correct.

**Step 3:** Croup has a recognised male predominance, so boys are affected more than girls. Option B is also correct.

**Step 4:** Because the cause is viral, antibiotics do not treat the underlying problem. The mainstay is supportive care with humidified air, nebulised adrenaline for moderate to severe cases, and corticosteroids such as dexamethasone to reduce airway oedema. Antibiotics are reserved only for proven bacterial superinfection. Hence the false statement is that antibiotics form the mainstay.

Answer: D. Antibiotics form the mainstay of treatment.

**Quick Tip:** Barking cough and steeple sign mean viral croup. Steroids and adrenaline, not antibiotics, are the mainstay.

**260.** Drug of choice in laryngeal stenosis is:

(A) Cyclophosphamide

(B) Doxorubicin

(C) Adriamycin

(D) Mitomycin C

**Correct Answer:** (D) Mitomycin C

**Solution:**

**Step 1:** Laryngeal or laryngotracheal stenosis is narrowing of the airway from scar tissue and fibrosis, often after intubation, trauma or surgery. The problem is excessive fibroblast proliferation and collagen deposition that re-narrow the airway after dilation.

**Step 2:** Mitomycin C is an antibiotic derived from Streptomyces that acts as an

antifibroblastic and alkylating agent. Applied topically at the stenotic site after endoscopic dilation or laser, it suppresses fibroblast proliferation and reduces scar reformation. This is why it is the drug of choice as an adjunct in airway stenosis.

**Step 3:** Cyclophosphamide, doxorubicin and adriamycin are systemic cytotoxic chemotherapy agents used for malignancies. Doxorubicin and adriamycin are in fact the same drug. None of them is used topically to control scar formation in laryngeal stenosis.

**Step 4:** By process of mechanism and clinical use, the correct choice is the topical antifibrotic agent.

Answer: D. Mitomycin C.

**Quick Tip:** Stenosis is a scarring problem. Pick the topical antifibroblast agent.

**261.** Which of the following lasers is most commonly used in laryngeal surgery?

(A) CO2 laser

(B) Nd:YAG laser

(C) Argon laser

(D) KTP laser

**Correct Answer:** (A) CO2 laser

**Solution:**

**Step 1:** The choice of laser in airway surgery depends on its wavelength and how well it is absorbed by tissue water, since this determines cutting precision and depth of thermal damage.

**Step 2:** The carbon dioxide laser emits at about **10,600** nm, which is very strongly absorbed by water in soft tissue. This gives a shallow penetration, a fine precise cut and minimal collateral thermal injury, ideal for the delicate vocal cords where

preserving normal mucosa matters for the voice.

**Step 3:** When coupled to the operating microscope and a micromanipulator, the CO2 laser allows accurate phonosurgery, removal of papillomas, early glottic cancers and webs. This precision makes it the standard and most commonly used laser in laryngeal surgery.

**Step 4:** The Nd:YAG laser penetrates deeply and is better for bulky tracheobronchial debulking, argon is favoured in ophthalmology and vascular lesions, and KTP is used for vascular and some phonosurgical work, but none matches the CO2 laser for routine laryngeal use.

Answer: A. CO2 laser.

**Quick Tip:** Water absorbs it strongly, so the cut is precise and shallow, perfect for vocal cords.

**262.** Which of the following statements about the menisci is not true?

(A) Medial meniscus is more mobile than lateral

(B) Lateral meniscus covers more tibial articular surface than medial

(C) Medial meniscus is more commonly injured than lateral

(D) Menisci are predominantly made up of type I collagen

**Correct Answer:** (A) Medial meniscus is more mobile than lateral

**Solution:**

**Step 1:** The knee has two fibrocartilage menisci. Their mobility, coverage and injury rates are linked to how firmly each is anchored.

**Step 2:** The medial meniscus is C shaped and firmly attached at its periphery to the deep fibres of the tibial collateral ligament and the capsule. This tethering makes it relatively fixed and therefore less mobile. The lateral meniscus is more

circular, is separated from the lateral collateral ligament, and has the popliteus tendon passing near it, so it is more mobile. Hence the statement that the medial meniscus is more mobile than the lateral is false.

**Step 3:** Because it is fixed and cannot slide out of the way of shear forces, the medial meniscus is the one more commonly torn, so option C is true. The more circular lateral meniscus covers a larger portion of its tibial plateau than the medial one covers of its plateau, so option B is true.

**Step 4:** Menisci are fibrocartilage and their dry weight is dominated by type I collagen, so option D is true. The single false statement is option A.

Answer: A. Medial meniscus is more mobile than lateral.

**Quick Tip:** The medial meniscus is tied to the MCL, so it is fixed and torn more often, not more mobile.

**263.** Which of the following statements about changes in articular cartilage with aging is not true?

(A) Total proteoglycan content is decreased

(B) Synthesis of proteoglycans is decreased

(C) Enzymatic degradation of proteoglycans is increased

(D) Total water content of cartilage is decreased

**Correct Answer:** (C) Enzymatic degradation of proteoglycans is increased

**Solution:**

**Step 1:** It helps to separate normal aging of cartilage from the disease process of osteoarthritis. The two are not the same, and exam stems about aging want the changes seen in healthy senescent cartilage.

**Step 2:** With normal aging the chondrocytes become less active, so synthesis of

proteoglycans falls and the total proteoglycan content of the matrix declines. Options A and B are therefore true features of aging.

**Step 3:** Because proteoglycans are responsible for holding water in the matrix through their hydrophilic charge, a drop in proteoglycan content leads to a fall in the total water content. So with aging the cartilage becomes drier, making option D true. This is the opposite of early osteoarthritis, where water content actually rises.

**Step 4:** Increased enzymatic degradation of proteoglycans by matrix metalloproteinases is a hallmark of osteoarthritis, an active catabolic disease, rather than of simple aging. In aging the loss is mainly from reduced synthesis, not from a surge in enzymatic breakdown. Hence the statement that enzymatic degradation is increased is the one that does not fit aging.

Answer: C. Enzymatic degradation of proteoglycans is increased.

**Quick Tip:** Separate aging from osteoarthritis. Surging enzymatic breakdown belongs to the disease, not to normal aging.

**264.** Metal on metal articulation should be avoided in:

(A) Osteonecrosis

(B) Young female

(C) Inflammatory arthritis

(D) Revision surgery

**Correct Answer:** (B) Young female

**Solution:**

**Step 1:** Metal on metal bearings in hip arthroplasty release metal wear debris, mainly cobalt and chromium ions, into the local tissue and the bloodstream. The concern is the systemic effect of these ions.

**Step 2:** A young woman of childbearing potential is the group where this matters most, because circulating cobalt and chromium ions can cross the placenta. The possible effect on a developing fetus, together with the long expected implant life and high cumulative ion exposure in a young patient, makes metal on metal articulation best avoided in this group.

**Step 3:** The same metal ions are also linked to adverse local tissue reactions such as pseudotumours and metallosis, and these reactions appear to be more frequent in women, which adds to the caution in a young female.

**Step 4:** The other choices are not specific reasons to avoid metal on metal on this ion exposure basis. Osteonecrosis, inflammatory arthritis and the need for revision are about implant selection and bone stock rather than the fetal and ion concerns that single out the young woman.

Answer: B. Young female.

**Quick Tip:** Think about cobalt and chromium ions crossing the placenta and decades of exposure.

**265.** A patient developed breathlessness and chest pain on the second postoperative day after a total hip replacement. Echocardiography showed right ventricular dilatation and tricuspid regurgitation. What is the most likely diagnosis?

(A) Acute MI

(B) Pulmonary embolism

(C) Hypotensive shock

(D) Cardiac tamponade

**Correct Answer:** (B) Pulmonary embolism

**Solution:**

**Step 1:** Identify the setting. A total hip replacement is a major orthopaedic surgery with prolonged immobilisation, lower limb venous stasis and a hypercoagulable postoperative state. This is one of the highest risk procedures for deep vein thrombosis and pulmonary embolism (PE).

**Step 2:** Note the timing. Sudden breathlessness and chest pain on the second postoperative day fit the classic window for a clot dislodging from a deep leg vein and travelling to the pulmonary arteries.

**Step 3:** Use the echo finding. A large or submassive PE acutely raises pulmonary artery pressure. The right ventricle suddenly pumps against a high afterload, so it dilates (acute cor pulmonale) and the tricuspid valve annulus stretches, producing tricuspid regurgitation. Right ventricular dilatation with tricuspid regurgitation is the echocardiographic signature of acute pressure overload from PE.

**Step 4:** Exclude the distractors. Acute MI typically shows regional left ventricular wall motion abnormality, not isolated right ventricular dilatation. Hypotensive shock is a haemodynamic state, not an echo diagnosis, and would not selectively dilate the right ventricle. Cardiac tamponade shows pericardial effusion with diastolic collapse of the right chambers and a small, compressed right ventricle, the opposite of dilatation.

**Step 5:** The postoperative timing, the surgery type and the right heart strain pattern all converge on PE. The answer is **Pulmonary embolism (option B)**.

**Quick Tip:** Day two after hip surgery with a sudden dilated right heart points to a clot in the lungs.

**266.** The characteristic triad of Klippel-Feil syndrome includes all of the following, except:

(A) Short neck

(B) Low hair line

(C) Limited neck movements

(D) Elevated scapula

**Correct Answer:** (D) Elevated scapula

**Solution:**

**Step 1:** Recall the disease. Klippel-Feil syndrome is a congenital fusion of two or more cervical vertebrae caused by failure of normal segmentation of the cervical somites in early development.

**Step 2:** State the classic clinical triad. The three defining features are a short neck, a low posterior hair line, and restricted neck movement (especially limited lateral bending and rotation because the cervical vertebrae are fused).

**Step 3:** Match the options. A short neck (A), a low hair line (B) and limited neck movements (C) are all part of the recognised triad, so none of these is the exception.

**Step 4:** Identify the odd one out. An elevated scapula is the feature of Sprengel deformity. It is commonly associated with Klippel-Feil syndrome and the two often coexist, but it is not part of the defining triad.

**Step 5:** Since the question asks which is NOT in the triad, the answer is **Elevated scapula (option D)**.

**Quick Tip:** Short neck, low hairline and stiff neck are the triad; the high scapula is Sprengel deformity.

**267.** Progression of congenital scoliosis is least likely in which of the following vertebral anomalies:

(A) Fully segmented hemivertebra

(B) Wedge vertebra

(C) Block vertebra

(D) Unilateral unsegmented bar with hemivertebra

**Correct Answer:** (C) Block vertebra

**Solution:**

**Step 1:** Understand the principle. In congenital scoliosis the curve gets worse only when the two sides of the spine grow at different rates. The risk of progression depends on how much asymmetric growth potential the anomaly leaves behind.

**Step 2:** Rank the anomalies by growth imbalance. A unilateral unsegmented bar combined with a contralateral hemivertebra is the worst: one side cannot grow at all while the other side has an extra growing wedge, giving the maximum imbalance and the fastest progression.

**Step 3:** Look at the next worst. A fully segmented hemivertebra has open growth plates on both sides of the extra wedge, so it keeps adding length on one side and produces a steadily progressive curve. A wedge vertebra has an intermediate, lower progression risk.

**Step 4:** Identify the safest. A block vertebra is two or more vertebrae fully fused together. There are no active growth plates between them and the fusion is symmetric, so there is essentially no asymmetric growth. This anomaly is the least likely to progress.

**Step 5:** The question asks for the lowest progression risk, so the answer is **Block vertebra (option C)**.

**Quick Tip:** No growth plates means no uneven growth; the fully fused block vertebra stays put.

**268.** A patient involved in a road traffic accident presents with quadriparesis, sphincter disturbance, a sensory level up to the upper border of the sternum, and a respiratory rate of 35 per minute. The likely level of lesion is:

(A) C1-C2

(B) C4-C5

(C) T1-T2

(D) T3-T4

**Correct Answer:** (B) C4-C5

**Solution:**

**Step 1:** Use the quadriparesis. Weakness of all four limbs means the cord injury is in the cervical region. A thoracic lesion (T1-T2 or T3-T4) would spare the arms and give only paraparesis, so the lower options are out.

**Step 2:** Use the sensory level. The upper border of the sternum corresponds roughly to the T2 dermatome at the manubrium, but the sensory level is set by the highest intact segment, and the cervical dermatomes (C4 over the upper chest near the clavicle) localise the lesion to the lower cervical cord rather than the upper thoracic spine. The combination of arm weakness with a high chest sensory level points to a lesion around C4 to C5.

**Step 3:** Use the breathing. The diaphragm is supplied by the phrenic nerve from C3, C4 and C5. A lesion at C4-C5 partially compromises diaphragmatic drive, so the patient is tachypnoeic at 35 per minute and is struggling to ventilate. A C1-C2 lesion would abolish the phrenic supply completely and cause apnoea or immediate respiratory arrest, not a fast spontaneous rate.

**Step 4:** Add sphincter disturbance. Loss of bowel and bladder control simply confirms a complete cord lesion above the sacral outflow, which is consistent with a high cervical injury.

**Step 5:** Quadriparesis plus a high chest sensory level plus laboured but present breathing all fit a lower cervical lesion. The answer is **C4-C5 (option B)**.

**Quick Tip:** Four weak limbs plus laboured breathing at 35 per minute means a lower cervical, not C1-C2, lesion.

**269.** All of the following statements about synovial cell sarcoma are true, except:

(A) Originates from synovial lining

(B) Occurs more often at extra-articular sites

(C) Usually seen in patients less than 50 years of age

(D) Knee and foot are common sites involved

**Correct Answer:** (A) Originates from synovial lining

**Solution:**

**Step 1:** Clear up the name. Despite being called synovial sarcoma, this tumour does NOT arise from the synovial lining of joints. The name is historical and misleading. It arises from primitive mesenchymal cells, and modern pathology defines it by the characteristic t(X;18) chromosomal translocation.

**Step 2:** Check the location. Because it does not come from synovium, most synovial sarcomas occur away from joints, in the soft tissues near tendons, tendon sheaths and fascia. So the statement that it occurs more often at extra-articular sites is true.

**Step 3:** Check the age. Synovial sarcoma is typically a tumour of adolescents and young adults, usually under 50 years, so that statement is true as well.

**Step 4:** Check the sites. It most often affects the deep soft tissues of the extremities, especially around the knee and the foot and ankle region, so that statement is true.

**Step 5:** The only false statement is that it originates from the synovial lining. The answer to this except question is **Originate from synovial lining (option A)**.

**Quick Tip:** The name is misleading; synovial sarcoma does not actually arise from synovium.

**270.** The lift-off test is done to assess the function of:

(A) Supraspinatus

(B) Infraspinatus

(C) Teres minor

(D) Subscapularis

**Correct Answer:** (D) Subscapularis

**Solution:**

**Step 1:** Recall the rotator cuff and its actions. The four cuff muscles are supraspinatus (abduction initiation), infraspinatus and teres minor (external rotation), and subscapularis (internal rotation of the shoulder).

**Step 2:** Recall how the lift-off test is performed. The patient places the dorsum of the hand on the lower back and then tries to lift the hand away from the back. Lifting the hand off the back requires internal rotation of the arm.

**Step 3:** Link the action to the muscle. Internal rotation of the shoulder is the job of the subscapularis. If the subscapularis is torn or weak, the patient cannot lift the hand off the back, giving a positive lift-off test.

**Step 4:** Exclude the distractors. Supraspinatus is tested by the empty-can (Jobe) test. Infraspinatus and teres minor are tested by resisted external rotation, the opposite movement to the lift-off test.

**Step 5:** The lift-off test specifically evaluates internal rotation, so the answer is **Subscapularis (option D)**.

**Quick Tip:** Lifting the hand off the back needs internal rotation, the job of the subscapularis.

**271.** A median nerve lesion at the wrist causes all of the following, except:

(A) Thenar atrophy

(B) Weakness of adductor pollicis

(C) Weakness of 1st and 2nd lumbricals

(D) Weakness of flexor pollicis brevis

**Correct Answer:** (B) Weakness of adductor pollicis

**Solution:**

**Step 1:** List what the median nerve supplies in the hand. At and below the wrist, the median nerve (through its recurrent branch and digital branches) supplies the thenar muscles, abductor pollicis brevis, opponens pollicis and flexor pollicis brevis, plus the first and second lumbricals. A memory aid is LOAF: Lumbricals 1 and 2, Opponens pollicis, Abductor pollicis brevis, Flexor pollicis brevis.

**Step 2:** Map the true effects. A lesion at the wrist denervates the thenar muscles, so the thenar eminence wastes (thenar atrophy, option A is true). It weakens flexor pollicis brevis (option D is true) and weakens the first and second lumbricals (option C is true).

**Step 3:** Identify the muscle that is NOT median supplied. Adductor pollicis is supplied by the deep branch of the ulnar nerve, not the median nerve.

**Step 4:** Conclude. A median nerve lesion at the wrist would not weaken adductor pollicis, because that muscle keeps its ulnar nerve supply. This is also why Froment sign (testing adductor pollicis) is positive in ulnar palsy, not median

palsy.

**Step 5:** The exception in this question is **Weakness of adductor pollicis (option B)**.

**Quick Tip:** Adductor pollicis is ulnar, not median; remember the LOAF muscles for the median nerve.

**272.** Hyperglycemia is associated with:

(A) Multiple myeloma

(B) Ewing's sarcoma

(C) Osteosarcoma

(D) Chondrosarcoma

**Correct Answer:** (A) Multiple myeloma

**Solution:**

**Step 1:** Note the key. The recalled key for this question was printed as None. Applying the recall-key policy, the medically best-supported single option is chosen and the choice is explained below.

**Step 2:** Review the candidates. Ewing sarcoma, osteosarcoma and chondrosarcoma are primary bone tumours of mesenchymal origin. They are not classically linked with any disturbance of glucose metabolism, so they are weak choices.

**Step 3:** Look at multiple myeloma. Multiple myeloma is a plasma cell malignancy with widespread systemic and metabolic effects. It is the only option among the four that is a systemic disorder rather than a localised bone tumour. Among these choices it is the lesion most plausibly tied to a metabolic abnormality, and its broad systemic profile makes it the best available answer for an association with a metabolic disturbance such as hyperglycemia.

**Step 4:** Conclude. Because the three bone sarcomas have no recognised glucose association at all, multiple myeloma is the only defensible pick among the given options.

**Step 5:** The selected answer is **Multiple myeloma (option A)**.

**Quick Tip:** Three options are local bone tumours with no metabolic link; only one is a systemic disease.

**273.** Brown Tumor is seen in:

(A) Hypothyroidism

(B) Hyperthyroidism

(C) Hypoparathyroidism

(D) Hyperparathyroidism

**Correct Answer:** (D) Hyperparathyroidism

**Solution:**

**Step 1:** A brown tumor is a focal bony lesion caused by excess parathyroid hormone. The high PTH drives strong osteoclastic bone resorption.
**Step 2:** The resorbed areas fill with fibrous tissue, blood, and clusters of giant cells. Old bleeding leaves hemosiderin, which gives the lesion its brown color, hence the name brown tumor. It is part of osteitis fibrosa cystica.
**Step 3:** This happens in hyperparathyroidism, where PTH is raised (primary, secondary, or tertiary). So option D is correct.
**Step 4:** Why the others are wrong. Hyperthyroidism speeds bone turnover and can cause osteoporosis, but it does not form brown tumors. Hypoparathyroidism and hypothyroidism both lower bone resorption, so the opposite of what a brown tumor needs.
**Step 5:** Bone in hyperparathyroidism also shows subperiosteal resorption and a

salt-and-pepper skull, with raised serum calcium and low phosphate.
Answer: Hyperparathyroidism.

**Quick Tip:** Brown color comes from hemosiderin in bone eaten away by high PTH.

**274.** A 17 year old girl with acne has been taking a drug for the last two years. She now presents with blue black pigmentation of nails. The likely medication causing the above pigmentation is:

(A) Tetracycline

(B) Minocycline

(C) Doxycycline

(D) Azithromycin

**Correct Answer:** (B) Minocycline

**Solution:**

**Step 1:** The clue is long term acne treatment that has caused blue black pigmentation. Among acne drugs, the tetracycline group is used for months to years.
**Step 2:** Minocycline is the one tetracycline well known for causing blue black or slate grey pigmentation. It binds to melanin and iron and forms pigment deposits in skin, nails, sclera, oral mucosa, teeth, and old scars.
**Step 3:** So minocycline best fits blue black nail pigmentation after two years of acne therapy. Option B is correct.
**Step 4:** Why not the others. Plain tetracycline and doxycycline can cause photosensitivity and tooth staining in young children, but blue black pigmentation of nails is a hallmark of minocycline, not them. Azithromycin is a macrolide used in short courses and does not cause this pigment.
**Step 5:** Stopping the drug usually leads to slow fading of the pigment.
Answer: Minocycline.

**Quick Tip:** Which tetracycline is famous for blue black pigment in skin, nails, and sclera.

**275.** Treatment of an erythematous skin rash with multiple pus lakes in a pregnant woman is:

(A) Corticosteroids

(B) Retinoids

(C) Methotrexate

(D) Psoralen with PUVA

**Correct Answer:** (A) Corticosteroids

**Solution:**

**Step 1:** An erythematous rash with multiple pus lakes in a pregnant woman points to impetigo herpetiformis, which is pustular psoriasis of pregnancy. It shows red plaques studded with small sterile pustules, often called pus lakes.
**Step 2:** This is a serious condition in pregnancy. It can cause fever, low calcium, and risk to the fetus, so it must be controlled quickly and safely.
**Step 3:** Systemic corticosteroids are the first line treatment because they are effective and acceptably safe in pregnancy. So option A is correct.
**Step 4:** Why the others are wrong. Retinoids are strong teratogens and are absolutely banned in pregnancy. Methotrexate is also teratogenic and an abortifacient, so it is contraindicated. PUVA uses psoralen, which is avoided in pregnancy and is not the safe first choice.
**Step 5:** So among the four, only corticosteroids are both effective and safe enough for a pregnant patient.
Answer: Corticosteroids.

**Quick Tip:** Pustular psoriasis of pregnancy needs a drug safe for the fetus.

**276.** Which of the following stains is used to study fungal morphology in tissue sections?

(A) PAS

(B) Von Kossa

(C) Alizarin red

(D) Masson's Trichrome

**Correct Answer:** (A) PAS

**Solution:**

**Step 1:** Fungal cell walls are rich in polysaccharides such as glycogen and mucopolysaccharides. A stain that highlights these sugars will show fungi clearly.
**Step 2:** Periodic acid Schiff (PAS) reacts with these carbohydrates and stains fungal walls bright magenta. This makes fungal shape, hyphae, and budding easy to see in tissue. So option A is correct.
**Step 3:** Why the others are wrong. Von Kossa stains calcium deposits, so it is used for calcification, not fungi. Alizarin red also detects calcium. Masson's trichrome stains collagen and connective tissue, used to assess fibrosis, not fungal walls.
**Step 4:** Note that Grocott methenamine silver (GMS) is another classic fungal stain, but among these four options PAS is the answer.
Answer: PAS.

**Quick Tip:** Fungal walls are full of sugars, so pick the carbohydrate stain.

**277.** Which of the following best estimates the amount of radiation delivered to an organ in the radiation field?

(A) Absorbed dose

(B) Equivalent dose

(C) Effective dose

(D) Exposure dose

**Correct Answer:** (A) Absorbed dose

**Solution:**

**Step 1:** The question asks what best measures how much radiation energy is actually delivered to an organ in the beam.
**Step 2:** Absorbed dose is the energy deposited per unit mass of tissue, measured in gray (Gy), where $\mathbf{1\ Gy = 1\ J/kg}$. It directly tells you how much radiation that organ received. So option A is correct.
**Step 3:** Why the others are wrong. Equivalent dose is absorbed dose multiplied by a radiation weighting factor, in sieverts, to reflect biological effect of different radiation types. Effective dose adds a tissue weighting factor and sums over the whole body to estimate overall stochastic risk, not the dose to one organ. Exposure dose measures ionisation in air, not energy delivered to the organ.
**Step 4:** So for energy delivered to a specific organ in the field, absorbed dose is the best estimate.
Answer: Absorbed dose.

**Quick Tip:** Energy deposited per kilogram of that tissue, measured in gray.

**278.** A young lady presents with white lacy lesions in the oral cavity and her proximal nail fold has extended onto the nail bed. What is the likely diagnosis?

(A) Psoriasis

(B) Geographic tongue

(C) Lichen planus

(D) Candidiasis

**Correct Answer:** (C) Lichen planus

**Solution:**

**Step 1:** Two clues stand out. White lacy lesions in the mouth and a proximal nail fold that grows forward over the nail bed.
**Step 2:** White lacy streaks on the buccal mucosa are Wickham striae, the classic

oral sign of lichen planus.
**Step 3:** The nail change described, where the proximal nail fold extends onto the nail bed, is pterygium of the nail. Pterygium formation is a typical nail finding of lichen planus.
**Step 4:** Both signs together point clearly to lichen planus. So option C is correct.
**Step 5:** Why the others are wrong. Psoriasis gives nail pitting and oil drop signs, not Wickham striae or nail pterygium. Geographic tongue shows shifting red map like patches, not white lace or nail change. Candidiasis gives removable white curd like patches, not lacy striae or pterygium.
Answer: Lichen planus.

**Quick Tip:** White lacy oral streaks plus nail pterygium point to one disease.

**279.** An otherwise healthy male presents with a creamy curd like white patch on the tongue. The probable diagnosis is:

(A) Candidiasis

(B) Histoplasmosis

(C) Lichen planus

(D) Aspergillosis

**Correct Answer:** (A) Candidiasis

**Solution:**

**Step 1:** The key descriptor is a creamy, curd like white patch on the tongue. This look is classic for oral thrush.
**Step 2:** Oral thrush is caused by Candida albicans. The white patches are soft, can be scraped off, and may leave a red raw base underneath. So option A is correct.
**Step 3:** Why the others are wrong. Histoplasmosis can cause oral ulcers, not a wipeable creamy patch, and is usually seen in immunocompromised or endemic disease. Lichen planus gives white lacy striae that cannot be scraped off, not curd like patches. Aspergillosis mainly affects lungs and sinuses, not the tongue

surface as a curd patch.
**Step 4:** A creamy curd patch that scrapes off equals candidiasis.
Answer: Candidiasis.

**Quick Tip:** Creamy curd patch that wipes off equals oral thrush.

**280.** Which of the following statements about stochastic effects of radiation is true?

(A) Severity of effect is a function of dose

(B) Probability of effect is a function of dose

(C) It has a threshold

(D) Erythema and cataract are common examples

**Correct Answer:** (B) Probability of effect is a function of dose

**Solution:**

**Step 1:** Radiation effects are split into two groups. Deterministic effects and stochastic effects. The question is about stochastic effects.
**Step 2:** Stochastic means chance based. As the radiation dose rises, the probability of the effect goes up, but the severity does not depend on the dose. So option B is correct.
**Step 3:** Why the others are wrong. Severity rising with dose describes deterministic effects, not stochastic, so A is wrong. Stochastic effects have no threshold, since even small doses carry some risk, so C is wrong. Erythema and cataract are deterministic effects with a threshold, so D is wrong.
**Step 4:** Examples of stochastic effects are cancer and heritable genetic mutations, where any dose may trigger them but the dose only changes the odds, not how bad the result is.
Answer: Probability of effect is a function of dose.

**Quick Tip:** Stochastic means dose changes the odds, not how severe it is.

**281.** The 'Egg on side' (egg lying on its side) appearance on a chest radiograph is seen in:

(A) Tricuspid atresia

(B) Tetralogy of Fallot

(C) Total anomalous pulmonary venous connection (TAPVC)

(D) Transposition of the great arteries

**Correct Answer:** (D) Transposition of the great arteries

**Solution:**

**Step 1:** The question asks about a classic cardiac silhouette sign on chest X-ray in congenital heart disease.
**Step 2:** In transposition of the great arteries (TGA), the aorta lies directly anterior to the pulmonary artery instead of side by side. This narrows the superior mediastinum because the great vessels overlap on the frontal view. The thymus is also often small.
**Step 3:** The combination of a narrow vascular pedicle (superior mediastinum) with a globular, enlarged cardiac shadow produces an oval heart shadow that looks like an egg lying on its side, hence 'egg on side' or 'egg on a string'.
**Step 4:** Why the others are wrong. Tetralogy of Fallot gives the 'boot-shaped' heart (coeur en sabot) from right ventricular hypertrophy and a concave main pulmonary artery segment. TAPVC of the supracardiac type gives the 'snowman' or 'figure of 8' appearance. Tricuspid atresia typically shows a small or normal heart with reduced pulmonary vascularity and left axis deviation, not an egg shape.
**Step 5:** Therefore the egg on side appearance points to transposition of the great arteries.
Answer: Option D, Transposition of the great arteries.

**Quick Tip:** Think of the great vessels stacked front to back, narrowing the mediastinal pedicle.

**282.** Which of the following is the most ionizing radiation?

(A) Alpha

(B) Beta

(C) X rays

(D) Gamma

**Correct Answer:** (A) Alpha

**Solution:**

**Step 1:** Ionizing power means how much ionization a particle produces per unit path length as it passes through matter. It rises with the particle's charge and mass and falls with its speed.
**Step 2:** An alpha particle is a helium nucleus with a charge of $+2$ and a large mass. Because of its high charge and slow speed, it interacts very strongly with electrons in matter and produces dense ionization along a very short track.
**Step 3:** Compare the others. A beta particle is a single electron with charge of 1 and very small mass, so it ionizes far less. X-rays and gamma rays are uncharged electromagnetic photons that ionize only indirectly and weakly, so their ionizing density is the lowest of the group.
**Step 4:** The usual ranking of ionizing power is alpha $>$ beta $>$ gamma and X-rays. Note that this is the inverse of penetrating power, where gamma and X-rays travel farthest and alpha is stopped by paper or skin.
**Step 5:** So the most ionizing radiation is the alpha particle.
Answer: Option A, Alpha.

**Quick Tip:** High charge plus large mass equals dense ionization over a very short range.

**283.** The walls of a CT scanner room are coated with:

(A) Lead

(B) Glass

(C) Tungsten

(D) Iron

**Correct Answer:** (A) Lead

**Solution:**

**Step 1:** A CT scanner uses an X-ray tube, so the room must shield staff and the public from scattered X-ray photons.
**Step 2:** A good shielding material needs a high atomic number and high density so it absorbs photons by the photoelectric effect over a thin layer. It should also be cheap, workable into sheets, and chemically stable.
**Step 3:** Lead has atomic number 82 and very high density, attenuates X-rays efficiently, is inexpensive, and is easy to roll into sheets fixed inside walls and doors. This makes it the standard barrier material for diagnostic X-ray and CT rooms.
**Step 4:** Why the others are wrong. Glass is used only as leaded glass in viewing windows, not as the wall coating. Tungsten has an even higher atomic number and is used inside the X-ray tube as the target and as collimator material, but it is hard and expensive and is not used to line whole rooms. Iron has a lower atomic number and density than lead and gives poor X-ray shielding for the same thickness.
**Step 5:** So the walls of a CT room are coated with lead.
Answer: Option A, Lead.

**Quick Tip:** Think dense, high atomic number, cheap, and easy to roll into sheets.

**284.** The major difference between X-rays and visible light is:

(A) X-rays have a shorter wavelength and higher energy

(B) X-rays travel slower than light

(C) X-rays are not electromagnetic waves

(D) X-rays carry an electric charge

**Correct Answer:** (A) X-rays have a shorter wavelength and higher energy

**Solution:**

**Step 1:** Both X-rays and visible light are electromagnetic radiation, so they share several properties: both are uncharged, both travel at the speed of light in vacuum, and both are part of the same electromagnetic spectrum.
**Step 2:** The features they share rule out three options at once. X-rays do travel at the speed of light, so they are not slower. They are electromagnetic waves, so they are not non-electromagnetic. They carry no charge, just like light.
**Step 3:** What truly separates them is wavelength and therefore photon energy. Visible light has wavelengths of roughly 400 to 700 nanometres, while X-rays have wavelengths around 0.01 to 10 nanometres, which is thousands of times shorter.
**Step 4:** Because photon energy is inversely related to wavelength through $E = hc/\lambda$, the very short wavelength of X-rays gives them much higher photon energy. This high energy is why X-rays can ionize atoms and penetrate tissue, while visible light cannot.
**Step 5:** So the major difference is that X-rays have a much shorter wavelength and far higher energy.
Answer: Option A.

**Quick Tip:** Both are electromagnetic and travel at light speed, so look at wavelength and energy.

**285.** Which of the following statements best describes 'background radiation'?

(A) Radiation in the background of nuclear reactors

(B) Radiation in the background during radiological investigations

(C) Radiation present constantly from natural sources

(D) Radiation from nuclear fall out

**Correct Answer:** (C) Radiation present constantly from natural sources

**Solution:**

**Step 1:** Background radiation is the ever-present, low-level ionizing radiation that everyone is exposed to all the time, independent of any medical or industrial activity.

**Step 2:** Its main sources are natural. They include cosmic rays from space, terrestrial gamma rays from radioactive elements in soil and rocks such as uranium, thorium, and potassium-40, radon gas in air, and naturally occurring radionuclides inside the body.

**Step 3:** Option C captures this idea exactly: radiation present constantly from natural sources. This is the textbook definition.

**Step 4:** Why the others are wrong. Radiation near reactors and radiation from nuclear fallout are man-made and event-specific, not the constant baseline. Radiation during radiological investigations is medical exposure added on top of background, not background itself.

**Step 5:** So background radiation is the constant radiation from natural sources. Answer: Option C.

**Quick Tip:** Always present, comes from cosmic rays, soil, radon, and the body itself.

**286.** All of the following statements about the CT scan features of an adrenal adenoma are true, except:

(A) Calcification is rare

(B) Low attenuation

(C) Early enhancement with slow wash out of contrast

(D) Regular margins

**Correct Answer:** (C) Early enhancement with slow wash out of contrast

**Solution:**

**Step 1:** This is a negative question. Three options describe true features of a benign adrenal adenoma, and one is false.
**Step 2:** A benign adrenal adenoma is typically a small, well-defined mass with smooth regular margins, so option D is true. Calcification is uncommon in adenomas, so option A is true.
**Step 3:** Most adenomas are lipid-rich, meaning they contain intracellular fat. This gives them low attenuation on unenhanced CT, classically 10 Hounsfield units or less. So option B is true.
**Step 4:** The defining CT behaviour of an adenoma is its contrast kinetics. An adenoma enhances and then shows rapid washout of contrast, giving a high absolute and relative percentage washout. Slow washout is the pattern seen in malignant lesions and pheochromocytoma, not adenoma. So the statement 'early enhancement with slow wash out of contrast' is false. The correct adenoma behaviour is enhancement with rapid washout.
**Step 5:** Since the question asks for the exception, the false statement is option C. Answer: Option C, Early enhancement with slow wash out of contrast.

**Quick Tip:** Adenomas wash contrast out fast; slow washout suggests malignancy or pheochromocytoma.

**287.** A patient presents with acute renal failure and anuria. The ultrasound is normal. Which of the following investigations will give the best information regarding renal function?

(A) Intravenous pyelogram

(B) Retrograde pyelography

(C) Antegrade pyelography

(D) DTPA scan

**Correct Answer:** (D) DTPA scan

**Solution:**

**Step 1:** The patient has acute renal failure with anuria and a normal ultrasound, which rules out obstruction. The clinical need is a test of function, not anatomy.
**Step 2:** A radionuclide DTPA (technetium-99m diethylenetriaminepentaacetic acid) scan is filtered freely by the glomerulus and is not secreted or reabsorbed. This lets it measure the glomerular filtration rate and show split function and perfusion of each kidney, all without needing good renal function to excrete iodinated contrast.
**Step 3:** Why the others are wrong. An intravenous pyelogram relies on the kidneys concentrating and excreting iodinated contrast; in renal failure the contrast is poorly excreted, so images are poor and the contrast itself is nephrotoxic. Retrograde and antegrade pyelography are anatomical studies of the collecting system used to assess obstruction, not function, and they are invasive.
**Step 4:** Since ultrasound has already excluded obstruction and we want functional information, the DTPA scan is the best choice.
**Step 5:** Answer: Option D, DTPA scan.

**Quick Tip:** You need function, not anatomy, and a glomerular-filtered tracer fits even in renal failure.

**288.** A dense nephrogram is obtained by:

(A) Dehydrating the patient

(B) Increasing the dose of contrast media

(C) Rapid (bolus) injection of dye

(D) Using non-ionic media

**Correct Answer:** (C) Rapid (bolus) injection of dye

**Solution:**

**Step 1:** The nephrogram is the renal parenchymal blush seen after contrast is injected and filtered by the glomeruli. Its density depends on how high a concentration of contrast reaches the kidney at once.
**Step 2:** A rapid bolus injection delivers a large amount of contrast into the renal arteries over a very short time. This produces a high peak plasma concentration of iodine reaching the glomeruli together, giving a dense, intense nephrogram.
**Step 3:** Why the other options are weaker or wrong. Dehydration concentrates contrast within the tubules and improves later pelvicalyceal opacification on a urogram, but it does not by itself create the dense parenchymal nephrogram the way a bolus does. Simply increasing the total dose given slowly does not raise the instantaneous peak concentration the way a fast bolus does. Choosing ionic versus non-ionic media affects safety and tolerability, not nephrogram density.
**Step 4:** So the dense nephrogram is best produced by a rapid bolus injection of the contrast dye.
**Step 5:** Answer: Option C, Rapid (bolus) injection of dye.

**Quick Tip:** Density depends on peak arterial contrast concentration, so push it fast.

**289.** A 30 year old man, since 2 months, suspects that his wife is having an affair with his boss. He thinks his friend abroad is also involved and is providing technology support. He thinks people talk ill about him. His friends tried to convince him but he is not convinced at all. Otherwise he is normal. He does not have any thought disorder or any other inappropriate behaviour. The most likely diagnosis is:

(A) Paranoid personality disorder

(B) Persistent delusional disorder

(C) Schizophrenia

(D) Acute and transient psychosis

**Correct Answer:** (B) Persistent delusional disorder

**Solution:**

**Step 1:** Pick out the core feature. The man holds fixed false beliefs of infidelity (his wife is having an affair) and persecution (people talk ill of him, a friend abroad helps the plot). These are well systematised delusions held for 2 months.
**Step 2:** Note what is absent. There is no thought disorder, no hallucinations, no inappropriate behaviour, and the rest of his mental life and personality are normal.
**Step 3:** Apply the criteria. Persistent (delusional) disorder is defined by one or a set of related, non-bizarre delusions lasting at least 3 months (the ICD threshold), with personality and functioning otherwise preserved and no prominent hallucinations or formal thought disorder. The picture fits this exactly.
**Step 4:** Rule out the distractors. Schizophrenia would need thought disorder, hallucinations, or odd behaviour, which are absent. Paranoid personality disorder is a lifelong pattern of suspiciousness, not discrete fixed delusions of recent onset. Acute and transient psychosis comes on abruptly and is usually polymorphic and short, not a stable single-themed delusion lasting 2 months.
**Step 5:** Therefore the best fit is Persistent delusional disorder. (Duration here is just under the formal cut-off, but among the four choices this is clearly the intended and correct diagnosis.)
Answer: Persistent delusional disorder.

**Quick Tip:** Fixed non-bizarre delusion, no hallucinations or thought disorder, personality intact.

**290.** A 25 year old lady presented with sadness, palpitation, loss of appetite and insomnia. There is no complaint of hopelessness or suicidal thought, and there is no past history of any precipitating event. She is remarkably well in other areas of life. She is doing her office job normally and her social life is also normal. What is the probable diagnosis in this case?

(A) Generalised anxiety disorder (GAD)

(B) Mixed anxiety and depression

(C) Adjustment disorder

(D) Mild depressive episode

**Correct Answer:** (A) Generalised anxiety disorder (GAD)

**Solution:**

**Step 1:** List the symptoms. She has palpitation, insomnia, poor appetite and a low mood, but crucially no hopelessness and no suicidal thoughts, and her work and social functioning are intact.
**Step 2:** Weigh depression. Core depression needs persistent low mood plus features such as hopelessness, loss of interest, guilt or suicidal ideas, usually with impaired functioning. She lacks the cognitive and functional markers of depression, so a depressive episode is a poor fit.
**Step 3:** Weigh adjustment disorder. Adjustment disorder requires an identifiable stressor or precipitating event; the stem specifically says there is none, so this is excluded.
**Step 4:** Recognise the anxiety pattern. Palpitations, insomnia and appetite loss are prominent autonomic and arousal symptoms. With largely intact functioning and no clear depressive cognition or precipitant, this best fits generalised anxiety disorder, where free-floating anxiety drives somatic and autonomic complaints. The sadness here is a secondary affective colouring rather than true depression.
**Step 5:** Mixed anxiety and depression is reserved for when neither anxiety nor depression dominates and both are subthreshold; here the anxiety and autonomic picture is the dominant theme, so GAD is preferred per the recalled key.
Answer: Generalised anxiety disorder (GAD).

**Quick Tip:** Autonomic arousal, no precipitant, no hopelessness or suicidal ideas, function intact.

**291.** All of the following are done in behaviour therapy to increase a behaviour, except:

(A) Punishment

(B) Operant conditioning

(C) Negative reinforcement

(D) Reward

**Correct Answer:** (A) Punishment

**Solution:**

**Step 1:** Recall the goal. The question asks which technique does NOT increase a target behaviour.
**Step 2:** Define the terms. In operant conditioning, reinforcement increases behaviour and punishment decreases it. Positive reinforcement (a reward) adds something pleasant, increasing the behaviour. Negative reinforcement removes something unpleasant, which also increases the behaviour.
**Step 3:** Define punishment. Punishment, whether adding an aversive (positive punishment) or removing a pleasant stimulus (negative punishment), is designed to reduce or suppress a behaviour, not to increase it.
**Step 4:** Match to options. Reward and negative reinforcement both increase behaviour; operant conditioning is the broad framework under which reinforcement strengthens behaviour. Only punishment works to decrease behaviour.
**Step 5:** Therefore the exception is punishment.
Answer: Punishment.

**Quick Tip:** Reinforcement increases behaviour; punishment decreases it.

**292.** All of the following are parts of the cognitive behaviour change technique (stages of change), except:

(A) Pre-contemplation

(B) Consolidation

(C) Action

(D) Contemplation

**Correct Answer:** (B) Consolidation

**Solution:**

**Step 1:** Identify the model. This refers to the Transtheoretical (Stages of Change) model by Prochaska and DiClemente, used to describe how people change a health behaviour.
**Step 2:** List the recognised stages. They are pre-contemplation, contemplation, preparation, action, maintenance, and (relapse). Termination is sometimes added.
**Step 3:** Check each option. Pre-contemplation, contemplation and action are all genuine stages of the model.
**Step 4:** Spot the odd one out. Consolidation is not a named stage in this model; the equivalent later stage is called maintenance, not consolidation.
**Step 5:** Therefore consolidation is the exception.
Answer: Consolidation.

**Quick Tip:** Stages: pre-contemplation, contemplation, preparation, action, maintenance.

**293.** A 60 year old man had undergone cardiac bypass surgery 2 days back. Now he started forgetting things and was not able to recall the names and phone numbers of his relatives. What is the probable diagnosis?

(A) Depression

(B) Post-traumatic psychosis

(C) Cognitive dysfunction

(D) Alzheimer's disease

**Correct Answer:** (C) Cognitive dysfunction

**Solution:**

**Step 1:** Note the timeline. The memory problems appeared just 2 days after coronary artery bypass surgery, an acute post-operative onset.
**Step 2:** Recognise the syndrome. Post-operative cognitive dysfunction (POCD) is a well known complication after cardiac bypass, with impaired memory, attention and recall. It is linked to anaesthesia, cardiopulmonary bypass, microemboli and cerebral hypoperfusion, and is common in older patients.
**Step 3:** Match the picture. Recent memory failure (forgetting relatives' names and numbers) appearing acutely after bypass fits cognitive dysfunction precisely.
**Step 4:** Exclude the others. Alzheimer's disease develops insidiously over years, not over 2 days, so the sudden post-op onset rules it out. Depression can cause poor concentration but is not the best fit for acute isolated memory loss after surgery. Post-traumatic psychosis would feature delusions or hallucinations, which are absent.
**Step 5:** Therefore the answer is cognitive dysfunction.
Answer: Cognitive dysfunction.

**Quick Tip:** Acute memory loss days after bypass surgery equals post-operative cognitive dysfunction.

**294.** Alcohol paranoia is associated with:

(A) Fixed delusions

(B) Drowsiness

(C) Hallucinations

(D) Impulse agitation

**Correct Answer:** (A) Fixed delusions

**Solution:**

**Step 1:** Define the entity. Alcoholic paranoia, also called alcohol-induced delusional disorder, is a chronic alcohol-related psychosis dominated by delusions, classically delusions of jealousy or infidelity (pathological jealousy, the Othello syndrome).
**Step 2:** Identify the hallmark. Its defining feature is fixed, persistent delusions, usually of marital infidelity, held with firm conviction.
**Step 3:** Separate it from look-alikes. Prominent hallucinations point instead to alcoholic hallucinosis, a different disorder, so hallucinations are not the marker here. Drowsiness suggests intoxication or sedation, not a paranoid delusional state. Impulsive agitation describes withdrawal or delirium, not the stable delusional theme of alcoholic paranoia.
**Step 4:** Match to the options. The feature that uniquely belongs to alcoholic paranoia is fixed delusions.
**Step 5:** Therefore the answer is fixed delusions.
Answer: Fixed delusions.

**Quick Tip:** Alcoholic paranoia equals delusions of jealousy; hallucinations belong to alcoholic hallucinosis.

**295.** Autistic disorder is characterised by all of the following, except:

(A) Visual impairment

(B) Lack of social interaction

(C) Delayed development of speech

(D) Stereotypic movements

**Correct Answer:** (A) Visual impairment

**Solution:**

**Step 1:** Recall the triad. Autism spectrum disorder is defined by impaired social interaction, impaired communication (including delayed or abnormal speech and language), and restricted, repetitive, stereotyped behaviours or movements.
**Step 2:** Test each true feature. Lack of social interaction is a core deficit. Delayed development of speech is a classic communication feature. Stereotypic movements (hand flapping, rocking, spinning) are the repetitive-behaviour component.
**Step 3:** Test the odd one out. Visual impairment is a sensory/ophthalmological problem and is not a defining feature of autism. Children with autism have normal vision as a rule; their gaze abnormalities (poor eye contact) are social, not due to defective sight.
**Step 4:** Conclude. The feature that does not belong is visual impairment.
Answer: Visual impairment.

**Quick Tip:** Autism triad: social deficit, speech delay, stereotypies; vision is normal.

**296.** Which of the following is not a cognitive error or dysfunction?

(A) Catastrophic thinking

(B) Arbitrary inference

(C) Overgeneralization

(D) Thought block

**Correct Answer:** (D) Thought block

**Solution:**

**Step 1:** Define cognitive errors. In Beck's cognitive theory, cognitive errors (cognitive distortions) are faulty patterns of thinking seen in depression and anxiety. They include catastrophising, arbitrary inference, overgeneralisation, selective abstraction, magnification, minimisation, personalisation and dichotomous thinking.
**Step 2:** Check the cognitive-error options. Catastrophic thinking (expecting the worst), arbitrary inference (drawing a conclusion without evidence) and overgeneralisation (applying one event to all situations) are all recognised cognitive distortions.
**Step 3:** Examine thought block. Thought block is a disorder of the stream/form of thought, where the train of thought suddenly stops. It is a sign seen in schizophrenia, not a cognitive distortion of the depressive type.
**Step 4:** Distinguish the categories. Cognitive errors are about distorted content/interpretation of thinking, whereas thought block is a disturbance in the flow of thought. Thought block therefore does not belong to the list of cognitive errors.
**Step 5:** Hence the answer is thought block.
Answer: Thought block.

**Quick Tip:** Thought block is a formal thought disorder, not a Beckian cognitive distortion.

**297.** A patient presents to the emergency department with self harm and indicates suicidal intent. Which of the following conditions does not warrant an immediate specialist assessment?

(A) Formal thought disorder

(B) Acute severe intoxication

(C) Chronic severe physical illness

(D) Social isolation

**Correct Answer:** (B) Acute severe intoxication

**Solution:**

**Step 1:** Understand the clinical setting. A patient with self harm and stated suicidal intent must be risk stratified. The question asks which feature does NOT call for immediate psychiatric specialist assessment.
**Step 2:** Acute severe intoxication is the answer. When a person is acutely and severely intoxicated with alcohol or drugs, their mental state cannot be reliably assessed. Suicidal statements made while heavily intoxicated may not reflect true intent. Standard practice is to first medically stabilise and let the patient become sober, then do the formal psychiatric assessment. So immediate specialist assessment is deferred, not done at once.
**Step 3:** Why the others do warrant immediate assessment. Formal thought disorder (option A) points to an active psychotic illness such as schizophrenia, which raises suicide risk and needs urgent psychiatric review. Chronic severe physical illness (option C) is an established high risk factor for suicide and needs prompt evaluation. Social isolation (option D) is a recognised risk factor that increases the danger and requires urgent attention.
**Step 4:** Therefore the condition that does not warrant immediate assessment is acute severe intoxication, because assessment is only meaningful once the patient is sober and medically stable.
**Answer: Option B (Acute severe intoxication).**

**Quick Tip:** You cannot get a reliable mental state exam from someone who is heavily drunk or drugged.

**298.** Lymphatics from the spongy urethra drain into the following lymph nodes:

(A) Superficial inguinal nodes

(B) Internal inguinal nodes

(C) Deep inguinal nodes

(D) Sacral nodes

**Correct Answer:** (C) Deep inguinal nodes

**Solution:**

**Step 1:** Recall the parts of the male urethra: prostatic, membranous, and spongy (penile) urethra. The question is about lymphatic drainage of the spongy urethra.
**Step 2:** Lymph from the spongy (penile) urethra drains mainly to the deep inguinal nodes. Some lymphatics may also reach the external iliac nodes, but the deep inguinal nodes are the principal and best answer among the options given.
**Step 3:** Compare with neighbouring structures so the distractors are clear. The skin of the penis and the scrotum drain to the superficial inguinal nodes. The glans penis and the spongy urethra drain to the deep inguinal nodes. The prostatic and membranous urethra drain to the internal iliac nodes.
**Step 4:** Evaluate the options. Superficial inguinal nodes (A) take skin of penis and scrotum, not the spongy urethra. Internal inguinal nodes (B) is not a standard named group. Sacral nodes (D) drain the rectum and pelvic viscera, not the spongy urethra. Deep inguinal nodes (C) is correct.
**Answer: Option C (Deep inguinal nodes).**

**Quick Tip:** Spongy urethra and glans follow the same path as the distal penis, into the deep inguinal group.

**299.** Branched chain ketoacid decarboxylation is defective in:

(A) Maple syrup urine disease

(B) Hartnup disease

(C) Alkaptonuria

(D) GM1 gangliosidosis

**Correct Answer:** (A) Maple syrup urine disease

**Solution:**

**Step 1:** Identify the metabolic step. The branched chain amino acids leucine, isoleucine and valine are first transaminated to their branched chain alpha ketoacids. The next step is oxidative decarboxylation of these ketoacids by the

branched chain alpha ketoacid dehydrogenase complex.
**Step 2:** A deficiency of branched chain alpha ketoacid dehydrogenase causes Maple syrup urine disease (MSUD). The branched chain ketoacids and their amino acids accumulate in blood and urine, giving the urine a characteristic sweet maple syrup smell.
**Step 3:** Rule out the distractors. Hartnup disease (B) is a defect in the renal and intestinal transport of neutral amino acids, especially tryptophan, not branched chain ketoacid metabolism. Alkaptonuria (C) is a deficiency of homogentisate oxidase in the tyrosine pathway, causing dark urine and ochronosis. GM1 gangliosidosis (D) is a lysosomal storage disease from beta galactosidase deficiency, unrelated to amino acid catabolism.
**Step 4:** The only condition with a defect in branched chain ketoacid decarboxylation is Maple syrup urine disease.
**Answer: Option A (Maple syrup urine disease).**

**Quick Tip:** Defective branched chain alpha ketoacid dehydrogenase gives urine that smells of maple syrup.

**300.** A 60 year old male is brought by his wife. He thinks that he has committed sins all through his life. He is very depressed and has considered suicide but has not thought about how to go about it. He has also attended sessions with a spiritual guru. He is not convinced by his wife that he has led a pious life. He does not want to hear anything to the contrary. How will you treat him?

(A) Antipsychotic plus antidepressant

(B) Antidepressant alone

(C) Antipsychotic alone

(D) Electroconvulsive therapy alone

**Correct Answer:** (A) Antipsychotic plus antidepressant

**Solution:**

**Step 1:** Read the picture. A 60 year old man has low mood, guilt, suicidal thoughts, and a fixed false belief that he has sinned and is bad, which he holds even when his wife gives evidence to the contrary. This is depression with mood congruent delusions of guilt, that is, psychotic depression.
**Step 2:** The OCR cut off three options. The standard MSQ choices for this classic stem are: A. Antipsychotic plus antidepressant, B. Antidepressant alone, C. Antipsychotic alone, D. ECT alone. The printed key is A.
**Step 3:** Treatment principle for psychotic depression. A single antidepressant is not enough because of the delusions, and an antipsychotic alone does not treat the core depression. The recommended drug treatment is the combination of an antidepressant and an antipsychotic.
**Step 4:** Why not the others. Antidepressant alone (B) leaves the psychotic features untreated. Antipsychotic alone (C) leaves the mood disorder untreated. ECT (D) is highly effective for psychotic depression and is in fact a strong option, but it is usually reserved for severe, refractory, or high suicide risk cases, so the first line answer chosen here is the drug combination.
**Answer: Option A (Antipsychotic plus antidepressant).**

**Quick Tip:** Depression plus a fixed delusion of sin means psychotic depression, so treat both the mood and the psychosis.