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:

The question is really asking which non-depolarising blocker you can give safely when both the liver and the kidney are not working well. The clues are a high bilirubin (8 mg/dl) and a high creatinine (1.9 mg/dl).

Think about how each drug leaves the body. Pancuronium and vecuronium both lean on renal excretion, and vecuronium also uses biliary excretion, so a damaged kidney or liver makes their effect last far too long. Rocuronium is mostly handled by the liver, so a jaundiced patient will stay paralysed longer than expected.

Atracurium breaks down on its own through Hofmann elimination, a spontaneous chemical degradation at normal body pH and temperature, plus ester hydrolysis in plasma. Neither pathway needs a functioning liver or kidney. That makes its duration predictable even when both organs fail, which is exactly this patient's situation.

So the safe pick is atracurium.

Atracurium

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

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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:

Focus on one contradiction in the vignette. The woman is fully awake and aware, but her chest and upper body barely move and she cannot breathe well on her own. Her heart rate and blood pressure are fine.

If the problem were the opioid itself, fentanyl would have dulled her consciousness, yet she is alert, so deep opioid respiratory depression does not fit. Fentanyl chest wall rigidity is a real event, but it strikes during induction while the patient is anaesthetised, not in the recovery room after extubation. A pulmonary embolus would knock her vitals around with a fast heart and falling pressure, which is not described.

The one diagnosis that explains an awake brain sitting on top of weak muscles is leftover muscle relaxant. Pancuronium is long lasting and depends on the kidneys, and in an overweight patient its recovery is hard to predict. If reversal was incomplete, she stays conscious but cannot move her respiratory muscles properly.

That is residual neuromuscular blockade.

Incomplete reversal of pancuronium

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

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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:

The trick here is that succinylcholine causes a depolarising (phase I) block, and a depolarising block behaves like a steady, even weakening of every response rather than a progressive collapse.

Run through the monitoring signs. On a train of four, all four twitches shrink together with no fade, so the no-fade statement is correct. After a tetanic burst there is no boost to the next twitch, so the no post-tetanic-facilitation statement is also correct. The train of four ratio stays high, comfortably above 0.4, so that statement is correct too.

The one that does not belong is fade on tetanic stimulation. Fade, whether on the train of four or on tetanus, is the hallmark of a non-depolarising block (or a phase II block), where acetylcholine release cannot keep up. A clean depolarising block holds its tetanic response without fading.

Since the stem asks for the exception, the false statement is the fade on tetanic stimulation.

Fade on tetanic stimulation

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

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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:

Take the two clues one at a time. First, an end tidal CO2 was recorded, which tells you the tube is sitting in the airway. If the tube had slid into the oesophagus, you would see almost no CO2, so you can drop the oesophageal option straight away.

Second, the left lung is quiet while the right is fine. With the tube confirmed in the trachea, a quiet left side after a hurried, fast push usually means the tube went in too far and slipped into the right main bronchus. The right main bronchus is the natural destination because it is wider, shorter and more in line with the trachea, so only the right lung gets ventilated and the left falls silent.

A blocked tube would shut down ventilation altogether and kill the CO2 trace, and bronchospasm would tighten both lungs with wheeze and high pressures, neither of which matches a one-sided quiet chest.

So a deeply placed tube has gone down the right bronchus.

Endobronchial intubation

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

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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:

Start from where the hypogastric sheath actually lives. It is deep inside the pelvis, slung between the lateral pelvic wall and the pelvic viscera, acting as a conduit for the internal iliac vessels and the nerves heading to the bladder, uterus and rectum.

Anything that sits deep in the pelvis and wraps the pelvic organs has to be part of the pelvic (endopelvic) fascia, because that is the connective-tissue system of the pelvic cavity. The hypogastric sheath is simply a thickened, organised part of that visceral pelvic fascia.

Now rule out the others by location. Scarpa's fascia is up on the lower abdominal wall. Colles' fascia is the membranous fascia of the perineum below. The inferior layer of the urogenital diaphragm, the perineal membrane, is also a perineal structure. All three are outside the pelvic cavity proper, so none can form a pelvic visceral sheath.

That leaves the pelvic fascia as the parent layer.

Pelvic fascia

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

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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:

Picture the upper abdomen at the L1 level. The aorta runs straight down the midline, hugging the front of the spine. The celiac plexus is the dense net of autonomic fibres wrapped around the celiac trunk as it leaves the aorta.

Since the celiac trunk springs forward off the front of the aorta, the plexus that surrounds it naturally sits in front of and on either side of the aorta. That is the meaning of anterolateral to the aorta. It cannot be behind the vessel, so a posterolateral position is wrong.

The sympathetic chains are a separate landmark, running down the sides of the vertebral bodies, lateral to the plexus. Describing the celiac plexus by the sympathetic chain misplaces it, because the plexus actually lies anterior and medial to those chains, not as options C and D phrase it.

This periaortic, anterolateral seat is also why a celiac plexus block is aimed there to relieve pain from pancreatic and other upper abdominal cancers.

So the best description is anterolateral to the aorta.

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:

Group the gluteal region by the two named gluteal nerves. The superior gluteal nerve comes out above the piriformis and takes care of gluteus medius, gluteus minimus and tensor fasciae latae. The inferior gluteal nerve comes out below the piriformis and serves only one muscle, the big gluteus maximus.

Gluteus medius belongs to the upper group, so its nerve is the superior gluteal nerve. A useful clinical anchor is the Trendelenburg sign: if the superior gluteal nerve is injured, the hip abductors fail and the pelvis sags toward the normal side while walking.

The remaining choices are easy to reject. The inferior gluteal nerve is committed to gluteus maximus. The nerve to obturator internus and the nerve to quadratus femoris run the deep short rotators, not gluteus medius.

The same logic carries over to the blood supply asked in the second part: the superior gluteal artery travels with its matching nerve above the piriformis and feeds gluteus medius, rather than the inferior gluteal, obturator or ilioinguinal vessels.

So both the nerve and the artery of gluteus medius are the superior gluteal ones.

Superior gluteal nerve

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

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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:

The sustentaculum tali is a small ledge on the inner side of the calcaneus that props up the talus, and the key is to know what touches its lower surface.

On the under-surface there is a smooth groove, and the tendon that glides through it is flexor hallucis longus on its way to the big toe. The shelf acts like a pulley for that tendon, which is the whole point of remembering this relation. So the tendon passing below the sustentaculum tali is flexor hallucis longus.

The distractors fall away with location. Tibialis anterior stays on the front of the ankle and inserts on the medial cuneiform and first metatarsal. Flexor digitorum longus runs on the medial side and lies above, not below, the sustentaculum.

The companion part of the question asks what attaches to the sustentaculum, and that is the tibialis posterior, which fans out onto the navicular and tarsal bones and gives a slip to the sustentaculum tali. So one tendon grooves under the shelf (flexor hallucis longus) while another attaches to it (tibialis posterior).

Therefore the tendon passing below the sustentaculum tali is flexor hallucis longus.

Flexor hallucis longus

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

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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:

The question tests which named structure traverses the foramen magnum. Think of the foramen magnum as the gateway where the brainstem becomes the spinal cord. Walking through it you would find the medulla oblongata wrapped in meninges, the two vertebral arteries climbing into the cranium, the single anterior and paired posterior spinal arteries, the spinal portion of the accessory nerve, and supporting ligaments such as the apical ligament of the dens and the tectorial membrane.

From the four options, only the vertebral artery clearly belongs to this list. It ascends through the transverse foramina of the upper cervical vertebrae, then pierces the dura and arachnoid and enters the skull through the foramen magnum to join its fellow and form the basilar artery.

The internal carotid artery uses a completely different route, the carotid canal in the petrous temporal bone, so it is excluded. The sympathetic chain stays outside the skull entirely, lying along the vertebral bodies in the neck and thorax. The accessory nerve is a deliberate trap: only its spinal root passes upward through the foramen magnum, while the nerve trunk leaves the skull through the jugular

foramen, so labelling cranial nerve XI as the foramen magnum structure is incorrect.

∴ the vertebral artery is the structure that passes through the foramen magnum.

Vertebral artery

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

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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:

Raising the arm overhead is a team effort between the ball and socket joint and the shoulder girdle, captured by the scapulohumeral rhythm in which roughly two thirds of the arc comes from the glenohumeral joint and one third from the scapula.

Run through each option as a true or false statement. Elevation of the humerus is the basic action of lifting the arm, so it is true. For the scapula to swing its socket upward and keep the head of the humerus seated, the scapula must turn so the glenoid faces up; anatomically this is upward or lateral rotation. As the scapula rotates, the clavicle has to pivot, and because of the curved shape of the clavicle this includes a twist about its long axis, the axial or crankshaft rotation at the sternoclavicular joint, so that statement is true. None of this scapular and clavicular motion is possible without sliding at the acromioclavicular and

sternoclavicular joints, so acromioclavicular joint movement is also true.

That leaves the description medial rotation of the scapula. Since the scapula actually rotates laterally (glenoid upward) during abduction, calling it medial rotation is wrong, and an except question wants the false statement.

⇒ the movement that does not occur is medial rotation of the scapula.

Medial rotation of scapula

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

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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:

The key idea here is the definition of a composite muscle: one that arises from two separate myotomic masses and as a result carries a dual nerve supply, with each part keeping the nerve of its original mass.

Test the options against that rule. Flexor digitorum profundus splits its innervation between the median nerve through the anterior interosseous branch for the index and middle finger portion and the ulnar nerve for the ring and little finger portion, a textbook dual supply. Pectineus straddles the boundary between the anterior and medial thigh compartments, so it commonly receives both the femoral nerve and the obturator nerve. Biceps femoris is built from a long head

and a short head that take their nerves from the two divisions of the sciatic nerve, tibial for the long head and common fibular for the short head. All three therefore qualify as composite.

Flexor carpi ulnaris breaks the pattern. It comes from the single common flexor origin and is innervated only by the ulnar nerve, with no second nerve and no second mass. It is a simple, not composite, muscle.

∴ the muscle that is not composite is flexor carpi ulnaris.

Flexor carpi ulnaris

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

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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:

Picture the urogenital diaphragm as a triangular sandwich slung between the ischiopubic rami in front of the perineum. The filling of the sandwich is muscle, and the two slices of bread are fascia.

The muscular filling is the deep transverse perinei together with the sphincter urethrae. The lower fascial slice is the perineal membrane (inferior fascia of the urogenital diaphragm), and the upper slice is the superior fascia of the urogenital diaphragm. So the deep transverse perinei, the perineal membrane and the

sphincter urethrae are all legitimate building blocks of this structure.

Colles fascia is something else entirely. It is the membranous part of the superficial fascia of the perineum, lying superficial to the perineal membrane and forming the floor of the superficial perineal pouch. It is not part of the deep urogenital diaphragm, which is exactly what an except question is looking for.

⇒ Colles fascia is not a component of the urogenital diaphragm.

Colles fascia

Quick Tip: The diaphragm is two muscles between two fascial layers; one named fascia belongs to the superficial pouch instead.

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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:

Post-ductal coarctation places a tight waist in the aorta just beyond the left subclavian artery, so the body must build a detour to carry blood from the high-pressure arch territory down to the low-pressure descending aorta. The detour relies on vessels that branch off the subclavian arteries above the block and anastomose with vessels that empty into the aorta below the block.

The workhorse of this detour is the link between the internal thoracic artery and its anterior intercostal branches on one side and the posterior intercostal arteries

on the other. The scapular anastomosis adds extra capacity, with the suprascapular and subscapular arteries enlarging and feeding blood toward the intercostal system. The enlarged, tortuous posterior intercostal arteries are what erode the lower borders of the ribs and produce the classic rib notching on a chest film. So the suprascapular, subscapular and posterior intercostal arteries are all real collateral routes.

The vertebral artery does the opposite job: it heads upward to supply the hindbrain and has no role in shunting blood around the aortic narrowing into the descending aorta. It is the odd one out.

∴ the vessel that does not form a collateral in post-ductal coarctation is the vertebral artery.

Vertebral artery

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

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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:

To answer this, trace the embryology of the great veins. Early in development paired anterior cardinal veins drain the head end of the embryo. On the right side the system goes on to form the normal right superior vena cava. On the left side the anterior cardinal vein is meant to disappear after the left brachiocephalic vein

carries its blood across to the right.

When that left vessel fails to regress, it persists as a left superior vena cava running down the left side of the mediastinum. The question is where this extra vein delivers its blood. The left horn of the sinus venosus has already become the coronary sinus, and the persistent left superior vena cava drains straight into that coronary sinus.

Because the coronary sinus opens into the right atrium, the blood does finally reach the right atrium, but only after passing through an enlarged coronary sinus. So the immediate and correct termination named in the options is the coronary sinus, not the right atrium directly. Drainage into the left atrium is a rare cyanotic variant and into the pericardial space does not occur, so those options are wrong.

⇒ a persistent left superior vena cava typically drains into the coronary sinus.

Coronary sinus

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

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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:

Keep the two lung blood supplies separate in your mind. The pulmonary circulation is the functional supply that takes venous blood to the alveoli for

oxygenation, while the bronchial circulation is the nutritive supply, a branch of the systemic circulation that feeds the airways and lung framework.

Grade each statement against that division. Saying the bronchial arteries make up around 2% of the systemic output is accurate. Saying they nourish the lung is exactly their defining function, so that is accurate too. The trickier statement is venous admixture: a portion of bronchial venous blood, which is deoxygenated, empties into the pulmonary veins and mixes with oxygenated blood returning to the left heart, slightly lowering its oxygen content; this is one reason a normal person has a small alveolar to arterial oxygen gradient, so that statement is also true.

The statement that fails is the one crediting the bronchial circulation with gaseous exchange. Oxygen uptake and carbon dioxide removal happen at the alveolar capillary membrane served by the pulmonary circulation, not by the bronchial vessels. An except question wants this false statement.

∴ the untrue statement is that the bronchial circulation contributes to gaseous exchange.

Contributes to gaseous exchange

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

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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:

This item asks you to look past gas exchange and recall the metabolic side jobs of the lung. The pulmonary vascular bed is a busy biochemical organ: it filters microemboli, stores blood, deactivates serotonin and bradykinin, and most importantly contains angiotensin converting enzyme on its endothelium.

That ACE step is the link to the options. As blood passes through the lung, ACE converts angiotensin I into the active angiotensin II. Angiotensin II then stimulates aldosterone secretion, and aldosterone makes the kidney retain sodium. So the lung sits at the start of the cascade that controls how much sodium the body holds onto, tying the lung to sodium balance.

The other electrolytes do not run through the lung in this way. Potassium is handled mainly by the kidney and by insulin and acid base shifts, calcium is governed by parathyroid hormone, vitamin D, the gut and bone, and the broad anion balance is again a renal and metabolic affair. None of these depends on a pulmonary enzymatic step.

$\Rightarrow$ among the choices, the lung most clearly influences sodium balance through its ACE driven angiotensin and aldosterone pathway.

$\boxed{\text{Sodium balance}}$

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

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

(A) Stomach

(B) Jejunum

(C) Ileum

(D) Colon

Correct Answer: (B) Jejunum

SOLUTION:

Think of the question as asking which gut segment recovers the most water by volume.

The intestine receives about 9 litres of fluid per day once you add dietary intake to the various digestive secretions. The job of getting that water back falls almost entirely on the small bowel, which returns roughly 8 to 8.5 litres. The colon mops up only the leftover litre or so.

Water movement across the gut wall is passive and follows solutes, chiefly sodium that is pumped out and the sodium dragged along with glucose and amino acids. This active solute transport peaks in the proximal small intestine, which is the jejunum. Where solute uptake is highest, water uptake is highest too, so the jejunum wins.

Quick rule-out: the stomach absorbs essentially no water, the ileum specialises in bile salt and B_{12} recovery rather than bulk water, and the colon deals with a far smaller residual volume even though it concentrates the stool.

Jejunum

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

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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:

Nitric oxide in the digestive tract is best understood as the brake of the enteric nervous system. It is the main transmitter of the inhibitory NANC neurons, the nerves that tell gut muscle to let go rather than to squeeze.

Mechanistically, NO crosses into the smooth muscle cell and switches on soluble guanylyl cyclase. The resulting rise in cyclic GMP pulls calcium out of the cytosol, and with less calcium the contractile machinery cannot engage, so the muscle relaxes.

This relaxing action underlies several everyday gut functions: the stomach relaxing to accept a meal, the segment of bowel ahead of a bolus opening up so the bolus can advance, and sphincters like the lower oesophageal and internal anal sphincters loosening at the right moment.

The trap in the options is vasodilatation. NO certainly dilates vessels, but the stem specifically asks for its leading role inside the GI tract, which is muscle relaxation. Vasoconstriction and slow contraction describe the reverse of NO action, so both are wrong.

Gastrointestinal smooth muscle relaxation

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

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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:

The discomfort of an abdominal spasm is the result of cramping smooth muscle in the gut wall, so anything that relieves it must allow that muscle to settle and must quieten the pain pathway.

A hot water bottle delivers local heat. Warmth relaxes the cramped smooth muscle and boosts local circulation, helping clear the metabolites that build up during a sustained spasm. On top of that, warm sensation competes with pain signals at the spinal level, a counterirritation effect that reduces felt pain.

Now match this to the autonomic options. In the gastrointestinal tract the sympathetic (adrenergic) system relaxes smooth muscle and slows motility, while the parasympathetic (cholinergic) system drives contraction. A treatment that relieves spasm therefore behaves like an adrenergic, relaxing influence; cholinergic stimulation would only tighten the cramp, ruling out option B.

The receptor-based options do not fit either. Heat does not relieve pain by switching off cold receptors, and it certainly does not inhibit heat receptors; warmth actually activates the warm receptors. Among the choices given, the adrenergic, relaxation-oriented answer is the best fit, and it agrees with the printed key.

Stimulation of adrenergic fibers

Quick Tip: Spasm relief means smooth muscle relaxation, which in the gut is the

adrenergic effect.

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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:

Vitamin K is needed for the liver to finish making four specific clotting factors. Without it, these factors are produced but stay inactive because they cannot undergo gamma-carboxylation, the step that gives them calcium-binding ability and lets them sit on phospholipid membranes during coagulation.

The four factors to remember are II, VII, IX and X. The classic mnemonic is the year 1972, read as factors $10, 9, 7, 2$. The natural anticoagulants protein C and protein S share this vitamin K dependence, which is why warfarin affects both clotting and anticlotting proteins.

Scanning the answer choices, only option B, listing IX and X, gives a pair where both members belong to the vitamin K dependent group. Every other option is spoiled by at least one factor that is not vitamin K dependent: factor III (tissue factor), factor V, factor $VIII$ and factor XII all sit outside the group.

$$\boxed{\text{IX and X}}$$

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

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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:

The lung circulation is built to be soft and low in pressure. Its key trick is that flow can jump dramatically while pressure stays almost flat, which is exactly what happens when exercise pushes cardiac output up several fold.

The reason lies in spare capacity. At rest, a large portion of pulmonary capillaries, particularly toward the lung apex, are shut or nearly so because local pressure is too low to keep them open. They form a reserve waiting to be used.

During exercise the modest bump in pulmonary pressure pops these closed vessels open, a process termed recruitment, and it also widens vessels that were already carrying flow, termed distension. Adding more parallel channels lowers the overall pulmonary vascular resistance.

So as flow climbs, resistance falls in step, and the product, which determines pressure, hardly moves. The single best phrase for this among the choices is an increase in the number of open capillaries.

The distractors fail on basic anatomy and physiology: pulmonary arteries are thin walled with scant smooth muscle, the adaptation is mainly passive rather than a sympathetic distensibility effect, and the pulmonary capillary surface area is enormous rather than small.

Increase in the number of open capillaries

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

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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:

The phrasing asks for the odd one out: which listed item does not help blood get back up to the heart while a person stands still.

Standing is a challenge for venous return because blood tends to pool in the legs. The body counters this with several aids. The calf muscle pump is the leading one, since each calf contraction milks the deep veins upward. The valves in the perforating veins keep that flow one way, channelling blood from superficial to deep veins and blocking reflux. The firm deep fascia wrapping the calf turns muscle contraction into effective venous compression, sharpening the pump.

Gravity is the outlier. Standing increases the column of blood, raising pressure in the dependent veins and encouraging pooling in the legs. While the arterial pressure at the feet also rises, the net gravitational effect works against returning blood to the heart rather than helping it. That makes the gravitational increase in arterial pressure the exception.

Gravitational increase in arterial pressure

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

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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:

Sharp cardiac images depend on catching the heart when it is moving the least, because any wall motion smears the picture.

Across one heartbeat there are only two quiet intervals. One is at end systole, and the other is in the middle of diastole, a phase known as diastasis. Diastasis falls after the rush of early ventricular filling and before the atria contract, so for that brief span the ventricle is barely moving.

For routine imaging at usual or slow heart rates, this mid diastolic diastasis is the longer and steadier of the two rest windows, which is why coronary CT is timed to it.

The remaining choices all coincide with movement: both mid and late systole feature forceful contraction, and late diastole brings the atrial kick that sets the heart moving again. That leaves mid diastole as the still point.

Mid diastole

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:

To find the inhibitor of insulin release, picture the pancreatic beta cell and what each listed agent does to it.

Epinephrine is the clear suppressor. By engaging alpha-2 adrenergic receptors on the beta cell, it cuts intracellular cyclic AMP and shuts down insulin output. This fits the logic of the stress response: when adrenaline surges, the body wants glucose available in the blood for muscle and brain, so storing it away with insulin would be counterproductive.

The rest of the list pushes insulin the other way. The gut hormones secretin and gastrin both nudge the beta cell to release more insulin in the fed state, sharing in the incretin-type response that prepares the body for an incoming glucose load. Growth hormone is a more subtle case: it opposes insulin action in tissues and is diabetogenic, yet it stimulates insulin secretion and tends to drive insulin levels up rather than down.

That leaves epinephrine as the single agent that inhibits insulin secretion, via its

alpha-2 effect on the beta cell.

Epinephrine

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:

Mammalian females show two broad patterns of reproductive cycling, and the trick here is to know which one applies to people. Most non-primate mammals run an ***estrous*** cycle: receptivity is restricted to a heat period, and an unused uterine lining is simply resorbed without external bleeding. Primates including humans run a ***menstrual*** cycle instead, in which the unused endometrium is visibly shed as bleeding and there is no fixed heat window. Within the human menstrual cycle we describe the events ovary side as the ovarian cycle (follicular then luteal phase) and the uterus side as the endometrial or uterine cycle (proliferative, secretory and menstrual phases). So menstrual, ovarian and endometrial cycles are all genuine human cycles, whereas the estrous cycle belongs to other mammals and is not found in humans.

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:

Sort the four choices by where they come from. The cerebellar hemispheres time and smooth voluntary limb movement, so when they are injured the patient cannot coordinate movement properly. This shows up as incoordination, as an unsteady ataxic gait, and as an intention tremor that grows as the finger nears its target. Three of the options are therefore standard cerebellar findings. The odd one out is resting tremor, which describes a tremor seen while the limb is relaxed and supported. That pattern points to the basal ganglia and is the hallmark of Parkinsonism, not of cerebellar pathology. Cerebellar tremor behaves in the opposite way, being quiet at rest and emerging only with action. The exception asked for is the resting tremor.

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:

Ask what is actually burning the calories at rest. The bulk of resting energy expenditure comes from organs and muscle that are metabolically busy all the time, and together these form the fat-free or lean body mass. Adipose tissue, by contrast, sits there using almost no energy, so a heavier person is not automatically a higher burner if the extra weight is fat. That is why two people of identical weight differ in resting expenditure when one is muscular and the other is obese, and it is why lean body mass tracks *BMR* better than total weight, body mass index or degree of obesity. Body surface area is only a reporting convention used to express the value in standard units, not the tissue performing the metabolism. The closest true determinant is lean body mass.

$$\boxed{\text{Lean body mass}}$$

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:

Three of these options clearly push metabolism up. An overactive thyroid floods the tissues with hormone that boosts oxygen use and heat output, so hyperthyroidism is a classic high ***BMR*** state. Eating triggers the thermic effect of food, the energy cost of digesting and processing nutrients, which lifts expenditure for hours after a meal. Physical exercise obviously drives muscle metabolism and oxygen consumption upward. That leaves obesity as the only choice associated with a lower value, because the surplus tissue in obesity is mostly inert fat; once the rate is normalised to body weight or surface area, the obese individual reads relatively low. So the decreased basal metabolic rate among the options belongs to obesity.

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:

Frame it as: which option breaks the low-protein pattern. Draining the subarachnoid space repeatedly, as with frequent taps or a persistent leak, removes protein-rich fluid quicker than it can be made, dropping the protein level. Idiopathic intracranial hypertension, the pseudotumor cerebri picture, is the high-pressure but normal-to-low protein syndrome, so it fits the low side. Infants are commonly grouped among the low CSF protein states. Hypothyroidism does the

opposite: myxedema is a textbook cause of a raised CSF protein, sitting alongside conditions like Guillain Barre and acoustic neuroma in that elevated-protein list. Since the stem wants the one that is not low, the thyroid-deficiency state is the outlier.

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:

Match each disorder to its enzyme and the molecule it cannot clear. The clue word is sphingomyelin, the substrate of acid sphingomyelinase. When that enzyme fails, sphingomyelin piles up inside lysosomes, producing the foamy storage cells, big liver and spleen, and in the infantile form the cherry-red spot of Niemann-Pick disease. Run the distractors to be sure: Farber disease lacks ceramidase and stores ceramide; Tay-Sachs lacks hexosaminidase A and stores *GM2* ganglioside; Krabbe lacks galactocerebrosidase and stores galactocerebroside. None of those touch sphingomyelin. The enzyme named in the stem therefore identifies Niemann-Pick disease.

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:

Think about how electrophoresis ranks lipoproteins. The separation depends on charge-to-size, and the apoprotein-rich, small ***HDL*** particles travel furthest as the alpha band. ***VLDL*** lands in the pre-beta zone and ***LDL*** in the beta zone, so all three creep toward the anode. Chylomicrons are the giants of the group: enormous, packed with triglyceride, and carrying very little protein per unit size, which leaves them with almost no effective surface charge to drag them through the gel. As a result they are stuck at the origin where the sample was loaded and fail to migrate toward the charged end. The non-migrating particle is therefore the chylomicron.

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:

The key is the bond chemistry of a fructan. Inulin is built from fructose residues stitched together through their anomeric carbon, which in fructose is $C2$, and crucially these bonds are in the beta orientation, typically beta(2 to 1). Our digestive machinery, the amylases of saliva and pancreas plus the small-intestinal brush-border enzymes, is specialised for alpha glycosidic bonds like those in starch and cannot touch beta linkages. Because every internal bond in inulin sits in that beta configuration at the anomeric C2, the molecule slips through the upper gut intact and only the colonic bacteria can ferment it, which is exactly what makes it a prebiotic. Stomach pH does not cleave these bonds, the alpha-linkage option is the opposite of the truth, and merely saying an enzyme is absent skips the actual chemical reason. The precise cause is the beta configuration at the anomeric C2.

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:

The clue in this question is the phrase about charged molecules being held and then swapped out by other charged molecules in the moving phase. That swapping of ions is the signature of one technique only.

Think about what each method actually sorts by. Affinity chromatography relies on a lock-and-key biological recognition, like a receptor grabbing its ligand, so charge alone is irrelevant. Adsorption chromatography ranks molecules by how strongly they stick to a surface, driven by polarity. Size-exclusion chromatography is essentially a molecular sieve, separating purely by physical size, with bulky molecules slipping past the porous beads first.

None of those involves an exchange of charged groups. The method built around fixed charged sites on the resin that capture oppositely charged solutes, then surrender them when competing ions flood the mobile phase, is ion-exchange chromatography. A resin bearing positive groups will trap negatively charged molecules and later let them go as more ions arrive, which is precisely what the stem narrates.

Ion-exchange chromatography

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:

This question is really asking which tool is versatile enough to do two jobs at the same time: read changes in the genetic code and read how busy genes are.

The three blotting methods are each single-purpose. The classic memory aid is SNoW DRoP: Southern handles DNA, Northern handles RNA, Western handles protein. So Southern alone could flag a DNA change, and Northern alone could report expression, but neither does both. Western is concerned only with the protein product.

Microarray technology breaks that one-job limit. A chip is printed with vast numbers of known probe sequences. When you hybridize genomic DNA to it, you can pick up single nucleotide polymorphisms, deletions and copy-number shifts, which is sequence variation. When you instead hybridize labeled transcripts (cDNA or cRNA), the signal intensity at each spot reflects how much of that gene's mRNA is present, giving a full expression snapshot across thousands of genes. One platform, two readouts.

Microarray

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:

To answer this we must separate tools that count chromosomes from tools that read gene activity. Aneuploidy is a problem of chromosome number, so the odd one out will be whatever cannot measure chromosome dosage.

Start with the genuine aneuploidy detectors. FISH lights up specific chromosomes with colored probes, and you literally count the spots: three signals for chromosome 21 means trisomy 21. QF-PCR is a rapid prenatal favorite that amplifies repeat markers and compares fluorescent peak heights, where an unusual ratio betrays an extra chromosome. Chromosomal microarray scans copy number genome-wide and will flag the gain of an entire chromosome just as easily as a small deletion, so it too detects aneuploidy.

That leaves RT-PCR. Its job is to turn messenger RNA into cDNA and quantify transcripts, which tells you how strongly a gene is being expressed, not how many copies of a chromosome a cell carries. It simply is not a chromosome-counting assay. Note the original answer key listed microarray, but that is wrong because microarray does catch whole-chromosome imbalances, so the corrected choice is RT-PCR.

$$\boxed{\text{RT-PCR}}$$

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:

Hemophilia is a textbook X-linked recessive condition caused by mutations in a single clotting-factor gene, factor VIII in type A and factor IX in type B. So prenatal diagnosis boils down to finding that mutation in fetal tissue obtained by chorionic villus sampling or amniocentesis.

The cleanest way to do that is to amplify the gene region with PCR and read the mutation directly. For severe hemophilia A, PCR can even pick up the common intron 22 inversion. Direct PCR mutation detection is fast, specific and does not depend on other family members being available, which is why it is the test of choice.

The other options fall short. Linkage analysis is an indirect, marker-tracking method reserved for situations where the family mutation has not been characterized, and it can be tripped up by recombination, so it is second-line. Cytometry quantifies cells and surface proteins, useless for spotting a coagulation-gene mutation. Microarray is geared to copy-number and large structural changes, not the small mutations that cause hemophilia. Direct PCR wins.

PCR

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:

Rothera's test is a bedside-style chemistry test, and the key ingredient to remember is sodium nitroprusside. That reagent is the giveaway for ketone testing.

When you saturate urine with ammonium sulfate, add a pinch of sodium nitroprusside, and layer strong ammonia or alkali on top, acetoacetate and acetone in the sample react to throw up a purple ring at the junction of the two layers. A vivid ring means the urine is loaded with ketone bodies, the situation you see in diabetic ketoacidosis or prolonged starvation. One caveat: the reaction misses beta-hydroxybutyrate, so it can underestimate total ketones.

Match this against the choices. Proteins would be checked with sulfosalicylic acid or heat-and-acetic-acid coagulation. Glucose is the classic target of Benedict's reagent. Fatty acids are not assessed this way at all. The reagent and the colored ring point squarely to ketones.

Ketones

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:

The trick here is to know which liver enzyme keeps a big share of itself inside mitochondria rather than floating in the cytoplasm or sitting on membranes.

AST, the old SGOT, comes in two flavors: one in the cytosol and one in the mitochondria, and the mitochondrial fraction is substantial in the liver. That is why severe, deep hepatocyte damage, such as in alcoholic hepatitis, spills extra AST and pushes the AST-to-ALT ratio above two. ALT, the old SGPT, lives almost entirely in the cytosol, which makes it more liver-specific but firmly non-mitochondrial.

The remaining two are membrane enzymes, not mitochondrial at all. GGT is anchored to canalicular and microsomal membranes and rises with cholestasis and enzyme induction. 5'-nucleotidase is a plasma-membrane enzyme used to confirm that a raised alkaline phosphatase is of hepatic origin. So the only enzyme with a major mitochondrial home is AST.

SGOT (AST)

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:

This is a true-except question, so the goal is to find the one listed action theophylline does not perform. Theophylline is a methylxanthine, and its real toolkit is well defined.

It blocks phosphodiesterase, so cyclic AMP is not broken down and accumulates, relaxing airway smooth muscle. It also antagonizes adenosine receptors, and since adenosine normally constricts bronchi and triggers mediator release, blocking it helps open the airways. A more modern mechanism is its boosting of histone deacetylase activity at low concentrations, which increases histone deacetylation, switches off inflammatory gene transcription, and even restores steroid responsiveness.

Now the odd one out. Stimulating beta-2 receptors is what direct beta-2 agonists such as salbutamol or formoterol do; they dock onto the receptor itself. Theophylline never works that way, it operates on the PDE, adenosine, and HDAC pathways instead. Therefore beta-2 receptor stimulation is the action that does not belong.

Beta-2 receptor stimulation

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:

Rivastigmine raises acetylcholine levels in Alzheimer's disease by blocking cholinesterase, so the logical drug-interaction concern is any agent that fights cholinergic transmission, namely anticholinergic drugs. The question is worded in an awkward double-negative recall style, asking which drug to pair with the odd-one-out marker.

Lining up the antidepressant classes by their antimuscarinic punch is the way to reason through it. Tricyclic antidepressants are notorious for strong anticholinergic effects, dry mouth, constipation and confusion, and they would work against a cholinesterase inhibitor. SSRIs are relatively clean of antimuscarinic activity, reversible inhibitors of monoamine oxidase A (RIMA) act on a different pathway, and the atypical antidepressants are likewise low in anticholinergic load.

This is a faithfully reproduced recall item, and the recorded answer key flags the tricyclic antidepressant as the selected option, so that is retained as the answer for this question.

Tricyclic antidepressant

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:

Ethosuximide belongs to the succinimide class and is famous for a single, very specific use. Its action is to shut down low-threshold T-type calcium channels inside thalamic relay neurons. These channels drive the pacemaker-like 3 Hz spike-wave discharges that define absence epilepsy. Remove the calcium current and the abnormal thalamocortical loop cannot fire, so the brief staring spells of absence seizures stop.

Walking through the choices: tonic clonic seizures are handled by sodium-channel and broad-spectrum agents such as phenytoin or valproate (*A* is wrong). Complex partial seizures classically respond to carbamazepine (*B* is wrong). Myoclonic jerks call for valproate or levetiracetam, and ethosuximide may actually aggravate them (*D* is wrong). That leaves absence seizures, where ethosuximide is the narrow but textbook drug of choice. Valproate is an alternative when other seizure types coexist, but for pure absence, ethosuximide wins on tolerability.

Absence seizures

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

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42. Narrow therapeutic index is seen with:

(A) Desipramine

(B) Lithium

(C) Penicillin

(D) Diazepam

Correct Answer: (B) Lithium

SOLUTION:

The therapeutic index measures how much room there is between a helpful dose and a harmful one. A high index means the drug is forgiving; a low or narrow index means tiny changes in dose can flip a patient from benefit to toxicity. The drugs we worry about most clinically are the narrow-index ones, because they need blood level checks.

Lithium is the standout here. Its useful plasma range is roughly **0.6** to **1.2** mEq/L, and toxicity begins not far above that. Dehydration, salt loss, or interacting drugs can push levels into the dangerous zone quickly, producing tremor, confusion, and seizures. For this reason lithium therapy is built around regular serum monitoring, which is the practical signature of a narrow therapeutic index.

Comparing the alternatives, penicillin is one of the safest drugs in medicine with an enormous margin of safety, diazepam is a benzodiazepine that is hard to fatally overdose on by itself, and desipramine, while toxic in overdose, is not the classic monitoring example. Lithium is the answer.

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:

Stomach acid is made by parietal cells using an enzyme called the proton pump, formally the H^+/K^+ ATPase. Drugs that bind this enzyme and switch it off are called proton pump inhibitors, and they are the most powerful acid suppressors we have. The easy clue is the name ending in -prazole.

Among the options, omeprazole is the original member of this group and fits the -prazole pattern perfectly, so it is the proton pump inhibitor. The other three act elsewhere: ranitidine and lafutidine both block the histamine H_2 receptor on the parietal cell, reducing acid by a different and weaker route, while misoprostol is a prostaglandin analogue that boosts mucosal protection rather than blocking the pump. Only omeprazole targets the pump itself.

$$\boxed{\text{Omeprazole}}$$

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:

Methyldopa lowers blood pressure by acting in the brain. After conversion to alpha-methylnoradrenaline it activates central α_2 receptors, which dampens the sympathetic drive to the heart and vessels. What makes it special is not its potency but its safety record, especially during pregnancy.

Decades of use have shown that methyldopa does not harm fetal development, so obstetricians rely on it as a preferred agent for raised blood pressure in pregnancy, including pregnancy induced hypertension. That is its primary clinical niche today.

The distractors fail because each belongs to a different drug class: Parkinsonism is managed with levodopa, hirsutism with antiandrogens like spironolactone, and resistant hypertension with strong multi-drug regimens rather than a modest central agent. The correct use is hypertension in pregnancy.

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:

The trick in this question is hidden in the word hydantoin. Phenytoin is chemically a hydantoin derivative, so any syndrome named fetal hydantoin syndrome must come from phenytoin exposure in the womb. The link is built into the terminology itself.

Babies affected by this syndrome show a recognizable cluster: an unusual facial shape, underdeveloped tips of the fingers with small nails, slowed growth, a small head, learning difficulties, and occasionally a cleft lip or palate. These effects are thought to arise from reactive epoxide breakdown products of phenytoin damaging fetal tissue.

The other antiepileptics cause their own distinct problems. Valproate and carbamazepine are both tied to neural tube defects such as spina bifida, and phenobarbitone has a broad teratogenic risk, but none of them carry the hydantoin name. Phenytoin is the cause.

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:

This is a negative-stem question, so the right pick is the wrong fact. Let us audit each claim about opioid antagonists.

Naloxone for constipation holds up: when given in forms that stay in or act mainly on the gut, it blocks intestinal opioid receptors and eases opioid induced constipation while sparing central pain control, so statement one is correct. Naltrexone for alcohol dependence is also correct, as it dampens the reward and craving linked to drinking and is licensed for that use. Nalmefene having a longer half life than naloxone is true as well; nalmefene lasts many hours whereas naloxone fades within an hour or two, which is exactly why naloxone often needs repeat dosing in overdose.

That leaves the claim that naloxone is more potent than naltrexone. In reality naltrexone is the more potent, orally active, and longer lasting of the two; naloxone is short acting and used by injection in emergencies. So this statement is the false one and therefore the answer.

Naloxone is more potent than naltrexone

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:

Levodopa only works after it is changed into dopamine inside the brain. The snag is that the converting enzyme, dopa decarboxylase, is everywhere in the body, so a large share of an oral dose becomes dopamine in the bloodstream and never reaches the brain. That wasted peripheral dopamine also triggers nausea and cardiovascular upset.

Carbidopa solves this neatly. It blocks dopa decarboxylase but is unable to cross into the brain, so its enzyme blockade is confined to the periphery. With peripheral conversion shut down, much more intact levodopa crosses the blood brain barrier and is converted to dopamine where it is actually needed. The practical results are a smaller required levodopa dose and far fewer systemic side effects.

The remaining options describe the opposite of reality: efficacy goes up not down, the needed dose falls not rises, and carbidopa never touches central conversion because it stays outside the brain. The intended purpose is to block peripheral decarboxylation.

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:

The stem hunts for the one wrong fact among true ones, so each claim about phenytoin needs checking.

Saturation kinetics is correct: phenytoin's elimination enzymes saturate at normal doses, so the drug switches to zero-order handling and plasma levels can climb sharply after a small dose increase. Teratogenicity is correct too, as phenytoin in pregnancy produces fetal hydantoin syndrome with facial, nail, and growth defects. High protein binding is also right, with roughly **90%** of the drug bound to albumin, which matters when albumin is low because free drug rises.

The remaining statement says phenytoin stimulates insulin secretion. This is the false one. Phenytoin in fact suppresses insulin release and tends to raise blood glucose, so claiming it boosts insulin is exactly backwards. That makes it the answer.

Stimulates insulin secretion

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:

Read the question as a true/false sorting task. Three of the four couplets describe established human teratogens, and one is fabricated.

Start with the drugs that have textbook syndromes. Phenytoin produces fetal hydantoin syndrome, in which orofacial clefting (cleft lip and palate) is a hallmark, so pairing A holds. Sodium valproate is the antiepileptic most feared for spinal dysraphism, so its link with neural tube defects in pairing C is genuine. Warfarin given in early pregnancy yields fetal warfarin syndrome, whose signature is a flattened, underdeveloped nose from nasal cartilage hypoplasia plus chondrodysplasia punctata, validating pairing D.

That leaves zidovudine. This nucleoside reverse transcriptase inhibitor is actually a pregnancy workhorse, given to cut mother-to-child HIV transmission, and it carries no reputation for cranial bone defects. Defects of the skull vault belong to other agents such as methimazole and ACE inhibitors. Hence the zidovudine and skull-defect link is the false statement.

∴ the mismatched option is 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:

Think of migraine prevention as a small toolkit shared with cardiology and epilepsy. The classic prophylactics are beta blockers, certain antiepileptics, tricyclics, and calcium channel blockers.

Propranolol heads the beta-blocker group and is often the first drug a clinician reaches for, so it belongs. Among antiepileptics, both valproate and topiramate carry strong evidence for cutting attack frequency, so they belong too.

Ethosuximide is the odd one out. Its single, narrow indication is the absence seizure, where it silences thalamic T-type calcium channels. It does nothing for the trigeminovascular mechanism of migraine and is never listed as a preventive agent.

So three antiepileptic/beta-blocker drugs are valid migraine preventives and only the absence-seizure drug is not.

Ethosuximide

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

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51. MAO inhibitors should not be used with:

(A) Pethidine

(B) Pentazocine

(C) Buprenorphine

(D) Morphine

Correct Answer: (A) Pethidine

SOLUTION:

The danger here turns on serotonin. An MAOI keeps serotonin from being degraded, so any partner drug that pushes serotonin levels higher can tip the patient into serotonin toxicity.

Most opioids work purely on opioid receptors and leave serotonin handling alone. Pethidine is the exception: along with its analgesic action it actively blocks the

reuptake of serotonin at nerve terminals. Layer that on top of an MAOI and you stack two serotonin-raising mechanisms, producing the well-described excitatory crisis (fever, rigidity, delirium, seizures) or, less often, a depressive crisis with coma and apnoea.

Morphine and buprenorphine lack this reuptake effect, so they are the preferred opioids in a patient on an MAOI. Pentazocine has only minor serotonergic activity, making it a distant second concern, which is why pethidine is graded the single best answer.

Pethidine

Quick Tip: Which opioid also blocks serotonin reuptake?

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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:

Sort each drug by what it does to serotonin signalling. Anything that adds serotonergic tone can trigger the syndrome; anything that blocks serotonin receptors cannot.

Buspirone is an outright 5-HT1A agonist, so it raises serotonin activity and qualifies as a trigger. Meperidine inhibits serotonin reuptake and is one of the most cited culprits, particularly alongside MAOIs. Pentazocine carries minor serotonergic effects and has been implicated as well.

Chlorpromazine moves in the opposite direction. As a phenothiazine it antagonises serotonin (and dopamine) receptors, lowering rather than raising serotonergic transmission. Receptor blockers of this kind are part of the treatment of serotonin syndrome, not a cause of it.

Therefore the one agent that cannot set off the syndrome is the receptor-blocking antipsychotic.

Chlorpromazine

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

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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:

The patient needs an agent that both kills the meningococcus and reaches the cerebrospinal fluid, while sidestepping the penicillin allergy.

The usual choices, penicillin G and ceftriaxone, are off the table or risky: one is the very allergen, the other is a beta-lactam that can cross-react. So look for a non-beta-lactam with proven meningococcal activity and good central nervous system entry. Chloramphenicol fits exactly. It is bactericidal for Neisseria meningitidis, penetrates the inflamed meninges freely, and has long been the recommended substitute when penicillins cannot be used.

The distractors fail for clear reasons. Meropenem still belongs to the beta-lactam family and shares cross-reactivity concerns. Ciprofloxacin's role in this disease is prophylactic eradication of throat carriage, not treatment of active meningitis. Teicoplanin only touches gram-positive bacteria and is useless against a gram-negative diplococcus.

So the safe, effective alternative is the broad-spectrum agent with reliable CSF levels.

Chloramphenicol

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:

Hirsutism is fundamentally an androgen-driven problem, so the trick is to find the agent that lowers androgen action rather than raising it.

Three options push androgens up or mimic them. Danazol is itself a synthetic androgen, so unwanted hair is a predictable side effect. Norethisterone is a 19-nortestosterone progestin that retains androgenic character, again favouring hirsutism and acne. Phenytoin works through a different route but is a classic drug cause of excess hair growth and coarse skin.

Flutamide is the mirror image. It is a pure androgen-receptor antagonist, and clinicians prescribe it to suppress androgen-dependent hair growth. A drug used to treat hirsutism cannot be the one that causes it.

So the exception is the antiandrogen.

Flutamide

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

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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:

Lithium is a mood stabiliser with a single famous fetal hazard, and matching it to the right organ system solves the question.

When lithium is used early in pregnancy, the heart is the target. The textbook lesion is Ebstein anomaly, where the tricuspid valve sits too low and the right ventricle is partly atrialised. Because this is a structural defect of the heart, the umbrella answer is congenital cardiac malformation.

The other choices belong to different drugs. Spinal and cranial neural tube defects point to valproate and carbamazepine. Fetal hydantoin syndrome is the signature of phenytoin. A primary fetal renal malformation is not the recognised lithium effect.

So the heart, specifically the tricuspid valve, is the answer.

Cardiac malformations

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:

Match the antibiotic to its signature toxicity and the monitoring choice follows.

Linezolid, an oxazolidinone reserved for stubborn gram-positive infections, suppresses the bone marrow when given for long courses. The first and most clinically relevant fallout is a dropping platelet count, so weekly full blood counts with attention to platelets are advised once therapy stretches beyond a couple of weeks.

The remaining options describe toxicities of other drugs. Renal impairment and hearing loss flag aminoglycoside and vancomycin therapy, not linezolid. Liver function testing is not the defining safety check for this drug either.

So the parameter that genuinely changes management during linezolid use is the platelet count.

Platelet count

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

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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:

Thalidomide is best remembered for two faces: a notorious teratogen of the past and a useful immunomodulator of the present. We must find the wrong claim.

Begin with the clinical uses. In leprosy, the inflammatory type 2 reaction (ENL) is driven by tumor necrosis factor alpha, and thalidomide brings this cytokine down, so it is a recognised therapy for ENL. That validates option A. Its anti-angiogenic property, blocking new blood vessel growth that tumors need, makes it effective in multiple myeloma for both first-line and relapsed disease, usually paired with steroids. That validates option C.

Now the history. The drug was sold around 1957 as a calming agent and to ease nausea of early pregnancy. When thousands of babies were born with phocomelia, the limb-shortening defect, it was pulled from the market. That validates option B.

Finally the side-effect profile. Typical complaints are drowsiness, constipation, tingling neuropathy and a real risk of clots in the veins, plus its severe damage to the unborn child. It does not characteristically cause loose stools, nor does it

produce a euphoric high. Therefore the statement that its commonest effects are diarrhea and euphoria is simply untrue.

∴ The false statement is the one about diarrhea and euphoria.

Most common side effects are diarrhea and euphoria

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

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58. Pancreatitis occurs with:

(A) Abacavir

(B) Zidovudine

(C) Lamivudine

(D) Didanosine

Correct Answer: (D) Didanosine

SOLUTION:

The trick here is matching each anti-HIV nucleoside drug to the adverse effect it is famous for. Pancreatitis is the target.

Take zidovudine first. Being the oldest NRTI, it hits the bone marrow, leading to low red cells and low neutrophils, and it can also injure muscle. Pancreas is not its problem. Lamivudine is essentially the gentle member of the group with very little toxicity, so it does not fit. Abacavir carries a unique danger: a hypersensitivity syndrome in patients who carry the HLA-B*5701 gene, which can be life threatening but is immunologic, not pancreatic.

That leaves didanosine. This drug is well known for two dose-related toxicities, an inflamed pancreas and a painful peripheral neuropathy. The pancreatitis can be serious, especially when the drug is given alongside other agents that stress

the pancreas. So the matching is clear.

∴ Didanosine is the NRTI that causes pancreatitis.

$$\boxed{\text{Didanosine}}$$

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:

To pick the right drug, identify the molecular driver of the tumor and then the inhibitor that hits it.

GIST grows because of an overactive c-KIT (CD117) receptor, a tyrosine kinase sitting on the pacemaker cells of the gut wall. The drug we want must shut down this kinase. Imatinib was designed against BCR-ABL for chronic myeloid leukemia, but it also blocks c-KIT and PDGFR very effectively, which is exactly why it became the cornerstone treatment for GIST.

The other choices target different kinases. Gefitinib and erlotinib both lock onto the epidermal growth factor receptor and earn their place in lung cancer therapy, not in stromal tumors. Sorafenib is a broad multikinase blocker reserved for kidney and liver cancers. None of these matches the c-KIT pathway that defines

GIST.

∴ Imatinib is the agent of choice for GIST.

Imatinib

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

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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:

Amphotericin B is a powerful antifungal that earns its old nickname amphoterrible because it damages the kidneys, causes chills and fever during the drip, and lowers potassium. We need the option that cuts this harm while keeping the drug working.

The smartest pharmaceutical fix is to repackage the drug. Liposomal and lipid-complex versions wrap amphotericin inside fatty carriers. These carriers steer the drug toward fungal membranes and the body's macrophage system and away from kidney tubules, so the renal injury falls sharply. This lets clinicians even use larger doses with less harm, the ideal trade.

Looking at the other suggestions: mixing it in glucose only concerns infusion preparation and does nothing for organ toxicity. Simply cutting the dose does reduce side effects but at the cost of curing the infection, so it is not a real solution. Adding flucytosine gives a synergistic combination that may permit a

lower amphotericin dose, yet flucytosine brings its own marrow suppression and is chosen for efficacy more than for safety.

∴ Liposomal delivery is the recognised way to lower amphotericin B toxicity.

Using liposomal delivery systems

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:

The key is to know which receptors each kinase inhibitor covers. HER1 is the epidermal growth factor receptor and HER2 is the ErbB2 receptor. We want one drug that silences both.

Lapatinib is the textbook dual inhibitor. It simultaneously blocks the kinase domains of EGFR (HER1) and HER2, which is why it works in HER2-positive breast cancer and is often combined with capecitabine. That fits the question exactly.

Erlotinib and gefitinib are narrower in scope; each clamps onto the EGFR (HER1) kinase alone and shines in lung cancer, but neither touches HER2. Canertinib is indeed a broad pan-ErbB blocker on paper, yet it stayed an experimental

compound and is not an established clinical drug, so it is not the answer the examiner intends among the recognised agents.

∴ Lapatinib is the dual HER1 and HER2 inhibitor.

Lapatinib

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:

Pick the disease whose growth runs through a tyrosine kinase, because that is exactly where these inhibitors strike.

GIST is the clean fit. Its tumor cells are powered by a constantly switched-on c-KIT tyrosine kinase, and the TKI imatinib turns this signal off, making it the recommended treatment. So this matches the drug class perfectly.

The rest do not. Acute myeloid leukemia is handled with old-fashioned cytotoxic agents like cytarabine plus an anthracycline; it is chronic myeloid leukemia, not the acute form, that responds to imatinib. Neurofibromatosis is an inherited tumor-prone syndrome managed by monitoring and surgery rather than kinase blockers. Small cell lung cancer is a chemotherapy and radiotherapy disease, while the EGFR inhibitors belong to the non-small-cell type.

∴ Tyrosine kinase inhibitors are used to treat GIST.

Gastrointestinal stromal tumors (GIST)

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

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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:

We need the incorrect claim about mycophenolate mofetil, so confirm each fact in turn.

Start with mechanism and form. The drug is given as an inactive ester that the body converts into mycophenolic acid, the active moiety, so it truly is a prodrug; option C stands. That active form blocks IMP dehydrogenase, choking purine production in lymphocytes, which makes it a strong immunosuppressant for transplant patients; so option B is also correct. Because it shares an antiproliferative, purine-targeting action with azathioprine, the two are not paired together, both being myelosuppressive; option D holds.

Now the toxicity claim. The real signature side effects of mycophenolate are gut symptoms such as diarrhea and cramping, plus a fall in white cells. The kidney is not its prime victim. Renal toxicity is the hallmark of the calcineurin inhibitors like cyclosporine and tacrolimus, not of mycophenolate. So saying nephrotoxicity is its most common adverse effect is the wrong statement.

∴ The untrue statement concerns nephrotoxicity.

Most common adverse effect is nephrotoxicity

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

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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:

In heparin induced thrombocytopenia the immune system attacks the heparin and platelet factor 4 complex, dropping platelets yet pushing the patient into dangerous clotting. The first move is to stop every form of heparin, then anticoagulate with a drug that has nothing to do with heparin.

Lepirudin fits this need. It is a lab-made version of hirudin that directly blocks thrombin and works independently of antithrombin or heparin, so it neither feeds the antibodies nor needs heparin to act. That is precisely why it is recommended in HIT.

The other agents miss the mark. Abciximab blocks the platelet GP IIb/IIIa receptor and is a tool for coronary intervention, not an anticoagulant for HIT. Warfarin is actually hazardous if started alone during acute HIT because it can trigger limb gangrene by knocking out protein C early; it is introduced only after

platelets recover. Alteplase is a clot-buster for acute stroke or heart attack and does not address the underlying clotting tendency of HIT.

∴ Lepirudin, a direct thrombin inhibitor, is the recommended drug for HIT.

Lepirudin

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

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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:

Trientine is a second-line copper chelator for Wilson disease, kept in reserve for people who develop intolerance or toxicity to penicillamine. Let us test the statement that the question wants us to flag as false.

The claim in option B holds up. Trientine is precisely the recognised substitute when penicillamine cannot be continued, which is why both drugs exist for the same indication.

The claim in option C also holds. Since trientine grabs onto metal ions, it will chelate iron from any oral iron supplement taken too close to it. Clinical advice is to keep a gap of about two hours between the two so each is absorbed properly.

The claim in option D is correct as well. With prolonged use, trientine can remove enough iron to produce iron deficiency anemia, so iron status is monitored.

That leaves option A. Two errors are packed into it. First, trientine is a weaker copper chelator than penicillamine, not a more potent one. Second, its oral bioavailability is poor, so describing it as well absorbed is also incorrect. Both halves of that statement are wrong, making it the exception the question is asking for.

More potent than penicillamine and orally absorbed (option A)

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

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66. Allopurinol is used in the treatment of:

(A) Osteoarthritis

(B) Gout

(C) Rheumatoid arthritis

(D) Ankylosing spondylitis

Correct Answer: (B) Gout

SOLUTION:

The key to this question is to follow the uric acid pathway. Allopurinol is a structural analogue of hypoxanthine and it shuts down xanthine oxidase. Because that enzyme is the final step in making uric acid, the result is a fall in serum urate.

Now ask which of the four diseases is driven by uric acid. Only one is. Gout results from urate crystals settling in joints when blood uric acid runs high. A drug that reduces urate production therefore treats and prevents gout, which is exactly why allopurinol is prescribed for it in the chronic setting.

The remaining choices have nothing to do with uric acid. Osteoarthritis is wear-

and-tear cartilage loss, rheumatoid arthritis is an immune-mediated synovitis managed with disease-modifying agents, and ankylosing spondylitis is an inflammatory spinal arthritis managed with anti-inflammatories and biologics. None would respond to a xanthine oxidase inhibitor.

So the only disease that fits the drug is gout.

Gout (option B)

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

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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:

Topical corticosteroid potency is not just about the molecule; the base it is mixed into matters a great deal. The reason is penetration. Whatever helps the steroid cross the skin barrier raises its effective strength at the same concentration.

Picture how each preparation behaves on skin. An ointment is oily and forms a sealing layer over the surface. This occlusion holds water in the outer skin, softens and hydrates the stratum corneum, and lets far more drug diffuse through. That occlusive boost is why an ointment delivers the strongest effect of the four.

A cream is a water-and-oil emulsion that feels lighter and is only partly occlusive, so it pushes less drug through. Gels and lotions are even lighter, often evaporate

or dry on the skin, and provide the least occlusion, so they give the weakest penetration of all.

Lining them up from strongest to weakest gives ointment, then cream, then the gel and lotion forms. The winner for potency is therefore the ointment.

Ointment (option C)

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

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68. Caspases are involved in:

(A) Cell division

(B) Necrosis

(C) Apoptosis

(D) Inflammation

Correct Answer: (C) Apoptosis

SOLUTION:

Start from the name. Caspases are cysteine proteases that cut their targets specifically after aspartate residues. That single property is the engine of programmed cell death.

In apoptosis a cascade runs in sequence. Upstream initiator caspases switch on in response to death signals, and they in turn activate downstream executioner caspases. These executioners chop up structural proteins, the nuclear lamina, and DNA-repair enzymes, neatly packaging the dying cell into membrane-bound apoptotic bodies that are swept up by phagocytes without leaking contents.

The other answers do not fit. Cell division runs on the cyclin and CDK machinery. Necrosis is messy, passive death from injury and swelling, with no controlled protease cascade. Inflammation is the work of cytokines and immune cells; one

caspase touches that world by processing a cytokine, but the textbook association demanded here is cell suicide.

So the function that defines caspases is apoptosis.

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:

This double question tests both what bradykinin does and which of its actions matters most in inflammation.

Take the list of its effects first. Bradykinin is a powerful vasodilator, it widens the gaps between endothelial cells to raise vascular permeability, and it directly stimulates nerve endings to cause pain. On the airways, though, it does the opposite of relaxation: it constricts bronchial smooth muscle. So any statement crediting it with bronchodilatation is the odd one out, since the true effect is bronchoconstriction.

Now for the role that counts most in acute inflammation. The defining event of

the acute response at the microvasculature is leakage of plasma proteins through a more permeable wall, producing exudate and tissue swelling. Bradykinin is a major driver of this permeability change, and that is the action highlighted as its chief contribution.

The alternatives fall short. Vasodilatation is produced by a long list of mediators, so it is not uniquely bradykinin. Pain is real but secondary to the vascular role. Bronchoconstriction is not what acute inflammation is about. Hence the headline answer is increased vascular permeability.

Increase in vascular permeability (option A)

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

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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:

This is a population-genetics classic. A gene that causes disease in the homozygous state can still spread in a population if carrying just one copy helps survival. Sickle cell trait is the textbook case of this heterozygote advantage.

The selective pressure is falciparum malaria. People with one sickle gene handle the infection better: parasitised red cells deform and sickle, the spleen pulls them out of circulation early, and conditions inside the cell hinder the parasite. The net result is milder malaria and better survival, which keeps the sickle gene common

exactly where malaria is common.

Run through the distractors. G6PD deficiency is its own enzyme disorder; sickle trait does not shield against it. Thalassemia is a separate globin-chain disorder with no protective link. Dengue is caused by a virus and has nothing to do with the red-cell changes of sickle trait.

So the protection conferred is specifically against malaria.

Malaria (option B)

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

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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:

The trick with translocation questions is to anchor on the driver gene. For Burkitt lymphoma that gene is MYC, which sits on chromosome 8. Putting MYC under the control of a strongly active immunoglobulin promoter switches it on permanently and drives the very high proliferation that defines this tumour.

The commonest way this happens is a swap between chromosome 8 and chromosome 14, where the immunoglobulin heavy-chain gene lives. That gives t(8;14), the signature of Burkitt lymphoma.

The remaining choices belong to other malignancies. The cyclin D1

rearrangement t(11;14) marks mantle cell lymphoma. The t(15;17) fusion that joins PML and RARA defines acute promyelocytic leukemia. The BCL2 rearrangement t(14;18) is the marker of follicular lymphoma. None of these is the MYC translocation.

So the correct association for Burkitt lymphoma is t(8;14).

t(8;14) (option A)

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

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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:

The single most informative part of this question is the breakpoint at 8q24, because that band carries the MYC gene. Whenever a B-cell tumour shows a break at 8q24, the disease in play is Burkitt lymphoma, where MYC is forced into permanent overexpression.

Burkitt lymphoma comes with three related rearrangements. The usual one is t(8;14), joining MYC to the immunoglobulin heavy chain. There are also two light-chain variants: t(2;8), which lines MYC up with the kappa light-chain gene on chromosome 2, and t(8;22), which uses the lambda light-chain gene. The translocation given here, t(2;8)(p12;q24), is exactly the kappa variant.

Cross-check the wrong options. The Philadelphia chromosome t(9;22) belongs to

CML. AML carries translocations like t(15;17) and t(8;21). T-cell ALL is driven by T-cell receptor gene rearrangements, not by 8q24. None matches the clue.

The answer therefore is Burkitt lymphoma.

Burkitt lymphoma (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:

Think about what physically happens to a cell as it dies in each pathway. In apoptosis the cell actively pulls itself inward. It loses water and shrinks. Because the volume drops while protein content stays the same, the cytoplasm packs tightly and turns intensely pink on H and E, which we call eosinophilia. The chromatin is squeezed against the inner nuclear membrane, producing the condensed clumped nucleus described as nuclear compaction or pyknosis. The genius of apoptosis is that the membrane is never breached; the dying cell parcels itself into sealed apoptotic bodies so its enzymes never spill out, which is why there is no surrounding inflammation. Now contrast this with necrosis. Here the injured cell can no longer run its Na^+/K^+ ATPase pumps, sodium and water rush in, and the cell balloons up. That ballooning is cellular swelling, and it is a marker of reversible injury and necrosis, the exact opposite of the tight shrinkage of apoptosis. So when the question asks which listed item is not an apoptotic feature, the misfit is cellular swelling. The other three, packed pink cytoplasm,

condensed nucleus, and an unbroken membrane, are all defining apoptotic changes.

Cellular swelling

Quick Tip: Shrinkage defines apoptosis; swelling defines necrosis.

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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:

Start from the cause and work down to the surface proteins. PNH begins as a mutation in the ***PIGA*** gene inside a haematopoietic stem cell. The job of ***PIGA*** is to build the GPI anchor, a greasy tether that pins certain proteins to the outer membrane of the blood cells. When the anchor cannot be assembled, none of the anchor-dependent proteins can stay attached, so they all drop off together. Among these proteins are the two key complement brakes, CD55 (decay accelerating factor) and CD59 (membrane inhibitor of reactive lysis). Their normal role is to stop the complement system from punching holes in the red cell. Strip them away and complement runs unchecked, lysing the red cells inside the vessels and spilling free haemoglobin into the urine, classically at night. Now look at the options. Yes, CD55 and CD59 are individually deficient, but each one is only missing as a downstream consequence of the absent anchor. The choice that captures the true upstream lesion, and therefore both deficiencies in one stroke, is the deficiency of the GPI anchored protein. LFA is simply an adhesion molecule

on lymphocytes and is irrelevant to the complement-mediated lysis of PNH, so it is discarded.

GPI anchored protein

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

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

(A) IgG

(B) IgM

(C) IgA

(D) IgE

Correct Answer: (B) IgM

SOLUTION:

Decode the name first. A lymphoplasmacytic or plasmacytoid lymphoma is made of B lymphocytes caught halfway in their journey to becoming plasma cells, and like plasma cells they pump out a single clone of antibody. The key is to recognise which antibody this particular tumour favours. The famous disease produced by this lymphoma is Waldenstrom macroglobulinemia, and the word macroglobulin is itself a clue: it refers to the bulky pentameric IgM molecule. Because the cells secrete enormous amounts of IgM, the plasma turns thick and sticky, leading to the hyperviscosity picture of headaches, blurred vision, mucosal bleeding, and sluggish circulation. Now weigh the alternatives. IgG is what you expect in classic multiple myeloma, a frank plasma cell malignancy rather than a lymphoma. IgA myeloma is again a plasma cell tumour and not the lymphoplasmacytic type. IgE secreting tumours are vanishingly rare curiosities. So the antibody class that defines plasmacytoid lymphoma, through its link to Waldenstrom disease, is IgM.

IgM

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

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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:

The question is really asking which vegetation is the most likely to fall apart and shower emboli. Picture the building material of each. In infective endocarditis a clump of fibrin and platelets grows around colonies of living bacteria, most aggressively with Staphylococcus aureus. Because microbes keep digesting the surrounding tissue, these masses are bulky, soft, and poorly anchored, so chunks break free readily and lodge in distant organs. That softness is the definition of friability. Compare this to rheumatic heart disease, where the vegetations are tiny firm verrucae lined up neatly along the valve closure line and stuck down tightly, so they almost never embolize. Libman-Sacks endocarditis, which is simply the heart lesion of systemic lupus, throws up small sterile deposits on both faces of the valve cusps; being sterile and organised they lack the crumbly quality of an infected mass. Note also that the SLE option and the Libman-Sacks option point to the same disease, so neither can outrank infection. The clear winner for friability is infective endocarditis.

Infective endocarditis

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

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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:

Trace the path of the offending chemicals. A carcinoid tumour pours serotonin and related amines into the venous blood. This blood returns to the right heart first, so the right-sided endocardium and valves soak in high amine concentrations and react by laying down fibrous plaques. Crucially, the lung then metabolises and inactivates these amines, which is why the left heart escapes harm in most patients. The result is a coating of fibrous, collagen-rich, smooth-muscle plaque on the right ventricular lining, the tricuspid valve, and the pulmonary valve. Functionally this stiffens and tethers the tricuspid valve, producing regurgitation, and narrows the pulmonary outflow, producing pulmonary stenosis. Reading the options through this lens, the best match is the one naming fibrous endocardial thickening of the right ventricle, tricuspid valve, and pulmonary valve. The choice mentioning endometrial thickening is a misprint and clinically meaningless, and severe tricuspid stenosis is not the rule. The option stressing elastic deposits is wrong because the plaques are notably deficient in elastic tissue, and the calcification option describes degenerative valve disease, not carcinoid plaque.

Fibrous endocardial thickening of RV, tricuspid and pulmonary valve

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:

The histology line is doing all the work here, so anchor on the two buzzwords. First, fibrosis that is heterogeneous and patchy means the scarring varies from place to place and from old to new, with damaged lung sitting right beside preserved lung. Second, fibroblastic foci are little nests of actively proliferating fibroblasts that mark ongoing injury. Put these together and you have the textbook signature of usual interstitial pneumonia, the tissue pattern underlying idiopathic pulmonary fibrosis, which classically presents with relentless progressive breathlessness. Now sweep the distractors. Non-specific interstitial pneumonia is the opposite in character: its fibrosis is uniform and time-synchronous across the lung and it pointedly lacks fibroblastic foci, so it cannot be the answer. Cryptogenic organizing pneumonia fills the distal airspaces with granulation-tissue plugs called Masson bodies rather than scarring the interstitium. Desquamative interstitial pneumonia, tied to smoking, packs the alveolar spaces with pigment-laden macrophages and again lacks the patchy heterogeneity. The only pattern that fits heterogeneous fibrosis plus fibroblastic foci is UIP.

Usual interstitial pneumonia

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:

This is a matching exercise between renal cell carcinoma subtypes and their chromosome fingerprints, so build the map and then read off the answer. The clear cell type, by far the most common, owes its biology to the VHL gene that sits on the short arm of chromosome 3; accordingly it carries VHL mutations and loss of 3p material. The papillary type is the one defined by extra chromosomes, classically gains of chromosomes 7 and 17 along with loss of the Y chromosome. The chromophobe type behaves differently again: instead of gaining material it loses whole chromosomes, ending up hypodiploid, with the textbook losses being monosomy of chromosome 1 and of the Y chromosome among several others. Lining the options against this map, VHL mutations and 3p deletions are clear cell features, and trisomy 7 and 17 is the papillary signature, so both are wrong for chromophobe. The fingerprint that belongs to chromophobe carcinoma is the monosomy of chromosomes 1 and Y.

Monosomy of 1 and Y

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

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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:

Sort the four vasculitides into granuloma-forming and non-granuloma-forming groups, then pick the one that does not fit. A granuloma is a tight cluster of activated epithelioid macrophages, frequently joined by multinucleate giant cells. Three of the listed diseases form them. Wegener's granulomatosis, now called granulomatosis with polyangiitis, is named for the necrotising granulomas it builds in the nose, lungs, and kidneys. Takayasu arteritis attacks the aorta and its main branches with granulomatous inflammation of the vessel wall. Giant cell arteritis, true to its name, studs the temporal and other large arteries with giant cells inside granulomas. That leaves classic polyarteritis nodosa, a disease of medium-sized muscular arteries whose hallmark is transmural fibrinoid necrosis with a mixed acute and chronic infiltrate, but pointedly without granuloma formation. Because PAN is the only member of the group that does not produce granulomas, it is the exception the question is hunting for.

Polyarteritis nodosa (classic PAN)

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

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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:

Think about what each test contributes in a glomerular disease. Light microscopy shows overall architecture, immunofluorescence shows antibody and complement deposits, and electron microscopy shows the fine structure of the basement membrane and any electron-dense deposits.

Alport's syndrome is a genetic disorder of type IV collagen (alpha-5 chain). The basement membrane is built abnormally, so its layered structure is disturbed. Under EM the glomerular basement membrane looks alternately thin and thick, with the lamina densa split into several layers. This is described as a basket-weave or lamellated pattern, and it is the hallmark that confirms the diagnosis.

The other choices do not depend on EM. Goodpasture's syndrome shows smooth linear staining for IgG along the basement membrane on immunofluorescence, which is its signature. Wegener's granulomatosis and Churg-Strauss syndrome are small-vessel vasculitides identified by ANCA testing and by their light-microscopy picture of necrotizing inflammation, granulomas, or eosinophilia, with little immune deposit on the kidney.

So among the listed conditions, the one where the diagnosis truly rests on the electron microscope is the hereditary collagen defect.

Alport's syndrome

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

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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:

A paraganglioma comes from neuroendocrine tissue, the same cell line that makes and releases catecholamines. The key question is what such a cell looks like at very high magnification.

Hormone-secreting neuroendocrine cells package their product into small round vesicles bounded by a membrane. The core of each vesicle is packed with electron-opaque material, so it appears dark and is named a dense-core granule. When a pathologist sees plentiful dense-core granules on EM, it confirms the tumour is neuroendocrine in nature, and a paraganglioma fits this picture exactly.

Now rule out the distractors. Mitochondrial shrinkage is a degenerative or artefactual change and points to no specific diagnosis. A prominent Golgi simply means active secretion in general and is not unique to paraganglioma. Counting mitoses is a light-microscopy proliferation index and tells you nothing about ultrastructure or cell origin.

The single ultrastructural detail that brands a tumour as a paraganglioma is therefore the secretory granule with a dense centre.

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:

The disease is defined by the cell that drives it, the Langerhans cell, a special dendritic cell of the skin and other tissues. To prove a lesion is Langerhans cell histiocytosis, the pathologist looks for the markers unique to that cell.

The defining surface molecule of the Langerhans cell is CD1a. Alongside it, S-100 protein is positive and langerin (CD207) is positive, and the electron microscope shows the tennis-racket shaped Birbeck granule. Of the four antigens offered, only CD1a belongs to this cell.

Working through the rest helps confirm the choice. CD10 is tied to lymphoid precursors and follicular-centre B cells. CD30 lights up the malignant cells of Hodgkin lymphoma and anaplastic large cell lymphoma. CD56 belongs to NK cells and to neuroendocrine tissue. Not one of these three is a dendritic-cell marker.

So the antigen that signals a Langerhans cell origin, and therefore LCH, is clear.

$$\boxed{\text{CD1a}}$$

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

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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:

Clotting reflects a balance between procoagulant and anticoagulant forces. Nephrotic syndrome disturbs that balance because the leaky glomerulus filters out plasma proteins by size.

Antithrombin III is roughly the size of albumin, so it passes into the urine and its blood level drops. Since antithrombin III normally inhibits thrombin and other clotting factors, losing it releases the brakes on coagulation. The body responds with extra hepatic output of large proteins like fibrinogen and with more reactive platelets, pushing the patient further into a clot-prone state, classically seen as renal vein thrombosis.

The wrong options each contradict the physiology. Fibrinogen actually rises, not falls. Vitamin K handling is not the central problem here. Protein C is itself an anticoagulant, so more of it would protect against clots rather than cause them.

The single best explanation for the prothrombotic tendency in nephrotic syndrome is the urinary depletion of the body's natural thrombin inhibitor.

Loss of antithrombin III

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

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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:

GIST grows from the interstitial cells of Cajal, the natural pacemakers of gut motility. The tumour is driven in most patients by a gain-of-function mutation in c-KIT, a gene for a tyrosine kinase receptor.

Because that receptor protein is strongly expressed, an antibody against it, named CD117, stains almost every GIST. This makes CD117 the signature marker and explains why these tumours can be treated with the kinase inhibitor imatinib.

The other antibodies are not specific. CD34 is often positive in GIST too, but it also appears in vascular tumours and solitary fibrous tumour, so it cannot confirm the diagnosis by itself. CD23 belongs to follicular dendritic cells and chronic lymphocytic leukemia. S-100 points to schwannoma, neurofibroma, or melanoma. Only one antibody links directly to the c-KIT receptor.

So the marker that reliably identifies a gastrointestinal stromal tumour is the one against the KIT receptor.

$$\boxed{\text{CD117}}$$

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

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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:

Down's syndrome means three copies of chromosome 21. To answer this, rank the ways an extra chromosome 21 can arise by how often each happens.

The dominant mechanism, in roughly 19 of every 20 patients, is nondisjunction during meiosis. When chromosome 21 fails to separate, one gamete carries two copies and, after fertilization, the child ends up with three. Crucially, this error happens chiefly in the maternal egg, especially in the first meiotic division, and becomes more likely as the mother gets older. That is why maternal nondisjunction tops the list.

The remaining causes are minor. Paternal nondisjunction is uncommon. A Robertsonian translocation contributes only a small share but matters for genetic counselling because it can run in families. Mosaicism, where only some cells are trisomic, is rarest of all.

Putting the numbers together, the leading cause of trisomy 21 is failure of chromosome separation on the mother's side.

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:

Start by fixing the parents' genotypes. Cystic fibrosis needs two mutant alleles to show. Both parents look healthy, yet they have an affected child who must be homozygous for the mutation. That child got one mutant allele from each parent, which proves both parents are silent carriers.

Write the carrier as Aa, where A is the working allele and a is the faulty one. When two carriers have a child, simple Mendelian counting gives the genotypes AA, Aa, Aa, aa in equal quarters. Only the aa child shows the disease.

That single aa outcome out of four equally likely combinations gives a risk of one in four for any pregnancy. Genetics has no memory, so the previously affected daughter does not raise or lower the odds for the next baby; the chance stays the same each time.

Therefore the chance that the next child is affected is one quarter.

$$\boxed{\frac{1}{4}}$$

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:

To find male predominance, look at how the sexes differ in chromosome dosage. A man carries a single X and a Y, so for any gene on the X he has just one copy. A woman carries two X chromosomes, so she has a backup copy of every X-linked gene.

In an X-linked recessive disorder this asymmetry matters a lot. One faulty allele on a man's single X is sufficient to cause disease, since there is no partner X to cover for it. A woman would need the same fault on both of her X chromosomes to be affected, which is uncommon. The result is that males are affected much more frequently. Classic examples are haemophilia, Duchenne muscular dystrophy, and red-green colour blindness.

The other modes do not favour males. Autosomal dominant and recessive genes lie on non-sex chromosomes, so men and women are hit about equally. X-linked dominant disease leans towards females, and in several such conditions affected males die before or soon after birth.

So the inheritance pattern in which men predominate is the X-linked recessive type.

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:

El Tor is one biotype of ***Vibrio cholerae***, the cause of epidemic cholera, and this item wants the single wrong statement.

Reservoir: Cholera is a purely human disease. There is no maintained animal host, so people are the only reservoir. Statement A matches the facts.

Environmental survival: Cholera vibrios persist in clean cold water for weeks. Survival in ice cold water for 2 to 4 weeks is well described and supports its spread through contaminated water supplies, so statement B is correct.

Heat killing: These organisms are not heat resistant. A brief boil, about half a minute, kills them, which is the basis of advising boiled drinking water during outbreaks. Statement C is correct.

Toxin target: Cholera toxin works only at the gut. Its B subunit attaches to the GM1 ganglioside on enterocytes, then the A subunit drives a rise in cyclic AMP and a flood of fluid into the lumen, producing rice water stools. It has no direct action on tissues outside the intestinal lining. Therefore the statement that the toxin acts directly on other tissues is wrong.

The false option is D.

Enterotoxin acting on tissues other than intestinal epithelium

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

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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:

This asks which part of an MHC class I molecule actually holds the peptide shown to CD8 T cells.

Build of class I: It is one heavy alpha chain folded into three domains called alpha1, alpha2 and alpha3, paired with a small invariant partner, beta2 microglobulin.

Where the peptide sits: The cleft that grips the antigenic peptide is made by alpha1 together with alpha2. In space these two domains lie at the top of the molecule, away from the membrane, so they are the distal domains of the alpha chain. Both come from the same alpha subunit.

Role of the rest: The alpha3 domain is close to the membrane and is the docking point for CD8. Beta2 microglobulin is structural support and never lines the groove.

Checking options: A single proximal domain is alpha3, which is a CD8 contact,

not a peptide holder, so A is out. Any answer that pairs alpha with beta domains describes class II, where the groove is shared between an alpha and a beta chain, so C and D do not apply to class I. That leaves the distal domains of the alpha chain.

Distal domains of the alpha subunit

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

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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:

We need the one wrong line about ***Staphylococcus aureus***.

Who carries it: The bug lives quietly in the front of the nose and on skin. About a third of healthy individuals are persistent or intermittent nasal carriers. That makes statement B correct.

Origin of disease: Since carriage is so common, the usual source of a staph infection is the person's own resident flora, an endogenous source. Spread from another infected patient does happen, especially in hospitals, but it is not the most common source overall. So calling cross infection from infected people the commonest source is the inaccurate statement.

Superantigen toxins: TSST 1 is the textbook superantigen, and the epidermolytic (exfoliative) toxins linked to scalded skin syndrome are also counted among the superantigens. Statement C stands.

Basis of methicillin resistance: MRSA owes its resistance to mecA, which sits on the chromosomal SCCmec cassette and encodes an altered penicillin binding protein, PBP2a. That is chromosomal, so statement D is right.

The exception is the claim about cross infection being the leading source.

Most common source is cross infection from infected people

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

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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:

A septic infant with this culture pattern is a Group B streptococcal case. The reconstructed traits, beta hemolysis, bacitracin resistance, and a positive CAMP reaction, line up only with ***Streptococcus agalactiae***.

Hemolysis: Clear beta hemolysis tells us we are dealing with a pyogenic Streptococcus, leaving Group A and Group B in play.

Bacitracin: The cheap bedside differentiator. Group A is killed by bacitracin, Group B is not. A resistant isolate is Group B.

CAMP test: Group B Streptococcus secretes the CAMP factor, which synergizes with the staphylococcal beta lysin to widen hemolysis into an arrowhead. A positive CAMP test seals the identification as Group B.

Clinical setting: Group B Streptococcus is the classic agent of neonatal early onset sepsis and meningitis, so a septic infant is exactly the expected host.

Eliminating the rest: S. pyogenes is bacitracin sensitive and CAMP negative. S. pneumoniae gives green alpha hemolysis and is optochin sensitive. Enterococcus grows in bile and 6.5 percent salt but is not CAMP positive. None fits the full profile.

Streptococcus agalactiae (Group B Streptococcus)

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

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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:

The trick here is the precise meaning of isolation, which means growing the organism, not merely finding its parts.

Biology of Chlamydia: These are obligate intracellular bacteria. They have an energy parasite lifestyle and cannot multiply outside a host cell, so artificial agar will not grow them. To isolate them you must provide living cells.

Available living systems: The two traditional ways to culture Chlamydia are inoculation into the yolk sac of an embryonated egg and inoculation into susceptible cell lines like McCoy cells. Of the choices given, only yolk sac inoculation is such a growth method.

Why detection tests do not count: An ELISA picks up chlamydial antigen, a DFA stains elementary bodies with a labeled antibody on a slide, and a PCR amplifies chlamydial DNA. All three confirm presence but none recovers a viable organism, so none qualifies as isolation.

Therefore the correct isolation method is yolk sac inoculation.

Yolk sac inoculation

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

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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:

This is a straight recall of where chickenpox virus hides between episodes.

Family habit: VZV belongs to the herpesviruses, a group famous for going latent for life after the first infection. The question is simply where VZV chooses to hide.

Path of the virus: Chickenpox is the first encounter. As it clears, virus particles move backward up the sensory nerves and settle in the neuron cell bodies of the sensory ganglia, the dorsal root ganglia and cranial counterparts like the trigeminal ganglion. The virus is neurotropic.

What reactivation looks like: Years later, with falling cell mediated immunity, the virus wakes up, runs back down a single sensory nerve, and erupts as a painful band of vesicles in one dermatome, that is shingles. Trigeminal involvement gives ophthalmic herpes zoster.

Ruling out lymphoid sites: B cells are the latent home of EBV, while CD4 T cells and macrophages harbor HIV. VZV does not reside in these immune cells, so options B, C and D are wrong.

Trigeminal (sensory) ganglion

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

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

(A) Chlamydia

(B) Herpes

(C) Syphilis

(D) Candida

Correct Answer: (B) Herpes

SOLUTION:

HIV patients lose cell mediated immunity, and that decides which genital problem shows up most.

Effect of immunosuppression: With CD4 counts falling, the body cannot keep latent viruses in check. Herpes simplex virus, which normally sleeps in the sacral ganglia, reactivates often and produces recurrent and frequently severe genital ulcers. This makes genital herpes the leading genital lesion in HIV.

The reinforcing loop: Genital herpes ulcers also raise the chance of catching or passing on HIV because the open sores breach the mucosa, while HIV makes the herpes worse. The two diseases travel together, which is why herpes is the expected genital lesion.

Why not the others: Chlamydial infection mainly gives urethritis and cervicitis rather than an obvious lesion. Syphilis can ulcerate and behaves more aggressively in HIV, yet it is less frequent than recurring herpes. Candida produces vaginitis or balanitis, which is an inflammatory infection rather than the classic genital ulcer the question is after.

Herpes

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

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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:

A diabetic with a destructive nose and orbit infection screams invasive mould disease, and the laboratory note is the tie breaker.

Setting up the differential: Rhino orbital invasive fungal infection in a diabetic is caused either by the Mucorales group, which includes ***Mucor*** and ***Rhizopus***, or by ***Aspergillus***. Clinically they overlap, so we lean on the microscopy.

Reading the hyphae: The report says branching septate hyphae. Septate means the filaments have regular cross walls. Aspergillus is the mould that shows uniform septate hyphae branching at sharp acute angles around 45 degrees. So septate equals Aspergillus.

Excluding the zygomycetes: Mucor and Rhizopus are aseptate or only rarely septate, with broad ribbon like hyphae that branch widely at right angles. They cannot be the answer when the slide clearly shows septate hyphae.

Excluding the yeast: Candida is not a filamentous mould of this kind. It makes budding yeast cells and pseudohyphae, not true branching septate hyphae, so it is ruled out.

The septate description fits only Aspergillus.

Aspergillus

Quick Tip: Septate branching hyphae mean Aspergillus; broad aseptate hyphae would

mean Mucor or Rhizopus.

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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:

Penicillin binding proteins are the molecular targets of beta-lactam drugs, and this item wants the one claim that does not hold. Think about where these proteins actually live. They are membrane-bound enzymes anchored in the cytoplasmic membrane, with their active sites turned toward the growing peptidoglycan, not parked on the outside surface of the finished cell wall. That single fact makes statement A incorrect, so A is what we pick. Now check the rest. Statement B is sound because PBPs run the last assembly steps of the murein sacculus; if they stop working, the wall cannot be sealed and the organism bursts under its own turgor. Statement C is also correct, since these proteins behave as transpeptidases that stitch adjacent glycan strands together and as carboxypeptidases that clip off the terminal D-alanine, and the beta-lactam ring imitates that D-Ala-D-Ala end to jam the enzyme. Statement D is true as well, because methicillin-resistant Staphylococcus aureus expresses PBP2a from the mecA gene, a variant with poor drug affinity that lets wall synthesis continue despite the antibiotic. Everything except the location claim is accurate. A is the false option.

PBPs are localized on the outer face of cell wall

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

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98. Which of the following may cause biliary obstruction?

(A) Ancylostoma

(B) Enterobius

(C) Strongyloides

(D) Clonorchis

Correct Answer: (D) Clonorchis

SOLUTION:

To answer this, match each worm to the organ it occupies and decide which one can plug the bile drainage. The key idea is that only a fluke that physically dwells in the bile ducts can obstruct them. Among the four choices, Clonorchis sinensis is the Chinese liver fluke, and adult worms reside inside the intrahepatic biliary radicles. Their bodies and eggs trigger duct wall thickening, periductal fibrosis and mechanical blockage, and long-standing infection is a known driver of bile duct cancer. That fits the question perfectly. The other three are gut-dwellers with no biliary home. Hookworm (Ancylostoma) clamps onto the duodenal and jejunal mucosa and bleeds the patient into anaemia. Pinworm (Enterobius) settles in the large bowel and causes nocturnal anal itch via perianal egg laying. Strongyloides occupies the upper small intestine, with a self-sustaining autoinfective cycle and risk of disseminated hyperinfection, again nowhere near the bile ducts. So the single parasite that explains biliary obstruction is the liver fluke. ***D*** is the answer.

Clonorchis

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

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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:

The way to crack this is to lock onto the morphology described and let the allergy history confirm it. We are told the cultured material shows numerous hyphae that split into two equal arms at a sharp angle near 45 degrees. That branching pattern, paired with septate filaments, is the textbook signature of Aspergillus species. Layer on the background of asthma, prior allergy and nasal polyps with inspissated secretions, and the whole story reads as allergic fungal rhinosinusitis, a hypersensitivity-driven condition for which Aspergillus is the prototypical culprit. So the pick is option B. The remaining fungi do not fit the picture. Rhizopus and Mucor both belong to the Mucorales and produce wide, ribbon-shaped, aseptate hyphae that branch at near right angles, and they tend to cause angioinvasive mucormycosis in diabetic or immunocompromised hosts rather than allergic polyposis. Candida is a yeast that forms buds and pseudohyphae and does not display the orderly acute-angle dichotomous branching this stem stresses. The angle of branching and the allergic context both converge on the same organism. ***B*** is correct.

Aspergillus

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:

This is about the order of events when a moving car hits a standing person. The very first contact is what forensic medicine calls the primary impact, and to locate it you simply ask which part of the vehicle reaches the body first. That part is the front bumper, and a bumper sits at roughly leg height for an upright adult. So the primary impact injury, the classic bumper fracture, lands on the legs, typically the lower leg around the level of the bumper. That makes option C the right choice. The trunk and head injuries come later in the sequence. Once struck low, the body rotates and is scooped up onto the bonnet, slamming the chest, abdomen and head against the car, and these are termed secondary impact injuries. A further set, the secondary injuries, occurs when the victim is finally flung onto the road surface. Because chest, abdomen and head all belong to those later phases, they cannot be the primary impact site. The bumper-to-leg contact is what defines the answer. *C* is correct.

$$\boxed{\text{Legs}}$$

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:

Start by separating two different kinds of law. The penal sections describe offences and their punishments, while the procedural code says who may investigate and order a post-mortem. Since the question asks specifically which section lets a magistrate authorise the autopsy, the answer has to be a procedural provision, not a penal one. That immediately rules out the two IPC choices. Section 302 IPC is merely the punishment for murder and Section 304 IPC covers culpable homicide (with 304B being the dowry death offence); neither grants any authority to order an examination of the body. Now compare the two CrPC sections. Section 174 is the police inquest, where the investigating officer looks into an unnatural death and forwards the body for autopsy, so it is a police function rather than a magistrate's. Section 176, by contrast, gives a magistrate the power to conduct an inquiry into the cause of death and to direct post-mortem and even exhumation, and it is the section invoked when a married woman dies in suspicious circumstances within seven years of marriage. That is exactly this fact pattern, so the magistrate acts under Section 176 CrPC. ***B*** is correct.

Section 176 CrPC

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

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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:

The trick here is to read the symptom list as a single syndrome rather than a set of loose findings. We have a worker with heavy salivation, fine shaking, altered behaviour, sleeplessness and poor appetite. That combination is the recognised chronic mercury picture, built on three pillars: a characteristic tremor, the psychiatric state called erethism (shyness, irritability, insomnia, memory and personality change) and oral disease with excess salivation. A gum line can appear too. All of that maps onto mercury, so option A wins. The distractors each carry a more typical fingerprint that does not match. Lead is famous for the Burton blue line on the gums, yet its real story is abdominal colic, wrist drop, anaemia with basophilic stippling and encephalopathy, without the salivation-erethism combination. Arsenic favours gastrointestinal upset with rice-water stools, skin changes such as raindrop pigmentation and hyperkeratosis, plus Mees lines on the nails. Phosphorus tends to produce garlicky breath, vomiting and progressive liver injury. None of these reproduce the salivation, tremor and personality triad of the stem. A is correct.

$$\boxed{\text{Mercury}}$$

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

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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:

Decode the question by what each special bullet is built to do, then match it to the phrase visible mark along its path. The only round designed to be seen in flight is the tracer. It packs a small pyrotechnic compound in its base that ignites on firing and burns as the projectile flies, drawing a bright luminous streak through the air so the marksman can watch the trajectory and adjust fire. That is precisely the visible-track description, so option B is the answer. The other three serve completely different purposes. A tandem bullet describes the situation where one bullet stuck in the bore is driven out just ahead of the next shot, a barrel-obstruction phenomenon with no glowing trail. A dum dum is an expanding soft-point round meant to deform and create a larger wound channel on impact, again nothing to do with flight visibility. An incendiary bullet holds a fire-starting agent that flares when it strikes the target, so its effect appears at impact rather than as a steady streak across the sky. Since only the tracer is engineered to leave a seen path, the choice is clear. $\boldsymbol{B}$ is correct.

$$\boxed{\text{Tracer bullet}}$$

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:

This is an except question, so list the genuine ways lightning hurts a person and then spot the odd description left over. There are three well-described mechanisms. The first is electrical: the enormous current can flow through the victim and stop the heart, fry nerves and produce burns, which validates the direct-current option. The second is thermal: the bolt heats the air in its path to extreme temperatures, and that superheated air scorches and burns nearby tissue, which validates the superheated-air option. The third is mechanical: the sudden, violent heating makes the air expand explosively and then rush outward, the same expansion we hear as thunder, and this expanding, repelled air can hurl and injure the person, which validates that option too. What remains is the idea of a slug of compressed air being driven ahead of the current, and that is not how lightning blast injury is understood; the damage comes from rapid expansion and outward repulsion of air, not a pushed-forward compressed column. So the statement that does not belong is the compressed-air one. *D* is the exception.

Compressed air pushed in front of the current

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:

Aconite is a fast-acting cardiotoxic plant poison whose toxin, aconitine, jams the sodium channels of nerve and heart muscle in the open state. Because the channels stay open, nerves fire abnormally and the heart loses its normal rhythm.

Look at each choice. Hypersalivation happens early because the poison irritates the mouth and stimulates secretions. Tingling and numbness around the lips, tongue and limbs are the hallmark sensory complaint and appear within minutes. Chest pain occurs as the heart muscle becomes ischaemic and arrhythmic. All three are genuine signs.

Blood pressure in aconite poisoning typically *falls*. The patient becomes bradycardic, hypotensive and may collapse from a cardiac arrhythmia such as ventricular tachycardia. A rise in blood pressure does not fit the picture, so that is the odd one out.

Increased blood pressure

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:

The aim of narcoanalysis is to sedate the subject just enough that judgement and inhibition drop but speech remains possible, the so-called truth serum effect. For this you need an agent that produces a quick twilight state.

Scopolamine, also written hyoscine, is the agent in the list that fits. It is an antimuscarinic that crosses into the brain and causes drowsiness, amnesia and a dreamy disinhibited state, which is exactly why it became the original truth serum.

Now eliminate the rest. Atropine is also antimuscarinic but is used clinically to raise heart rate and reverse organophosphate poisoning, not for interrogation. Phenobarbitone is a barbiturate, but it is long acting and is prescribed for seizures; the barbiturates used in narcoanalysis are the short-acting thiopentone and amytal, neither of which is listed. Pethidine is simply a painkiller of the opioid class.

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:

Health behaviour is often broken into knowledge, attitude and practice. To answer, ask which of these is the one a person actually performs and which therefore needs a push to happen.

Knowledge is information held in the mind and does not by itself move a person. Belief is a conviction, again an internal idea. Attitude is the emotional leaning toward or against something; it has feeling attached but is still a disposition rather than an act.

Practice is the doing. Translating what you know and feel into a real action is where motivation matters most, because people frequently know the right thing yet fail to do it without a reason to act. So practice is the component most strongly shaped by motivation while also being tied to emotional drive.

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:

The ASHA scheme came in with the National Rural Health Mission to give every village its own health link worker. The key point is that she is a woman chosen from within the village itself, so her workplace is the village, not a clinic building.

The standard ratio is roughly one ASHA for each 1000 people in rural areas. She does home visits, promotes institutional delivery, immunisation and family planning, and guides people to the nearest facility.

The remaining choices are all formal health centres of the rural infrastructure. The sub-centre has an ANM, the PHC has a doctor, and the CHC is a referral hospital with beds. None of these is where the ASHA is posted, because her role is to stay in the community.

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:

Read the stem literally: it wants the word for shifting from one rung of the socioeconomic ladder to another. That is the definition of social mobility, so match the term to the concept.

Social mobility covers anyone changing position in society, whether they climb up or slide down, and whether the change is within a lifetime or between generations of a family.

The distractors describe different ideas. Social equality is about everyone being on the same level, not moving between levels. Social upliftment is an informal term for helping the poor rise and is not the standard answer. Social insurance is a way of funding healthcare and income support by sharing risk, which has nothing to do with movement between classes.

Social mobility

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

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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:

Break the abbreviation by its Hindi roots. Janani points to the mother and Suraksha points to protection, so JSY is about protecting mothers. That alone selects Janani Suraksha Yojana.

This programme was rolled out in 2005 within the National Rural Health Mission to cut down deaths during childbirth. It pays a cash incentive to poor expectant mothers who choose to deliver in a hospital or health centre instead of at home, with the amount differing for low-performing and high-performing states.

The remaining options simply spell out plausible sounding Hindi phrases that are not the actual title of any maternal scheme, so they can be set aside.

Janani Suraksha Yojana

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

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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:

The phrase primary health care is most strongly tied to one landmark event, so the task is to recall who first set out the concept on a world stage.

That event was the 1978 Alma-Ata conference jointly held by the WHO and UNICEF. It produced the Alma-Ata Declaration, which both defined primary

health care and gave the world the target of Health for All by the year 2000. It also listed the core elements like maternal and child health, immunisation, safe water and essential drugs.

The Indian committees in the options have their own roles. Bhore in 1946 shaped the rural health service tiers, and Shrivastava in 1975 pushed for village health workers. National Health Policy statements build on the Alma-Ata idea rather than create it. So none of them is the origin of the concept.

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:

Picture community participation as a staircase. At the bottom people simply go along with what experts decide; at the top people are in charge of decisions themselves. The correct answer is the choice that puts the most control in the hands of the community.

Compare the options on that scale. Cooperating with workers is bare minimum involvement, since the community is only assisting. Donating resources is a step up but still a supporting role. Designing an intervention around assessed needs is better because the community is considered, yet outsiders usually run the

assessment and the planning.

The strongest form is when the community does the planning of the intervention itself. Here the people set the agenda and direct the work, which is true empowerment and the peak of the participation ladder.

Planning of intervention by the community

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:

Maternal mortality is one of the most sensitive indicators of the health of mothers and of the obstetric services in a community. To read the formula correctly, separate it into top and bottom. The top counts women who die because of pregnancy related causes, including any death up to 42 days after the pregnancy ends. The bottom is the count of live births during the same time window, and the result is scaled, classically per 100,000 live births.

Now walk through the choices. Saying the numerator captures deaths within 42 days of delivery is correct, since the 42 day puerperal window is exactly how a maternal death is defined. Calling it loosely a rate is the conventional usage even though it is mathematically a ratio, so that option is not the trick answer here.

Expressing it per 100 reflects the older scaling that was once used.

The statement that breaks the rule is the one about the bottom of the fraction. The denominator is restricted to live births. Throwing still births and abortions into the denominator is simply not part of the definition, so that claim is the false one.

Denominator includes still births and abortions

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:

Think of the perinatal window as a bridge that starts in late pregnancy and ends one week after birth. It begins at 28 weeks of gestation and closes at the end of the seventh day of life. Because it straddles birth, it has to pull in events from both sides of delivery: babies who die in the womb late in pregnancy and are born dead (still births) and babies who are born alive but die in the first week.

So the correct numerator is still births added to early neonatal deaths, and the whole thing is reported per 1,000 total births.

Screening the options against this picture: pairing still borns with deaths in the first 7 days fits perfectly. Counting neonatal deaths up to 30 days stretches into the neonatal mortality definition instead. Pulling in abortions is wrong because

they occur before the 28 week boundary. Counting only deaths from day 7 to day 28 describes the late neonatal slice, not the perinatal one.

Still borns and deaths within 7 days of birth

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

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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:

The task is to find the disease that is fairly evenly spread across the countryside and the cities, while the rest cluster more in one setting. Use exposure logic. Lung cancer rides on tobacco, traffic fumes and factory smoke, all of which are denser in towns, so it leans urban. Chronic bronchitis follows the same polluted air and smoking trail, again tilting urban. Mental illness shows real environmental patterning, with the pressures of city life shifting the spectrum of disorders, so it too differs by place.

Tuberculosis behaves differently. It thrives wherever there is crowding, undernutrition and poverty, and those conditions are not the exclusive property of either rural or urban India. As a result its burden is broadly shared and does not split neatly along the rural urban line, making it the odd one out.

Tuberculosis

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:

Anchor the answer in the Alma-Ata list of 1978, which spells out eight building blocks of primary health care. They cover health education, control of locally common diseases, immunization, maternal and child health with family planning, supply of essential drugs, nutrition, treatment of common ailments and injuries, and clean water with sanitation.

Now scan the choices. Supplying essential drugs sits squarely on that list. Immunizing against major infections is on it too. Health education actually heads the list. Each of these is therefore a genuine component.

Cost effectiveness is a different kind of idea. It describes a quality we want services to have, not a defined element of the package. Because it is not one of the eight named components, it is the statement that does not belong.

Cost effectiveness

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:

Picture malaria coming back after it had been pushed down. The forces behind such a comeback are partly programme failures and partly biology. On the biological side, the parasite can learn to survive the drugs we give, and the mosquito can learn to survive the insecticides we spray. The parasite can also change its surface antigens to slip past the immune system, keeping transmission alive.

So drug resistance arising in the parasite is a genuine driver, insecticide resistance in the mosquito vector is a genuine driver, and antigenic variation in the parasite is a genuine driver.

The phrase that does not make sense is resistance to antimalarial drugs developing in the human host. People do not become drug resistant; it is the parasite that becomes drug resistant and the vector that becomes insecticide resistant. That mislabelled option is therefore the exception.

Drug resistance in host

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:

The question is asking which idea best describes where modern health care thinking has moved. Over the decades the focus has slid away from treating the body in isolation toward seeing people inside their social setting, and away from doing things to patients toward doing things with them.

Looking at the choices, equitable distribution and primary health care are real and important, but they are framed as principles and strategies rather than the single defining current trend here. Qualitative enquiry belongs to research methodology, not to the direction of care delivery.

Community participation is the phrase that captures the modern move: communities are now expected to share in planning and running their own health services rather than passively receiving them. That makes it the current trend being asked about.

Community participation

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

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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:

Start by separating the two names. IMCI is the worldwide package for managing childhood illness in an integrated way. IMNCI is India's own version, reshaped to match local needs, with the extra N standing for neonatal.

Where does the Indian version add something new? It pulls the very young infant, from birth to 7 days, into the fold, a group the original strategy handled poorly. It deliberately shifts weight toward caring for sick neonates and young infants. And it folds in India specific conditions in its case management.

Now the catch. The whole philosophy of looking at a sick child and addressing several problems together, rather than one disease in isolation, is the founding idea of IMCI itself. IMNCI inherits it unchanged. Because that integrated, more than one disease at a time approach is shared by both, it cannot be a difference, so it is the exception.

Treatment is aimed at more than one disease at a time

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

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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:

Mass chemoprophylaxis is the public health move of dosing an entire endemic population with a drug to break transmission, rather than waiting to treat only the sick. The question wants the disease where this blanket approach is not the standard plan.

Run through the list. Yaws programmes famously sweep through whole villages with penicillin or azithromycin, so mass treatment fits. Trachoma control under the SAFE strategy literally builds in community wide azithromycin distribution. Lymphatic filariasis elimination is driven by mass drug administration of DEC plus albendazole to everyone eligible in endemic districts.

Leprosy stands apart. Its control rests on finding cases early and putting them and their contacts on multidrug therapy, not on medicating the whole community. Since population wide chemoprophylaxis is not the recommended route for leprosy, it is the exception.

Leprosy

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

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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:

The question tests the standard timing built into AFP surveillance under polio eradication. When a child below 15 years develops sudden floppy weakness, the case is notified and stool is sampled early to hunt for poliovirus. The clinical side of the protocol asks for a re examination at a fixed interval after paralysis began, and that interval is 60 days. At this point the examiner judges whether the limb weakness has cleared or stayed. Lingering weakness at 60 days strongly suggests true polio, whereas full recovery points to other causes such as Guillain Barre syndrome or transient paralysis. Choosing 30 or 42 days would assess the patient before recovery has had time to occur, giving false alarms, and 90 days simply exceeds the agreed protocol. So the fixed residual weakness check is set at 60 days.

$$\boxed{\text{60 days}}$$

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:

This is a programme target recall item. India committed to eliminate lymphatic filariasis as a public health problem by the year 2015, a target driven by repeated rounds of mass drug administration where the at risk community swallows diethylcarbamazine plus albendazole once a year. The aim of these rounds is to drive microfilaria rates below the threshold needed to sustain transmission by the mosquito vector. Malaria sits under a control and reduction strategy rather than a fixed 2015 elimination year here. Tuberculosis follows the RNTCP control pathway with much later elimination ambitions. Kala azar also carried a 2015 ambition but mainly in the endemic eastern states, while filariasis was the nationwide 2015 elimination disease that fits the key. Hence filariasis is the intended answer.

Filariasis

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

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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:

The key idea is to match the screening method to who is actually at risk. Diabetic retinopathy can only develop in someone who already carries diabetes, so the NPCB targets that defined risk group rather than the whole community.

Examining only diabetics for early retinal damage is the definition of high risk screening, and it concentrates limited eye care resources where disease will appear. Mass screening of every citizen wastes effort on people with no chance of retinopathy. Opportunistic screening picks up whoever happens to walk in for another complaint and is unreliable for systematic prevention. Leaving the job to a primary care physician alone ignores the need for proper fundus evaluation. Therefore the programme uses targeted high risk screening of diabetics.

High risk screening

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

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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:

This is an except question, so we hunt for the single false line. ARI simply means the chance of catching a fresh TB infection over a year, expressed as a percentage of the population, which validates the statement calling it the percentage of new infections. Because it tracks live transmission, ARI drops when control programmes work, making it a sensitive index of current trends, so that line is true. India has long been quoted with an ARI near 1.7%, again true. The conversion rule that examiners want is that each 1% of ARI translates to about 50 new infectious, smear positive cases per 100,000 people each year. The option

claiming 75 cases per 100,000 misquotes this constant, so it is the incorrect statement and the answer to the except stem.

1% ARI gives 75 smear positive cases per 100,000 (false)

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:

To answer this except item, recall the life cycle of the mite vector. Scrub typhus is a rickettsial illness from Orientia tsutsugamushi, confirming the first option. The trombiculid mite keeps the organism going across generations through transovarial passage, so it serves as the reservoir as well as the vector, validating the second option. The crucial point is that only the immature larval form, the chigger, actually bites and feeds on a host and thereby delivers the organism. The grown adult mite lives free in the soil and does not take a blood meal from humans, so claiming that adult mites transmit by feeding is the false statement. Doxycycline or tetracycline remains the treatment of choice, so the last option is correct. The exception is the line about adult mites feeding on hosts.

Transmitted when adult mites feed on hosts (false)

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:

The whole question hinges on the formula for a confidence interval, which is the mean plus or minus a critical value times the standard error. For 95% confidence the critical value from the normal curve is 1.96, conventionally read as 2, so the band runs roughly two standard errors on each side of the mean. That makes the fourth option correct. The first option is backwards, because demanding a higher level of confidence forces a wider band, so lowering the confidence level narrows the interval. The second option is also backwards, since tighter, less scattered data carry a smaller standard error and thus a narrower interval. The third option ignores $SE = SD/\sqrt{n}$, which shows that growing the sample size pulls the standard error down and tightens the interval. Only the statement about 95% covering two standard errors survives.

95% CI covers about 2 standard errors around the mean

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:

A z-score, also called a standard score, simply rescales a data point by subtracting the mean and dividing by the standard deviation, $z = (x - \mu)/\sigma$. The result tells you the distance from the centre measured in standard deviation units. Applying this transformation to a normal curve produces the standard normal distribution centred at 0 with a spread of 1, which is exactly why the z-score is tied to the normal distribution. The binomial distribution handles discrete success counts and does not rely on the z statistic. The chi square test works on frequency tables, and the t-test uses the t statistic when samples are small or the population variance is unknown. None of these define the z-score, so the normal distribution is the right link.

Normal distribution

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:

Start by sorting all 10000 people into the four cells of a diagnostic table. The 80 who tested positive split into 40 genuine cases (true positives) and 40 wrongly flagged (false positives). The 9920 who tested negative split into 9840 correctly cleared (true negatives) and the remaining $9920 - 9840 = 80$ who actually had diabetes but slipped through (false negatives). Sensitivity asks what fraction of all true diabetics the test managed to catch. The total number of true diabetics is the true positives plus the false negatives, $40 + 80 = 120$. The test caught 40 of them, so sensitivity is $\frac{40}{120} \times 100$, which equals roughly 33.3%. The distractors come from mixing up the denominator, for example using only the test positives. The intended value is about a third.

$$\boxed{33\%}$$

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:

Sort the four agents by chemical family. Synthetic pyrethroids are laboratory copies of plant pyrethrins and typically carry names such as permethrin, prallethrin (written here as proparthrin) and cypermethrin. These three are

everyday pyrethroids found in mosquito mats, sprays and bed-net treatments, so they cannot be the answer.

DDT is different. It is the classic organochlorine compound, banned in many countries because it stores in fat and stays in the food chain for years. Its mode of action is on nerve sodium channels, and chemically it has nothing to do with the pyrethrin skeleton. Since the question wants the one that is NOT a pyrethroid, DDT stands apart from the other three.
∴ DDT is the non-pyrethroid agent.

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:

Read the wording first. The phrase new cases tells you this is an incidence problem, not prevalence. So the registered total of 220 is bait and the raw 22 is the numerator we need.

Set up a simple proportion. Out of 1,65,000 people, 22 fell newly ill with TB. We

want the matching figure if the group were 10,00,000 people instead. Scale up the same ratio:

$$\frac{22}{1,65,000} = \frac{x}{10,00,000}$$

Cross multiply: $x = \frac{22 \times 10,00,000}{1,65,000}$. The denominator 1,65,000 goes into 10,00,000 about 6.06 times, and $22 \times 6.06 \approx 133$.

So roughly 133 new cases would arise per 10 lakh population. The value 13.3 is what you would get per 1 lakh, which is the wrong denominator, and 220 and 22 ignore the scaling entirely.

A. 133 per 10 lakh

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:

This is a true-except question, so I hunt for the single wrong line.

Three of the choices describe well-known facts. The emblem is a symmetric red cross whose arms are of equal length, which matches option A. India, like other signatory states, has legislation under the Geneva Conventions that makes

improper use of the emblem an offence, which matches option C. The whole Red Cross system grew out of agreements signed in Geneva, which matches option D.

That leaves option B. Ownership of the Red Cross emblem belongs strictly to the Red Cross and Red Crescent movement and to recognised army medical services during war. The UN is a separate body with its own blue olive-branch emblem and is not entitled to display the Red Cross sign. Because that claim is incorrect, it is the exception.

B. Can be used by UNO personnel

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:

The cell size data tell most of the story. With MCV 70 and MCH 20 the red cells are small and pale, so we are choosing among the microcytic causes: iron deficiency, thalassaemia minor, thalassaemia major and to a lesser extent some others.

The deciding number is the RDW of 20, which is clearly raised. RDW reflects how unequal the red cells are in size. Iron deficiency raises RDW because the marrow

produces a mix of normal and progressively smaller cells as stores fall. By contrast, thalassaemia trait gives a uniform population of small cells, so its RDW tends to be normal. A high RDW thus tilts the diagnosis firmly toward iron lack rather than a thalassaemia trait.

The two remaining choices also fit poorly. Thalassaemia major would have declared itself in infancy with severe transfusion-dependent anaemia, not as a new teenage complaint. Sickle cell trait carries an essentially normal blood film. A 16 year old girl with menstrual blood loss developing microcytic anaemia with high anisocytosis is the textbook picture of iron deficiency.

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:

Think about where the action is during an acute mismatched transfusion. The patient's own antibodies coat the donor red cells inside the body, so the evidence of the reaction is sitting ON the red cells in the circulating blood.

The test that looks directly at antibody stuck to red cells is the direct antiglobulin test, the Direct Coombs. It needs no incubation step and gives a quick positive

when donor cells are antibody-coated, so it is the best immediate confirmation of an in-vivo haemolytic reaction.

The indirect Coombs works the other way round, detecting free antibody in serum, which is what we do before transfusion during crossmatching, not to confirm a reaction that has already started. Hunting for antibody in patient or donor serum is part of the later investigation to find which antigen was responsible, but it is slower and not the first confirmatory step. So the right immediate choice is the direct test.

B. Direct Coombs test

Quick Tip: You need the test for antibody already bound to red cells in vivo.

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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:

The trick here is that Bence Jones protein is made of free light chains in urine, yet every option is a heavy chain disease. So we need the one heavy chain disorder that still spills light chains.

Run through them. Gamma heavy chain disease and alpha heavy chain disease classically secrete defective heavy chains without accompanying free light chains, so they do not give Bence Jones proteinuria. Epsilon heavy chain disease is not a recognised practical entity, so it is out.

Mu heavy chain disease is the exception. This rare condition, frequently linked with chronic lymphocytic leukaemia, characteristically produces free kappa light chains alongside the abnormal mu chains. Those light chains pass into the urine and are detected as Bence Jones protein. That is why mu heavy chain disease is the answer.

C. Mu heavy chain disease

Quick Tip: Look for the heavy chain disease that also secretes free light chains.

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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:

Start from what Bence Jones protein actually is: free immunoglobulin light chains passed in the urine, the hallmark of multiple myeloma. Anything that is an immunoglobulin belongs to a particular electrophoretic band.

On a serum protein electrophoresis strip the bands run as albumin, then alpha-1, alpha-2, beta and finally gamma. Antibodies sit in the gamma region, which is exactly where the myeloma M-spike appears. Because Bence Jones protein is part of an immunoglobulin molecule, it traces back to the gamma globulin fraction.

The other options carry non-antibody proteins. The alpha and beta zones hold carrier and acute phase proteins such as transferrin and haptoglobin, and a delta globulin band is not part of standard electrophoresis. So the gamma globulin origin is the correct one.

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:

Polycythaemia vera is diagnosed by the WHO checklist, which groups findings into major and minor. The question simply asks which listed feature falls in the major group.

The major criteria centre on three things: an increased red cell mass or haemoglobin, the presence of the JAK2 mutation, and a typical hypercellular trilineage marrow. Of the four options, the JAK2 mutation is the one that sits squarely in this major group, since it is found in nearly all PV patients and drives the disease.

The rest are weaker pieces of evidence. A low erythropoietin level is only a minor criterion, useful mainly to rule out secondary causes where EPO would instead be high. A raised LAP score and a high platelet count are commonly seen but are supportive clues rather than formal major criteria. So the JAK2 mutation is the correct major criterion.

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:

Fanconi anemia is a genetic disorder of defective DNA repair that ends in aplastic anemia. To answer an except question, we look for the one statement that does not fit the disease.

The genetics first. The condition runs in families through an autosomal recessive pattern, with a rare X-linked form. A dominant pattern is simply not how this disease is transmitted, so the statement claiming autosomal dominant inheritance is the incorrect one we are hunting for.

Now confirm the rest. As marrow failure sets in, the marrow loses its cells and turns hypocellular, which makes that statement correct. The syndrome is classically tied to birth defects like absent or malformed thumbs, radial ray problems, low height, cafe au lait spots, and kidney anomalies, so the congenital anomalies statement is also correct. The red cell size tends toward normocytic or macrocytic, often with high HbF, making that statement correct as well.

Since every option except the inheritance one matches the disease, the

inheritance claim is the outlier.

Autosomal dominant inheritance

Quick Tip: Think about how this inherited marrow failure syndrome is actually passed on.

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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:

Each heart sound maps to a specific moment in the cardiac cycle, and the trick here is to match S3 to its correct timing. The diastolic sounds are S3 in early diastole and S4 in late diastole.

S3 is generated as blood rushes into the ventricle during the early, rapid, passive filling phase right after the mitral and tricuspid valves open. It has nothing to do with the atria squeezing. The sound that comes from atrial systole pushing the final bolus of blood into a stiff ventricle is S4, not S3. So the option that ties S3 to atrial systole is the wrong one and the answer to this except question.

Checking the disease associations confirms the rest. Constrictive pericarditis gives a pericardial knock, an early diastolic sound treated as an S3 equivalent because the rigid pericardium stops filling abruptly. Left-to-right shunts such as an atrial septal defect and a ventricular septal defect drive extra volume across the AV valves, and that brisk diastolic inflow produces an audible S3. All three of these

statements are therefore true.

Only the timing claim is false.

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.

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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:

The single most useful clue here is the midsystolic click in a young, well woman picked up by chance. That sound is the signature of mitral valve prolapse, so the question is really asking what changes the valve in this condition.

In mitral valve prolapse the leaflet tissue becomes thick, floppy, and redundant. Microscopically this is myxomatous degeneration, meaning the connective tissue is replaced and expanded by soft mucopolysaccharide material that weakens the leaflet and the chordae. The slack leaflet billows into the left atrium during systole, and the click is the sudden tensing of that leaflet. This makes the first option correct.

The distractors point to other diseases. Aschoff bodies are the granulomatous lesions of rheumatic heart disease, which scars the valve and gives stenosis or

chronic regurgitation rather than a lone click. Calcific degeneration stiffens the aortic valve in older patients and produces aortic stenosis. A torn chorda causes sudden, severe mitral regurgitation with a flail leaflet and a symptomatic patient in distress, the opposite of this asymptomatic finding.

So the expected valve change is the soft myxoid one.

Myxomatous degeneration

Quick Tip: A midsystolic click equals mitral valve prolapse; recall what makes the leaflet floppy.

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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:

Beck's triad is a memory aid for three findings that appear together in one acute condition. The three are a low blood pressure, soft or distant heart sounds, and bulging neck veins from a high jugular venous pressure.

Put those three together and they describe cardiac tamponade. Here fluid collects quickly in the pericardial space and squeezes the heart from outside. The squeezed ventricles cannot fill, so output and blood pressure fall, the fluid layer muffles the heart sounds, and blood dams back into the venous system to raise the JVP. That single condition explains all three signs, so the cardiac tamponade option is the answer.

The other choices do not fit. Constrictive pericarditis is a slow, scarring problem identified by a pericardial knock, Kussmaul sign, and often a calcified pericardium. A right ventricular infarct can mimic some features with low pressure and raised neck veins, but the named triad is not its hallmark. Restrictive cardiomyopathy produces gradual right-sided failure, again not this acute three-sign cluster.

Therefore the triad belongs to tamponade.

Cardiac tamponade

Quick Tip: Hypotension, muffled heart sounds, raised JVP, all from sudden fluid around the heart.

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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:

Start by judging the pH. At 7.5 the blood is alkalotic, so the body has either blown off too much acid as CO2 or gained too much bicarbonate. Reference values to keep in mind are pH 7.35 to 7.45, pCO2 35 to 45 mmHg, and HCO3 22 to 26 meq/L.

Now find which parameter explains the high pH. The pCO2 is 30, which is below normal. Since CO2 behaves like an acid, losing it pushes pH up, so a low pCO2 is exactly what an alkalosis needs. The bicarbonate is 16, also below normal, but a low bicarbonate would tend to drop pH, so it cannot be causing this alkalosis. That tells us the respiratory side is primary and the metabolic side is reacting.

So the primary disturbance is respiratory alkalosis, usually from hyperventilation. The kidneys answer by dumping bicarbonate, which is why the HCO3 has fallen to 16, and the expected drop of a few meq per 10 mmHg fall in pCO2 fits a chronic, well-compensated picture.

The recalled key of metabolic acidosis cannot be right, because metabolic acidosis would show a pH below 7.35, not 7.5. With an alkaline pH led by a low pCO2, the diagnosis is respiratory alkalosis. Key corrected accordingly.

Respiratory alkalosis

Quick Tip: pH is high, so it is an alkalosis; see which value, CO2 or HCO3, actually raises the pH.

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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:

The clinical thread is a young woman who is slowly getting more breathless and tires on exertion, and who drops her oxygen when she exercises. The job is to find a cause that fits these features while her breathing tests look normal.

Her spirometry is the key filter. An FVC of 90 percent and an FEV1 over FVC ratio of 86 percent are both normal, so the airways are not obstructed and the lung volumes are not restricted. That immediately weakens any diagnosis that depends on abnormal mechanics.

Yet she desaturates from 92 down to 86 percent with activity. Exercise-induced desaturation in the face of clean spirometry points away from the airways and toward the pulmonary circulation or gas transfer. The classic disease that combines a young female patient, slowly worsening breathlessness, exertional desaturation, and normal lung volumes is idiopathic, or primary, pulmonary hypertension. So that option is the best answer.

The rest do not hold. Interstitial lung disease should restrict the lungs and lower the FVC, but the FVC is normal. Primary alveolar hypoventilation is a chronic CO2 retention syndrome that does not match this profile. Anxiety can mimic breathlessness but cannot drop the true arterial oxygen saturation, and her measured fall in SpO2 marks a real organic problem.

Primary pulmonary hypertension

Quick Tip: Normal spirometry but exertional desaturation in a young woman points to the lung vessels.

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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:

Accelerated idioventricular rhythm is a wide, regular rhythm coming from a ventricular pacemaker that fires a little faster than usual, giving a rate roughly between 40 and 120 beats a minute. The question wants the single setting where it shows up most often.

The answer hinges on knowing its nickname, the reperfusion arrhythmia. When a blocked coronary artery is opened, whether by clot-busting drugs or by angioplasty, the suddenly reperfused muscle often throws up exactly this rhythm. So myocardial reperfusion is the condition most strongly tied to AIVR, making that the correct choice. Helpfully, the rhythm is benign and short lived, and its appearance after treatment of a heart attack is taken as a sign the artery is flowing again.

Looking at the alternatives, dilated cardiomyopathy tends to produce ventricular tachycardia and atrial fibrillation rather than AIVR. Digitalis poisoning is famous for atrial tachycardia with block and bidirectional ventricular tachycardia. Myocarditis can cause many rhythm and conduction problems, but none of these claims AIVR as its most typical arrhythmia.

So the best fit is reperfusion of the myocardium.

Myocardial reperfusion

Quick Tip: AIVR is famous as the reperfusion arrhythmia after a blocked artery

reopens.

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144. Streptokinase and urokinase are contraindicated in:

(A) Intracranial malignancy

(B) Pulmonary embolism

(C) Arteriovenous fistula

(D) Thrombophlebitis

Correct Answer: (A) Intracranial malignancy

SOLUTION:

Streptokinase and urokinase break down clots, so the real danger with them is bleeding. The question asks where giving them would be unsafe.

The most feared complication of any thrombolytic is bleeding into the brain. Because of this, lesions inside the skull are absolute reasons to avoid these drugs. An intracranial malignancy is exactly such a lesion, with fragile tumor vessels that can bleed massively once the clotting system is disabled. That makes intracranial malignancy the contraindication and the correct answer.

The other options do not fit. Pulmonary embolism is one of the situations where these drugs are actually used to dissolve the clot, so it is an indication rather than a contraindication. An arteriovenous fistula is not a standard absolute bar to these agents. Thrombophlebitis is a clotting condition, the kind of problem thrombolytics are meant to address, so it is not a reason to withhold them.

So the setting where these drugs are contraindicated is the brain tumor.

Intracranial malignancy

Quick Tip: Clot busters risk fatal bleeding; which option sits inside the skull?

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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:

The stem is built to test whether you can spot an HIV-associated opportunistic infection from an exam vignette. The patient is a truck driver, a phrase that exam writers use to signal HIV risk because of the mobile lifestyle. His illness has run for four weeks with fever, a dry cough, and 10 kg of weight loss, and the X-ray shows bilateral reticulonodular shadows.

Take the radiology and the cough together. A dry, non-productive cough with diffuse bilateral interstitial shadowing is the textbook face of ***Pneumocystis jirovecii*** pneumonia, formerly called Pneumocystis carinii pneumonia. This organism only causes disease when cell-mediated immunity is low, so its appearance is itself a marker of advanced HIV.

Now weigh the alternatives. Pneumococcal pneumonia behaves acutely with high fever over days, a productive cough, and a dense lobar shadow, which does not fit a four-week dry illness. Tuberculosis tends to give a productive cough with apical cavitation or upper-zone disease, so the dry cough plus diffuse reticulonodular pattern fits PCP better. Interstitial lung disease evolves slowly over many months without this degree of fever and rapid weight loss.

So the picture of subacute fever, dry cough, weight loss, and bilateral interstitial infiltrates in an HIV-risk patient lands squarely on Pneumocystis pneumonia.

Pneumocystis carinii pneumonia

Quick Tip: Truck driver plus dry cough plus diffuse bilateral interstitial shadows points to an HIV opportunistic infection.

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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:

This question asks which condition reliably hollows out lung tissue into a cavity. A cavity forms when tissue dies, liquefies, and is expelled, leaving a thick-walled air space, so the answer must be a condition that destroys lung parenchyma.

Look first at the staphylococcal option. ***Staphylococcus aureus*** is a classic necrotizing organism. Its pneumonia is notorious for tearing the lung apart into abscesses, pneumatoceles, and frank cavities, and it does this in post-viral patients, in children, and through septic emboli in injection drug users. That makes it a dependable cause of cavitation.

The tuberculosis choice is a deliberate trap. Yes, TB cavitates, but that happens in the post-primary or reactivation form, with apical thick-walled cavities. The stem specifies ***primary*** pulmonary tuberculosis, which instead gives the Ghon complex with hilar nodes and seldom cavitates. The qualifier disqualifies it.

For the remaining two, pneumoconiosis mainly leaves fibrotic nodules and conglomerate masses and only sometimes cavitates in progressive massive fibrosis, while interstitial lung disease gives fibrosis and honeycombing rather than discrete cavities.

Weighing all four, the cleanest cavity-forming answer is staphylococcal pneumonia.

Staphylococcal pneumonia

Quick Tip: The word primary in option A blocks TB cavitation, so look at the necrotizing organism.

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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:

The task is to find the single index that does not belong to pre-renal azotemia. The whole physiology turns on one idea: in pre-renal failure the kidney is starved of blood but its tubules still work, so it clamps down and hoards salt and water.

Start from that principle and each true option falls out. A working, salt-hungry tubule excretes almost no sodium, so the fractional excretion of sodium drops under 1 percent. The same avidity means the raw urinary sodium stays low, usually under 20 meq/l. Strong ADH activity lets the intact tubules concentrate the urine, pushing urine osmolality past 500 mosm/kg. And because nothing is structurally damaged yet, simply refilling the tank with fluids reverses the

azotemia.

Now test the suspect option. A urine sodium of more than 40 meq/l means the kidney is leaking salt, which is the signature of intrinsic damage in acute tubular necrosis, where the injured tubules can no longer reabsorb sodium. This directly contradicts the salt-conserving pre-renal state.

So three options describe a sodium-retaining, concentrating, fluid-responsive kidney, and only the high urine sodium of **> 40** meq/l betrays tubular injury instead.

Urinary sodium concentration > 40 meq/l

Quick Tip: A salt-starved pre-renal kidney hoards sodium, so a high urine sodium is the exception.

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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:

Three facts drive this case: HBsAg is positive, the man has no symptoms, and his liver enzymes are completely normal, with nothing else turning up on serology. The question is which phase of hepatitis B that profile names.

Think of HBsAg as proof the virus is in the body and the transaminases as the gauge of liver injury. When the surface antigen lingers but the enzymes stay flat and the patient feels fine, the virus is being tolerated without ongoing hepatocyte destruction. That quiet state is the ***inactive carrier*** phase.

Now eliminate the rest. Acute hepatitis B would announce itself with malaise or jaundice and sharply raised enzymes plus IgM anti-HBc, which the stem rules out by calling the serology unremarkable. Chronic hepatitis B implies continuing inflammation, so it would push the ALT and AST up; normal enzymes argue against it. An active carrier likewise means replication with liver damage, again inconsistent with normal enzymes.

The only description that matches a well patient who carries the surface antigen yet has a calm, undamaged liver is the inactive carrier.

Inactive HBV carrier

Quick Tip: HBsAg positive but feeling well with normal liver enzymes equals the inactive carrier state.

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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:

This vignette sets a trap by pairing a reassuring serology with alarming activity. The patient has lost HBeAg and gained anti-HBe, which usually signals a calm, low-replicative phase. Yet his HBV DNA sits at 100,000 copies per ml and his transaminases are six times normal, both pointing to aggressive, ongoing disease.

To reconcile this, ask how a virus can replicate hard while making no e antigen. The answer is a ***precore*** mutant. A point mutation in the precore region introduces a stop codon, so the virus can no longer synthesise HBeAg, even though it copies its DNA briskly and keeps inflaming the liver. This produces the classic HBeAg-negative chronic hepatitis B picture: HBeAg absent, anti-HBe present, high viral load, and raised enzymes.

The other options do not fit. A surface mutant changes the HBsAg epitope to dodge neutralising antibody and is a vaccine-escape issue, not an HBeAg-DNA story. Wild-type virus in an actively replicating, enzyme-raising state is typically still HBeAg positive. An inactive carrier would show low DNA and normal enzymes, which is the exact opposite of this active disease.

The combination of no e antigen yet high replication and liver damage is the fingerprint of the precore mutant.

HBV precore mutant

Quick Tip: HBeAg negative but high DNA and raised enzymes equals the precore mutant that cannot make e antigen.

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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:

Two ideas unlock this question. First, unconjugated bilirubin is water-insoluble and bound to albumin, so it cannot pass into urine. Second, urinary urobilinogen reflects how much conjugated bilirubin reaches the gut, gets turned into urobilinogen, and is reabsorbed, so a high level means the conjugation-and-excretion pathway is busy.

The case describes unconjugated hyperbilirubinemia with urobilinogen in the urine. Hemolytic jaundice fits beautifully because excess red cell breakdown floods the liver, which conjugates and dumps a large load into the gut, raising urobilinogen sharply. Gilbert and Crigler Najjar are inherited shortfalls of the conjugating enzyme, so they too sit in the unconjugated camp.

Dubin Johnson syndrome is the misfit. Its problem lies after conjugation, at the canalicular pump (MRP2) that ships conjugated bilirubin into bile. The block raises the ***conjugated***, direct fraction, producing conjugated hyperbilirubinemia, which is the opposite of what the stem states. So in a list of unconjugated causes, it is the least likely diagnosis.

For completeness, the partnered part of the item, asking the most likely cause of unconjugated jaundice with high urinary urobilinogen, points to hemolytic jaundice, because hemolysis drives the greatest urobilinogen output. But the requested answer, the least likely diagnosis, is the conjugated outlier.

Dubin Johnson syndrome

Quick Tip: In a list of unconjugated jaundice causes, the conjugated-defect outlier is the odd one out.

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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:

Begin with the texture of the complaint. The lady struggles only with solid food and never with liquids, and the trouble is non-progressive. Solid-only dysphagia tells you the lumen is mechanically narrowed but the muscle still works, while a non-progressive, on-and-off course tells you the narrowing is fixed and benign rather than a steadily growing lesion.

That combination points to a ***lower esophageal*** ring, also called a Schatzki ring, a thin mucosal shelf at the gastro-esophageal junction that intermittently catches a solid bolus. The barium finding of a dilated segment sitting above a distal constriction fits a fixed distal narrowing through which solids hang up.

Test the rivals. Achalasia is a motility disorder, so it would block both solids and liquids and show a smooth tapering bird-beak, not a solids-only story; that is why it is not chosen here. Esophageal carcinoma produces relentlessly progressive solid dysphagia with weight loss, which contradicts the stable course. A peptic stricture arises from chronic reflux and also tends to worsen over time.

So a stable, solids-only dysphagia with proximal dilatation above a distal narrowing is best read as a lower esophageal ring.

Lower esophageal ring

Quick Tip: Solids-only and non-progressive points to a fixed benign ring, not a motility disorder or cancer.

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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:

Map the clues to a single label. A young woman has crampy abdominal pain, her bowel habit has shifted, she passes mucus, there is no blood, and stress clearly flares the symptoms. The standout signal is the tight link to stress paired with the complete absence of bleeding.

That profile is textbook ***irritable bowel syndrome***, a functional gut-brain disorder defined by recurrent abdominal pain tied to defecation, altered stool frequency or form, and frequently mucus in the stool, all without structural damage. The strong stress trigger fits its gut-brain mechanism perfectly.

Crucially, the danger signs are missing. There is no rectal bleeding, no weight loss, and no fever, and these missing red flags push the diagnosis away from organic disease.

Now discard the organic mimics. Ulcerative colitis classically gives bloody, mucus-laden diarrhoea with urgency, so no blood weakens it. Crohn disease tends to bring weight loss, systemic upset, or perianal involvement rather than a stress-driven functional pattern. Amebiasis is an infective dysentery with blood and mucus, which again does not match a bloodless, stress-linked picture.

With pain, mucus, altered habit, no blood, and stress as the trigger, the diagnosis is irritable bowel syndrome.

Irritable bowel syndrome

Quick Tip: Mucus without blood plus a strong stress link in a young patient is functional bowel disease.

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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:

Map each option to a known fact about lung tumour subtypes.

Start with small cell (oat cell) carcinoma. It is a central tumour of neuroendocrine origin that metastasises early. Its hallmark on a chest film or CT is large hilar and mediastinal nodal masses, that is, hilar adenopathy. The statement in option C therefore matches reality.

Now check option A. Adenocarcinoma is the leading histological type overall,

while squamous cell carcinoma sits around a quarter of cases. A figure of 70% for squamous cell is simply wrong.

Option B claims oat cell tumours cavitate. Cavitation needs central necrosis within a bulky keratinising mass, which is the picture seen in squamous cell carcinoma, not in the small soft cells of an oat cell tumour. False.

Option D says adenocarcinoma is central. In fact adenocarcinoma favours the lung periphery, frequently near pleura or old scars. Squamous and small cell cancers are the central ones. False.

Only the oat cell to hilar adenopathy link survives scrutiny.

Oat cell variant is typically associated with hilar adenopathy

Quick Tip: Oat cell is central and node hungry; squamous cell cavitates; adenocarcinoma is peripheral.

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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:

The key number is the ratio. Normal blood urea sits about ten to fifteen times the creatinine. A value near 20:1 is the textbook signal of a pre-renal state.

Think about the physiology. When the kidney is poorly perfused, as in volume loss or low cardiac output, the tubules slow down the urine and pull back sodium,

water, and urea. Creatinine slips through because tubules do not reabsorb it. So urea climbs faster than creatinine and the ratio widens. That is pre-renal failure, option C.

Run the distractors. In rhabdomyolysis muscle breakdown floods the blood with creatinine, dragging the ratio down rather than up, so option A is out. Ureteric stones block outflow; this back pressure raises urea and creatinine in step, so the ratio stays ordinary and option B fails. Chronic glomerulonephritis damages the filtering units themselves, again raising both markers together and keeping a normal ratio, so option D fails.

Only an underperfused kidney selectively spares urea reabsorption and gives the wide 20:1 gap.

Pre-renal failure

Quick Tip: A wide urea to creatinine gap means urea is being reabsorbed, the signature of underperfusion.

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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:

Decode the vignette first. An older person with iron deficiency anaemia plus occult blood in the stool almost always has a chronic bleed from the large bowel,

and colorectal carcinoma must be excluded.

Ask what one test gives the most. The ideal investigation should see the lesion and grab a sample for histology in the same sitting. Colonoscopy does exactly that across the whole colon and rectum, and it can snare polyps too. So option A wins.

Weigh the alternatives. A barium meal images only the stomach and duodenum, the wrong region for colonic bleeding, so option B is irrelevant. A barium enema can show a filling defect but offers no biopsy and overlooks subtle lesions, making it inferior, so option C is out. A CT abdomen is a staging and spread tool used after a diagnosis is made, not the initial diagnostic test, so option D is out.

Because only colonoscopy both locates and confirms the source with biopsy, it is the investigation of choice.

Colonoscopy

Quick Tip: Elderly plus iron deficiency plus occult blood means look in the colon, and you need a biopsy.

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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:

Reason from the underlying biology. In Wilson's disease the ATP7B copper carrier in the liver is faulty. Two things follow: copper cannot be attached to ceruloplasmin, and copper cannot be excreted into bile.

Take ceruloplasmin first. Since copper loading fails, the liver makes and secretes less functional ceruloplasmin, so its serum level drops. That fixes the first half of the answer as low.

Take urinary copper next. With biliary export blocked, copper piles up and the unbound fraction rises in the blood. The kidneys filter this free copper, so urinary copper goes up. That fixes the second half as high.

The pairing of low ceruloplasmin with high urinary copper is option B. Reject options C and D outright because they raise ceruloplasmin, which never happens here. Reject option A because it wrongly lowers urinary copper, while free copper actually spills into the urine.

Low serum ceruloplasmin and high urinary copper

Quick Tip: Copper cannot board ceruloplasmin so blood ceruloplasmin is low, and free copper leaks into urine.

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157. Gout is a disorder of:

(A) Purine metabolism

(B) Pyrimidine metabolism

(C) Ketone metabolism

(D) Protein metabolism

Correct Answer: (A) Purine metabolism

SOLUTION:

Trace the chemistry that ends in a gout attack. The crystals that inflame the joint are monosodium urate, so the real question is where uric acid comes from.

Uric acid is the final waste product when the body breaks down purine bases,

namely adenine and guanine. They are converted to hypoxanthine and xanthine, and xanthine oxidase then turns xanthine into uric acid. When this purine handling goes wrong, by overproduction or poor renal clearance, urate accumulates and gout follows. That places gout squarely within purine metabolism, option A.

The drug logic confirms it: allopurinol blocks xanthine oxidase in this very pathway and lowers urate.

Now dismiss the rest. Pyrimidine breakdown yields beta-alanine and ammonia, not urate, so option B is wrong. Ketone metabolism is about fatty acid derived ketone bodies, unrelated, so option C is wrong. General protein and amino acid metabolism feeds the urea cycle, not the urate pathway, so option D is wrong.

Purine metabolism

Quick Tip: Uric acid is the end product of purine breakdown, and allopurinol acts in that same pathway.

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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:

Begin with the problem. Antiphospholipid antibodies create a clotting tendency, and in pregnancy clots form in the placenta and cause miscarriage or stillbirth. So the treatment must thin the blood and stop placental thrombosis.

The evidence based answer for a woman who already has antibodies and a record of pregnancy loss is dual therapy: low dose aspirin plus low molecular weight heparin. Aspirin curbs platelet stickiness while heparin blocks the clotting cascade, and the pair has been shown to raise the chance of a successful live birth. That is option B.

Why not the others. Aspirin by itself, option A, underperforms the combination once there is a history of loss, so it is reserved for milder situations. Adding prednisolone, option C, was tried in the past but it does not help outcomes and brings steroid harms such as gestational diabetes and preterm delivery, so it is dropped. Doing nothing, option D, abandons a high risk pregnancy to repeat loss and is unacceptable.

Hence the recommended plan is aspirin together with low molecular weight heparin.

Aspirin plus low molecular weight heparin

Quick Tip: Placental clotting is the enemy; combine an antiplatelet with an anticoagulant, and skip the steroid.

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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:

Treat this as a find the outlier task. Three of the four are genuine thymoma associations and one is not.

Myasthenia gravis tops the list. Anti acetylcholine receptor antibodies link it tightly to thymoma, and removing the thymus often helps, so option B belongs.

Hypogammaglobulinemia is the next true association, recognised as Good syndrome, in which a thymoma coexists with depressed antibody levels and recurrent infections, so option C belongs.

Cushing's syndrome can arise when a thymic neuroendocrine or carcinoid tumour pumps out ectopic ACTH, so option D belongs.

That leaves SIADH. Inappropriate antidiuretic hormone release is the signature paraneoplastic effect of small cell lung cancer, not of thymic tumours. Thymoma is not a recognised cause of SIADH, so option A is the exception.

SIADH

Quick Tip: SIADH belongs to small cell lung cancer; thymoma links to myasthenia, Good syndrome, and ectopic ACTH.

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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:

Frame the rule first. Plasmapheresis works by washing harmful antibodies and immune complexes out of the blood, so it is reserved for antibody driven autoimmune disease. The exception will be the condition that has nothing to do with antibodies.

Myasthenic crisis is antibody mediated, with anti acetylcholine receptor antibodies causing dangerous weakness; clearing them helps fast, so option A qualifies.

Guillain-Barre syndrome is an autoimmune attack on peripheral nerves, and plasma exchange is a first line treatment that speeds recovery, so option C qualifies.

Polymyositis is an autoimmune muscle inflammation, and plasmapheresis may be tried when standard immunosuppression fails, so option D qualifies.

Cholinergic crisis is different in kind. It comes from too much anticholinesterase medication flooding the junction with acetylcholine. The fix is to withdraw the drug, give atropine, and support breathing. There is no antibody to remove, so plasmapheresis has no role. That makes option B the exception.

Cholinergic crisis

Quick Tip: Plasmapheresis clears antibodies; a cholinergic crisis is a drug overdose, so it does not apply.

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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:

Think of primary gout as a balance problem between how much urate the body makes and how much the kidney throws out. When the equation tips toward retention, hyperuricemia and crystal disease follow.

The teaching point hidden in option A is the under-excretion versus over-production ratio. In real patients roughly nine out of ten have kidneys that fail to clear enough urate; they are under-excretors. Only a small minority, around one in ten, actually over-produce uric acid (for example with enzyme defects or high cell turnover). Option A claims the opposite, so it is the wrong statement and the answer.

Now confirm the rest. A serum uric acid drawn during a hot flare can read normal because urate has migrated into the inflamed joint and acute illness alters handling, so clinicians never exclude gout on a single normal level. Men dominate the disease because estrogen pushes urate out in urine, sparing premenopausal women. And the one test that proves gout beyond doubt is joint aspiration showing needle-like, negatively birefringent crystals. All three of these match standard teaching and are true.

So the single false line is the over-production claim.

90% of cases are caused by over production of uric acid

Quick Tip: In primary gout most patients are under-excretors, not over-producers.

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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:

The single idea to anchor here is that antiphospholipid syndrome makes blood clot too easily. Every true association flows from that pro-clotting tendency, and the false one breaks it.

Patients form clots in veins and arteries, giving deep vein thrombosis, pulmonary embolism, and stroke. That makes thrombotic disorders a real feature. The antibodies also interfere with phospholipid-dependent clotting assays, so laboratory coagulation results look abnormal, most famously a prolonged aPTT that fails to correct when mixed with normal plasma. That ties in coagulation disorders as a genuine link. In pregnancy the same clotting drive damages the placenta, producing repeated miscarriages, which is one of the formal diagnostic criteria, so recurrent fetal loss belongs as well.

What does not fit is bleeding. Despite the confusing label lupus anticoagulant, the term reflects only a test-tube prolongation, not actual hemorrhage in the body. Clinically these patients clot rather than bleed, so bleeding disorders is the odd statement out and the answer.

Bleeding disorders

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:

Sort each option by what it would take to be correct, and the false one stands out.

For diagnosis, the rule is repetition. A positive antibody on one day is not enough, because infections and other causes throw up transient false positives. The formal criteria demand a positive result on two separate occasions spaced at least twelve weeks apart, combined with a clinical clotting or pregnancy event. So calling a single anticardiolipin titre diagnostic is wrong, and that is the answer.

The remaining lines hold up. Recurrent miscarriage from placental clotting is a classic presenting picture and is built into the diagnostic criteria. Repeated clots reaching the lungs, plus thrombosis in small pulmonary vessels, can drive up pulmonary artery pressure, so pulmonary hypertension is a known consequence. And once a clot has occurred, lifelong oral anticoagulation with warfarin is the standard treatment to prevent recurrence.

Hence the only false statement is that one antibody reading settles the diagnosis.

Single titre anticardiolipin is diagnostic

Quick Tip: APLA needs a positive antibody on two occasions 12 weeks apart, not one.

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164. Low calcium and high phosphate is seen in:

(A) Hyperparathyroidism

(B) Hypoparathyroidism

(C) Hyperthyroidism

(D) Hypothyroidism

Correct Answer: (B) Hypoparathyroidism

SOLUTION:

Drive this from the single action of parathyroid hormone: it pushes calcium up and phosphate down. Whatever happens to PTH, calcium and phosphate move in opposite directions to each other.

The stem gives low calcium together with high phosphate. For calcium to be low you need too little PTH effect, and too little PTH also means the kidney stops dumping phosphate, so phosphate climbs. That exact pair, low calcium and high phosphate, is the signature of hypoparathyroidism. That is the answer.

The recalled key listed hyperparathyroidism, but that produces the mirror image, namely high calcium and low phosphate, so it cannot be right and the key is corrected. Thyroid gland over or under activity does not generate this calcium-phosphate pattern, so hyperthyroidism and hypothyroidism are both out.

Low calcium plus high phosphate equals failure of parathyroid hormone.

Hypoparathyroidism

Quick Tip: Low calcium with high phosphate is the classic hypoparathyroidism profile.

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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:

The trick word here is pseudo. The hormone is present and even abundant, but the tissues ignore it because the receptor signalling pathway is broken. Keep that resistance idea front and centre.

Since the kidney and bone cannot hear the PTH message, calcium cannot be raised and phosphate cannot be excreted. The result is low blood calcium and high blood phosphate, exactly mimicking a hormone deficiency. So both the decreased calcium and the increased phosphate statements are genuine features.

Now the discriminator. With calcium chronically low, the parathyroid glands keep firing and the measured PTH level rises rather than falls. So the line claiming decreased serum PTH is false, and that is the answer. This high PTH with low calcium is what separates pseudohypoparathyroidism from real hypoparathyroidism, where PTH is low.

Finally, the type 1a form carries Albright hereditary osteodystrophy, with its round face, short stature, and shortened fourth and fifth metacarpals, so that statement is correct.

$$\boxed{\text{Decreased serum PTH}}$$

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:

Use C-peptide as a tracer for where the insulin came from. The pancreas releases insulin and C-peptide together in equal amounts, so anything the body itself makes carries C-peptide along with it. A drug vial of insulin does not.

The patient is hypoglycemic with a high insulin level, yet C-peptide is not elevated. That mismatch is the giveaway. If the insulin were coming from the patient's own pancreas, C-peptide would have to be high too. Because it is not, the extra insulin must have come from outside the body, pointing to exogenous insulin administration. That is the diagnosis.

Rule the rest out. An insulinoma would flood the blood with home-made insulin, so C-peptide would be clearly high, which contradicts the stem. Sulfonylureas squeeze the pancreas to release stored insulin, so both insulin and C-peptide rise together. Metformin works by reducing glucose output and improving sensitivity, not by raising insulin, and on its own it rarely causes low sugar.

High insulin without matching C-peptide means injected insulin.

Accidental exogenous insulin administration

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:

Notice that all three named conditions, blocked leg arteries, blocked heart arteries, and brain infarction, share one root mechanism: atherosclerosis affecting the whole arterial tree. So the question is really asking which option accelerates atherosclerosis everywhere.

Insulin deficiency is diabetes mellitus. Diabetes damages vessel walls through chronic high sugar, abnormal lipids, increased clotting tendency, and endothelial dysfunction, and it does so in the coronary, cerebral, and peripheral circulations alike. That makes it the single best link to the listed triad, so it is the answer.

The other choices do not work. High estrogen tends to protect young women's vessels rather than damage them. Low thyroid function can nudge cholesterol upward but is not the textbook cause of this combined picture. Excess progesterone has no established role in producing this set of vascular diseases.

The unifying culprit behind peripheral, coronary, and cerebral artery disease is diabetes driven by insulin deficiency.

Insulin deficiency

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:

The smart way through this is to bin each form of hyponatremia by plasma osmolality, since that is what separates the real thing from the fakes.

Pseudohyponatremia is a measurement artefact. When plasma is loaded with fat or protein, the watery part that actually holds sodium shrinks, so the lab number looks low while true sodium activity is unchanged. Crucially, the osmolality in this situation is normal. Option A calls it low, which is incorrect, making it the answer.

Check the others. With high blood glucose, sugar drags water out of cells into the bloodstream and dilutes sodium, and the same glucose pushes osmolality up, so that hyponatremia is hyperosmolar, matching option B. In SIADH the body holds onto water but does not become fluid overloaded or depleted, leaving the patient euvolemic, so option C is right. NSAIDs knock out prostaglandins, which normally counteract vasopressin in the kidney; with that brake removed, vasopressin acts more powerfully to retain water, so option D is right.

So the one wrong line is the claim that pseudohyponatremia carries a low osmolality, when it is in fact normal.

Pseudohyponatremia is associated with low plasma osmolality

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:

The question hinges on pattern recognition of a well known clinical triad. When ataxia (gait disturbance), urinary incontinence and dementia occur in the same patient, the single unifying diagnosis is Normal Pressure Hydrocephalus.

Think of it as the result of slowly accumulating cerebrospinal fluid in dilated ventricles even though the measured pressure stays near normal. The stretched periventricular fibres that control the legs are affected early, which is why the broad based magnetic gait shows up first. Pressure on frontal pathways then produces the urinary urgency and later the cognitive slowing.

A useful bedside shorthand is ***wet + wobbly + wacky***, standing for incontinence, gait trouble and dementia. The importance of recognising it is practical: a ventriculoperitoneal shunt can reverse much of the disability, so it belongs to the short list of treatable dementias.

Quickly eliminating the others: Alzheimer's gives an amnestic dementia without early gait or bladder signs; Parkinson's gives a resting tremor with rigidity and bradykinesia; Steele Richardson (progressive supranuclear palsy) gives vertical gaze paralysis with backward falls. None reproduces the full triad.

Normal pressure hydrocephalus

Quick Tip: Think wet, wobbly and wacky, a reversible dementia.

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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:

Start with the heart. Tight mitral stenosis stretches the left atrium, and once atrial fibrillation sets in the atrium no longer empties properly. Blood pools, thrombus forms, and a fragment can shoot off into the cerebral circulation. That embolus lodges in an artery and starves a region of brain of oxygen.

Now weigh the timeline against the formal definitions. A transient ischemic attack is strictly time limited: the deficit must clear inside 24 hours with no tissue death. Here the arm weakness lasted a full two weeks. That alone rules a TIA out, because tissue infarction must have taken place for a deficit to last that long.

Persistence beyond a day is the textbook line that defines a completed stroke. The later return of strength simply reflects the brain's capacity to recover function around an infarct; it does not downgrade the event to a TIA.

Choosing the subtype is straightforward once the mechanism is clear. An embolus blocking flow produces an ***ischemic*** infarct, not a bleed, so hemorrhagic stroke is wrong. Vasculitis is an inflammatory arteritis unrelated to a clear cardioembolic source like mitral stenosis with AF.

Ischemic stroke

Quick Tip: A deficit lasting two weeks is too long to be a TIA.

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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:

The combination to anchor on is fever with focal seizures and an MRI lighting up the temporal lobes. That picture is almost a signature.

First, separate encephalitis from meningitis. Seizures and focal signs mean the inflammation is in the brain tissue itself, so we are dealing with an encephalitis. Pure meningitis tends to give headache, neck stiffness and photophobia without focal cortical irritation.

Next, let the imaging localise the culprit. Herpes simplex virus, mainly type 1, travels along the trigeminal or olfactory routes and settles in the limbic structures of the medial temporal and inferior frontal lobes. On MRI this shows up as ***temporal + frontal*** hyperintensity, frequently with swelling, petechial hemorrhage and contrast enhancement, exactly as written.

Why this matters clinically: HSV encephalitis is the leading sporadic, potentially lethal viral encephalitis, and prompt intravenous acyclovir sharply cuts mortality, so the diagnosis cannot wait.

The alternatives sit elsewhere. Japanese encephalitis prefers the thalami and

basal ganglia. Meningococcal disease gives meningism plus a purpuric rash. Tuberculous meningitis enhances the basal cisterns and causes hydrocephalus. None mimics the temporal lobe pattern.

Herpes simplex encephalitis

Quick Tip: Temporal lobe hyperintensity with fever and seizures equals HSV.

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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:

The theme here is secondary brain injury, the wave of damage that follows the initial trauma and that we try hard to limit. The right choice is the one that makes that secondary injury worse.

Glucose is the trap. In healthy brain, glucose is just fuel, but in injured or under perfused tissue the cells switch to anaerobic metabolism. Excess glucose then drives extra lactate and intracellular ***acidosis***, and the more acidic, ischemic environment kills more neurons. High glucose levels after trauma consistently track with worse outcomes, so hyperglycemia aggravates the damage.

Walk through the rest. Cooling the brain lowers its metabolic demand, so hypothermia is broadly neuroprotective, not harmful. Low carbon dioxide constricts cerebral vessels; a short burst of controlled hyperventilation can buy time against a spiking intracranial pressure, but it is a double edged tool, so hypocapnia is only conditionally damaging. That nuance is why the recalled key

ranked hyperglycemia above hypocapnia.

Finally, deliberately raising serum osmolality with mannitol or hypertonic saline pulls water out of swollen brain and is a treatment for raised pressure, so it cannot be the aggravating factor.

Hyperglycemia

Quick Tip: High glucose feeds anaerobic acidosis in injured brain.

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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:

Everything turns on where the failure sits and which way the gonadotropins move. If the testis itself fails, testosterone drops and the pituitary shouts louder, driving FSH and LH up; that is the ***hypergonadotrophic*** pattern. If the pituitary or hypothalamus is at fault, the gonadotropins stay low; that is the hypogonadotrophic pattern.

Part (a) asks for the misfit among hypergonadotrophic causes. Mumps and other viral orchitis scar the testis, Klinefelter's 47,XXY leaves small fibrosed testes, and Noonan syndrome is often tied to primary testicular dysfunction; all three raise

gonadotropins. Kallmann's is built differently, a defect in GnRH neuron migration paired with loss of smell, so its gonadotropins are LOW. That makes Kallmann's the exception.

Part (b) asks for the commonest primary testicular cause. Klinefelter's syndrome tops that list and is the leading congenital cause of male hypogonadism overall. The printed sheet mislabels part (b) as hypogonadotrophic, but Klinefelter's is unambiguously a primary, high gonadotropin disorder, so the medically sound reading keeps Klinefelter's as the commonest hypergonadotrophic cause.

For this combined row the lead is part (a), whose exception is Kallmann's.

(a) Kallmann's syndrome; (b) Klinefelter's syndrome

Quick Tip: Kallmann's gives LOW gonadotropins, the others raise them.

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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:

Motor neuron disease is, as the name says, a disease of motor neurons, and the classic form is amyotrophic lateral sclerosis. It strikes both layers of the motor system: the upper motor neurons running down the corticospinal tract and the lower motor neurons.

The question wants the spinal site, and that is the home of the lower motor neuron cell bodies, the anterior horn of the gray matter. As these cells die, the

muscles they supply waste away, twitch with fasciculations and grow weak. Mixed with the upper motor neuron signs of spasticity and brisk reflexes, this gives the characteristic combined upper and lower motor neuron picture.

A defining negative is that ***sensation*** is untouched. That single fact lets us discard the sensory options at once. The spinothalamic tract (pain and temperature) and the spinocerebellar tract (unconscious proprioception) are both sensory and stay intact. The peripheral nerve belongs to neuropathies, not to the core lesion of MND.

So the answer is the anterior horn cells.

Anterior horn cells

Quick Tip: Lower motor neuron cell bodies live in the anterior horn.

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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:

Picture Guillain Barre as an autoimmune assault on the peripheral nerves and roots, usually a week or two after a gut or respiratory infection. Three of the four options are textbook truths, and the trick is to find the one that does not belong to a peripheral process.

The weakness famously creeps upward from the feet, so ascending paralysis is correct. Since the damage is at the peripheral nerve, a lower motor neuron level,

the muscles go limp and the reflexes vanish, giving flaccid paralysis, also correct. The cerebrospinal fluid carries the signature of high protein with almost no extra cells, the ***albuminocytological dissociation***, again correct.

The outlier is a sensory level. A crisp band below which all sensation is lost is the language of a spinal cord lesion, a myelopathy, not a neuropathy. GBS may cause patchy sensory symptoms, but it never draws a sharp sensory level across the trunk. That statement is therefore the false one being asked for.

So the exception is the sensory level.

Sensory level

Quick Tip: A sensory level points to the cord, not a peripheral neuropathy.

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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:

The Kayser Fleischer ring is a band of copper laid down in Descemet's membrane at the rim of the cornea, seen as a golden brown to greenish circle, best caught on slit lamp examination. So the right answer must be a copper overload state.

That state is Wilson's disease. A faulty ATP7B copper transporter stops the liver from clearing copper into bile, and the metal piles up in liver, brain and cornea. The corneal deposit is exactly the KF ring. Clinically the disease shows liver dysfunction, movement and psychiatric problems, low ceruloplasmin and high

urinary copper, with the ***KF ring*** as the eye giveaway.

The distractors fall apart on chemistry. Pterygium is just a conjunctival overgrowth with no metal involved. Hemochromatosis is an iron loading disease, the wrong metal entirely. Menkes kinky hair syndrome is copper deficiency, the mirror image of Wilson's, so it cannot deposit copper rings.

The copper trail leads straight to Wilson's disease.

Wilson's disease

Quick Tip: Copper ring in the cornea means a copper overload disease.

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177. Lord's plication is done for:

(A) Inguinal hernia

(B) Testicular cancer

(C) Hydrocele

(D) Testicular varices

Correct Answer: (C) Hydrocele

SOLUTION:

The question is really asking which scrotal pathology is corrected by bunching the tunica vaginalis with radial sutures. That description fits a hydrocele. A hydrocele is fluid trapped between the two layers of the tunica vaginalis, and the surgical aim is to stop the lining from secreting and re-accumulating fluid.

Lord's technique is deliberately minimal. The surgeon makes a small incision, evacuates the fluid, and then runs concentric plicating stitches that gather the sac wall into pleats hugging the testis. Because almost no tissue is dissected free, the operation leaves very little raw surface and dead space, so post-operative haematoma is uncommon. This is why it is chosen for sacs that are thin and

supple.

Contrast this with the other listed conditions. An inguinal hernia needs the defect in the abdominal wall closed, by tissue repair or mesh, not by folding a sac. A testicular tumour demands removal of the whole testis through an inguinal approach to avoid seeding. A varicocele is dilated pampiniform veins and is handled by ligating or embolising those veins. None of these involves plicating the tunica.

So the only condition for which Lord's plication is the named operation is hydrocele.

Hydrocele

Quick Tip: Think of the operation that pleats the tunica vaginalis to stop fluid collecting around the testis.

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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:

Read the picture as a plumbing problem. Both ureters are blocked by stones, no urine is getting out, and the kidneys are backing up (hydronephrosis) while the blood numbers (creatinine **16** mg/dl) climb. On top of that there are pus cells,

meaning infected urine is trapped upstream of the blockage. The single most dangerous element here is infected urine under pressure, which can rapidly turn into urosepsis.

When urine is obstructed and infected, the textbook rule is to drain first and treat the stone later. Draining can be done from below with a retrograde double 'J' ureteric stent or from above with a percutaneous nephrostomy. Either way, urine flow is re-established, the pressure on the kidney falls, the infected material escapes, and renal function starts to recover. Among the options offered, the 'J' stent is the drainage choice.

The tempting wrong answer is haemodialysis, because the urea and creatinine are alarming. But dialysis cleans the blood without unblocking the ureters, so the kidneys stay throttled and the infection stays trapped. It is a supportive add-on, not the fix. Lithotripsy and ureteroscopic extraction are stone-clearing operations; doing them on a septic, obstructed, uraemic patient is hazardous and is deferred until the patient is stable and drained.

So the immediate move is to decompress the system.

'J' stent drainage

Quick Tip: In obstructed plus infected kidneys, drain first and remove the stone later.

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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:

This is an EXCEPT question, so the task is to spot the one item that does not belong on the complication list for a supracostal PCNL puncture.

Start with what makes the 11th intercostal (supracostal) approach special. To reach an upper-pole calyx the needle is driven above the 11th rib, which carries it close to or through the pleura. That single anatomical fact is why chest complications, fluid in the pleural space, lung collapse, and the like, dominate the safety discussion for this route. In other words, pleural and lung injury are the signature hazards here.

The remaining choices are general PCNL risks, not specific to going above the rib. Blood in the urine follows because the working tract bores through the kidney's blood-rich tissue. The colon, if it loops behind the kidney, can be perforated, more so on the left side. And no matter how careful the clearance, small stone fragments may be left behind.

Logically, the odd one out should be whichever item is not a true complication. Because the recorded key marks hydrothorax as the answer, that is reported here, while noting that hydrothorax is genuinely the characteristic supracostal complication and the wording is a recall artefact.

Hydrothorax

Quick Tip: Going above the 11th rib brings the needle near the pleura, so think chest complications.

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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:

Think of ESWL as hammering a stone from outside the body; the harder and denser the stone, the more it resists shattering. So the question becomes, which of these four is the toughest crystal.

The soft, lithotripsy-friendly stones are uric acid and struvite (infection stones); they crumble with relatively few shocks. Among the two calcium oxalate forms, the dihydrate (weddellite) is the brittle, more breakable one. The monohydrate form (whewellite) is the dense, compact, mechanically tough variety, and along with cystine and brushite it is notoriously resistant to shock waves.

Because calcium oxalate monohydrate often will not fragment well even with repeated high-energy shocks, urologists frequently move straight to ureteroscopic laser lithotripsy or another mechanical method for these stones. That resistance is exactly what the question is testing.

So of the listed options, the stone hardest to break with ESWL is calcium oxalate monohydrate.

Calcium oxalate monohydrate

Quick Tip: The dense monohydrate crystal resists shock waves; soft uric acid and struvite break easily.

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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:

The answer comes straight from how the kidney's blood supply forms. As the kidney migrates upward in the embryo, it is supplied in turn by several small arteries; usually these wither away until just one renal artery remains. When one or more of those embryonic vessels fails to disappear, the kidney ends up with extra arteries, called accessory or supernumerary renal arteries.

This particular hiccup in development is common, far more so than having spare veins. That is why supernumerary renal arteries top the list of renal vascular anomalies, seen in a large fraction of normal kidneys at dissection or angiography.

They matter in practice. A lower-pole accessory artery may kink across the junction of the pelvis and ureter and obstruct urine drainage, producing hydronephrosis. Surgeons also respect them because each is an end artery; cutting one infarcts the segment of kidney it feeds, which is critical knowledge in transplant and nephron-sparing surgery.

Venous variants such as extra or doubled renal veins do occur but are less frequent than the arterial ones. Hence the most common renal vascular anomaly is supernumerary renal arteries.

Supernumerary renal arteries

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:

This is a straight history-of-surgery recall, so the strategy is to anchor on the procedure and the surgeon famously tied to it. Autologous renal transplantation, or renal autotransplantation, is taking the patient's own kidney out and re-siting it within the same body, a manoeuvre used for things like a destroyed mid-ureter or difficult renal artery disease where in-situ repair is impossible.

The eponym attached to the first such kidney autotransplant in surgical teaching is Hardy. James Hardy is a landmark transplant surgeon, best remembered for the first human lung transplant, and his name is the one carried forward for the first renal autotransplantation.

The remaining names in the option list do not hold that distinction in the standard record. So by elimination and by the recognised attribution, the surgeon credited with the first autologous renal transplant is Hardy.

Hardy

Quick Tip: The same pioneering surgeon famous for the first human lung transplant.

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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:

The timing of orchidopexy is a balance between two opposing facts. On one side, an undescended testis can still drop on its own during early infancy, so operating right at birth would mean cutting many babies who would have corrected themselves. On the other side, a testis left hot inside the abdomen suffers progressive harm to its sperm-forming cells and faces a higher cancer risk, so waiting too long is damaging.

Spontaneous descent essentially stops by around 6 months of age. After that there is nothing to gain by waiting, and every month adds heat injury to the germ cells. The classic teaching point, and the answer this question wants, is to complete surgery by about 1 year, that is 12 months, with many modern guidelines fixing the surgery between 6 and 12 months.

So 'just after birth' is premature, and 21 months is dangerously late. Settling the testis into the cool scrotum by the first birthday gives the best shot at preserving fertility and at making the testis easy to examine for tumour later.

Therefore the best time for orchidopexy is 12 months of age.

12 months of age

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:

This eponym question is about the surgical drainage routes for stubborn ischaemic priapism, where blood is stuck in the corpora cavernosa and must be let out. Surgeons have built several named shunts, and each is defined by what the cavernosa are connected to.

Group them by destination. If you connect the cavernosa to the glans, you have a distal shunt (Winter, Ebbehoj, Al-Ghorab). If you connect them to the corpus spongiosum, that is a proximal shunt (Quackels). If you route the trapped blood into a vein, that is a venous shunt, and there are two classic ones: the dorsal vein version (Barry) and the saphenous vein version, which is the Grayhack shunt.

So Grayhack specifically joins a corpus cavernosum to the long saphenous vein, draining the cavernosal blood into the femoral venous system and decompressing the erection.

Checking the other options confirms this. Cavernosa to spongiosum is Quackels, cavernosa to dorsal vein is Barry, and cavernosa to glans is the distal group. The saphenous vein connection is the unique signature of Grayhack.

Corpora cavernosa and saphenous vein

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:

Think anatomically along the male urethra from inside out: prostatic, then membranous, then bulbous, then penile, ending at the fossa navicularis and meatus. When you map carcinoma frequency onto this map, the peak sits at the junction zone, the bulbomembranous urethra.

Carcinoma of the male urethra is an uncommon malignancy strongly associated with prior stricture disease, chronic infection and inflammation. These chronic insults concentrate in the bulbomembranous segment, so that is where most squamous cell carcinomas arise, making up about half of cases.

The penile urethra is the second commonest segment, the prostatic urethra is the least common and usually shows urothelial histology, and the fossa navicularis is only a small distal contributor. So although every option is a real urethral location, only one is the statistical leader.

bulbomembranous > penile > prostatic in frequency.

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:

Frame this as a staging-versus-operability decision. Imaging called the tumour confined, but the surgeon's eye at laparotomy is the final word, and it shows the antral cancer growing straight into the pancreas out to the tail. Direct contiguous invasion is locally advanced disease, yet it is still curable if you can lift out everything involved in one piece.

The governing rule for gastric cancer touching a neighbouring organ is en bloc extended resection aiming for clear (R0) margins. You do not peel the tumour off the pancreas, you take the involved pancreas with the stomach. Since the involvement runs through the body and tail, the matching pancreatic resection is a distal pancreatectomy, combined with a partial (subtotal) gastrectomy for the antral primary.

Why not the others. Simply closing up assumes inoperability, but there is no metastasis and the lesion is resectable, so abandoning surgery is wrong. Antrectomy with vagotomy is ulcer surgery and would ignore the pancreatic extension. Tacking on a splenectomy is only justified when the spleen or its hilar nodes are involved, which the stem does not describe, so it adds morbidity without benefit here.

en bloc R0 resection = partial gastrectomy + distal pancreatectomy

Partial gastrectomy and distal pancreatectomy

Quick Tip: Resectable cancer invading a neighbour means en bloc removal of both, no more than needed.

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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:

Run each claim through what we know about gut neuroendocrine tumours and find the odd one out. Carcinoids favour the GI tract, behave indolently, and have characteristic site and sex patterns.

Small bowel plus appendix making up roughly 60% of GI carcinoids fits the classic distribution, so that statement holds. The greater-than-60% five year survival also fits, because these are slow, well-differentiated tumours with a generally favourable outlook. The female excess of appendiceal carcinoids is a recognised teaching point as well.

That leaves the claim that the rectum is spared. This is plainly false: the rectum is in fact a leading site for carcinoids and they are often found by chance during colonoscopy or proctoscopy. Because the rectum is commonly involved rather than spared, this is the incorrect statement the question is hunting for.

small bowel + appendix ≈ 60%, 5-yr survival > 60% are correct; rectum is NOT spared.

Rectum is spared

Quick Tip: Which option claims a common carcinoid site is left out entirely?

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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:

Picture a collar of pancreas wrapped around the descending duodenum, pinching it. The treatment logic is simple: relieve the blockage while leaving the encircling pancreatic tissue undisturbed.

Cutting through the ring is tempting but dangerous, because it can trigger pancreatitis, a pancreatic fistula or duodenal leak, and it may not even fully open the lumen. So surgeons do not divide the annulus.

Instead they create a bypass that carries food past the constricted segment. Either a duodenoduodenostomy or a duodenojejunostomy achieves this; this item flags duodenojejunostomy as the chosen bypass. A standalone Roux-en-Y loop is a reconstructive trick used elsewhere and is not the standard named answer for the annular ring.

Goal: bypass the obstruction, do not divide the ring

Duodenojejunostomy

Quick Tip: Bypass the ring, never cut it.

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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:

Start from the biochemistry. Spells of giddiness and pain that go with a fasting glucose of 40 mg% and a high insulin level point straight to endogenous hyperinsulinism, and the small enhancing pancreatic nodule names the culprit as an insulinoma. The clinical story is essentially Whipple's triad.

The key facts that drive management are that insulinomas are typically tiny, single, and benign in about nine of ten cases. An 8 mm well-defined head lesion is the textbook benign insulinoma.

For such a lesion you want the least destructive curative operation, which is enucleation: shell out the tumour and keep the surrounding pancreas. A Whipple resection would needlessly sacrifice the duodenum and pancreatic head for a benign nodule. Radiotherapy adds nothing to a curable benign tumour. Streptozotocin is reserved for malignant, spread-out islet cell disease, which this

patient does not have.

small + benign + localized insulinoma ⇒ enucleation

Enucleation

Quick Tip: Small benign insulinoma wants the most tissue-sparing operation.

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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:

The decision hinges on the patient being young and fit. For complete (full-thickness) rectal prolapse, surgeons choose between an abdominal approach and a perineal approach, and the durable, low-recurrence abdominal route is favoured whenever the patient can tolerate it.

So in this healthy young man the operation of choice is abdominal rectopexy, in which the rectum is freed and anchored to the sacrum to abolish the prolapse and its recurrence.

The perineal Delorme operation is the alternative for frail or elderly patients or limited prolapse, and it carries a higher recurrence rate, so it is not picked for a young fit man. Plain anterior resection is not the standard prolapse operation. Goodsall is a rule for tracing anal fistulae and is simply an unrelated distractor here.

young and fit ⇒ abdominal (rectopexy), most durable

Abdominal rectopexy

Quick Tip: Young and fit favors the durable abdominal route.

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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:

Anchor on what each Strasberg letter means and match the lesion. The scheme runs A through E for post-cholecystectomy biliary injuries, from trivial leaks up to major duct transections.

A cystic duct stump that gives way, a cystic duct blow out, is a bile leak from a small duct that is still connected to the rest of the biliary tree. That definition is exactly Strasberg Type A, which also includes leaks from small ducts of Luschka in the liver bed.

The other types do not fit. Type B is an occluded aberrant right sectoral duct, Type C is that same kind of aberrant duct transected and leaking, and Type D is a partial side-wall injury of the common duct. The big circumferential common-duct injuries are Type E. None of these describes a simple cystic stump leak.

cystic duct stump leak (in-continuity minor duct) = Type A

Type A

Quick Tip: A leak from a minor duct still in continuity is the lowest grade.

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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:

The aim after sewing in the new liver is a leak-free, physiological route for bile to reach the bowel. The cleanest solution is to join the donor bile duct straight onto the recipient bile duct, an end-to-end duct-to-duct repair, because it keeps normal anatomy, retains the sphincter of Oddi, and leaves the duct reachable by ERCP for future intervention.

If the recipient's own duct cannot be used, such as in sclerosing cholangitis or with a marked size difference, surgeons switch to a Roux-en-Y choledochojejunostomy, bringing up a defunctioned jejunal limb to the donor duct.

The option that lists both the duct-to-duct repair and the Roux-en-Y alternative therefore covers standard practice. Hooking the duct to the duodenum is not done, a simple jejunal loop without a Roux limb invites reflux cholangitis, and routine temporary external drainage followed by delayed surgery is not the

default plan.

first choice: duct-to-duct; backup: Roux-en-Y hepaticojejunostomy

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.

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193. Most common cysts of the spleen are:

(A) Hydatid cyst

(B) Dermoid cyst

(C) Pseudocyst

(D) Lymphangioma

Correct Answer: (A) Hydatid cyst

SOLUTION:

Think about splenic cysts by their lining. A true cyst has a wall lined by epithelium or by a parasite; a false cyst (pseudocyst) has no lining and usually appears after blunt trauma. The question asks for the single most common variety. Globally, the parasitic true cyst dominates because ***Echinococcus granulosus*** infection is widespread in livestock-rearing regions, and the spleen is a recognised site for hydatid disease after the liver and lung. That makes the hydatid cyst the most frequent splenic cyst encountered in practice and in exams. The pseudocyst is the leading false cyst and is the commonest one seen specifically after trauma, but it ranks behind the hydatid cyst when all splenic cysts are pooled together. Dermoid cysts and lymphangiomas are uncommon congenital lesions and can be set aside. So the answer is the hydatid cyst.

Hydatid cyst

Quick Tip: Think parasitic true cyst, the same organism that famously affects the liver.

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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:

Sort each operation by how it produces weight loss. Restriction means making the stomach hold less food; malabsorption means rerouting bowel so fewer calories are taken up. Vertical banded gastroplasty staples off a tiny upper pouch and is therefore restrictive. The adjustable gastric band squeezes the top of the stomach with a silicone ring and is also purely restrictive. The Roux-en-Y bypass relies mainly on its very small pouch to limit intake, so it behaves restrictively in this list. The duodenal switch is different in kind: it adds a long biliopancreatic limb that diverts bile and pancreatic juice away from most of the small intestine, so the dominant mechanism is malabsorption. Because the question asks which one is not a primary restrictive procedure, the duodenal switch is the answer.

Duodenal switch operation

Quick Tip: Which listed operation works mainly by bypassing small bowel rather than shrinking the stomach?

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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:

TRISS exists to give a number for the chance a trauma patient will live. To do that fairly it needs three different angles on the patient. First it needs how sick the body looks right now, which is the Revised Trauma Score (RTS); the RTS is computed from coma score, systolic pressure, and breathing rate, so those vitals are already folded inside it. Second it needs how bad the actual anatomical damage is, which is the Injury Severity Score (ISS) built from the worst-injured body regions. Third it needs age, because an older person is less likely to survive the same injury. Add those together and you get RTS plus ISS plus Age. The distractors that list GCS or BP on their own are double-counting items that already sit inside the RTS, so they are wrong.

$$\boxed{\text{RTS} + \text{ISS} + \text{Age}}$$

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:

The whole case turns on one CT finding: the contrast blush. A blush is a focal bright spot of contrast inside or around the spleen, and it means an artery is actively leaking or has formed a pseudoaneurysm. Start with the version that has no blush. Here the patient is young, stable, and FAST positive but the bleeding is not active, so the safest plan is to watch him closely without surgery, which is conservative management. Now switch to the version with a blush. The patient is still stable, but the blush proves bleeding is continuing, so simply observing him risks a sudden deterioration. At the same time we want to keep his spleen for immune protection. The procedure that does both, stopping the arterial bleed while sparing the organ, is splenic artery embolization done through angiography. Open surgery, whether removing the spleen or repairing it, is held back for patients who become unstable or who fail the non-operative route. So the management asked for in the blush scenario is angioembolization.

Splenic artery embolization

Quick Tip: Stable patient, but the contrast blush means active bleeding, so save the spleen without open surgery.

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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:

Focus on timing and anatomy. The oxygen level crashed the instant gas was pushed into the belly, and the patient already had a torn spleen, an organ packed with thin-walled veins. When you insufflate the abdomen for laparoscopy, you pressurise it with carbon dioxide. If a large vein is lying open, that pressurised gas can be driven straight into the venous blood. The gas bubbles flow back to the right side of the heart and lodge in the pulmonary arteries, where they form an air lock that stops blood from picking up oxygen, so the pO_2 falls at once. Compare the alternatives. A diaphragm tear hurts breathing but does not give a knife-edge desaturation locked to insufflation. Squeezing the inferior vena cava drops the blood pressure rather than the oxygen. A colon injury causes contamination, not sudden hypoxia. The clean link between making the pneumoperitoneum and the immediate oxygen drop, in a patient with open splenic veins, is a carbon dioxide gas embolism.

Gas embolism through splenic vessels

Quick Tip: Open vein plus pressurised CO2 the moment you insufflate equals gas in the circulation.

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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:

Read the case as a matching problem. The lab gives you the single hardest constraint: the blood culture grew a Gram-negative bacillus. That immediately rules out three of the four options, because two of them are viruses and one is a protozoan, and none of those would ever be reported as a Gram-negative rod on a bacterial culture. Only ***Salmonella*** is a Gram-negative bacillus. Now check that it also fits the story. Enteric (typhoid) fever caused by ***Salmonella typhi*** produces a stepwise fever lasting weeks together with an enlarged spleen, and the spleen is the favourite organ for a typhoid abscess, which is exactly what the hypoechoic lesion near the hilum represents. Everything lines up, so the causative organism is Salmonella.

Salmonella

Quick Tip: Only one of the four options is a Gram-negative bacillus, and it causes enteric fever.

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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:

The trick here is that the spleen is the enemy of the transfused platelets. In ITP the spleen both makes the antibody against platelets and chews up the antibody-coated platelets. So if you pour platelets into the patient while the spleen is still receiving blood, the spleen simply destroys them and you gain nothing. The natural solution is to wait until the spleen's blood supply is cut. The splenic artery is the dominant feeding vessel, so the moment it is ligated the spleen can no longer filter and destroy circulating platelets. That is the exact window to transfuse: the platelets now survive in the bloodstream and are available to help with clotting during the remainder of the operation. Giving them before incision or at incision wastes them on an active spleen, and waiting until the whole spleen is out is unnecessarily late because the job was already done at artery ligation. Hence platelets are best given right after the splenic artery is tied.

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.

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200. The most common cause of abdominal aortic aneurysm is:

(A) Atherosclerosis

(B) Trauma

(C) Syphilis

(D) Vasculitis

Correct Answer: (A) Atherosclerosis

SOLUTION:

Match each cause to where and how it damages the aorta. The abdominal aortic aneurysm sits in the infrarenal aorta and turns up in older smokers with high blood pressure and high cholesterol, the exact crowd that gets atherosclerosis.

Atherosclerotic disease weakens the wall by destroying its elastin and muscle, so the vessel slowly balloons out, making it the leading cause. The other choices each fail a test. Trauma tends to produce a false aneurysm rather than a true degenerative AAA and is rare. Syphilis has a signature: it strikes the ascending thoracic aorta, not the belly. Vasculitis is an unusual contributor. So by location, risk factors, and mechanism, atherosclerosis is the answer.

Atherosclerosis

Quick Tip: Think older smoker with an infrarenal aorta worn down by the same plaque that clogs arteries.

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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:

Start with the disease. A subarachnoid hemorrhage is sudden bleeding under the arachnoid layer, classically from an aneurysm rupture, and the patient presents with a thunderclap headache. To guide treatment timing and to tell families what to expect, neurosurgeons grade the severity on admission.

The scale built for exactly this purpose is the Hunt and Hess grading, which runs from Grade I to Grade V. Grade I patients are alert with only a slight headache and do well. Grade V patients are comatose with extensor posturing and have very high mortality. Because the grade tracks directly with survival and surgical risk, it answers the question about prognosis.

Eliminating the rest is straightforward. The Glasgow coma scale is a generic consciousness score and is not SAH specific. The Glasgow-Blatchford score is a gastrointestinal bleeding risk tool. The ICH score forecasts death in deep brain (parenchymal) hemorrhage, a different bleed entirely.

So the SAH specific prognostic grade is Hunt and Hess.

Hunt and Hess scale

Quick Tip: Think of the SAH specific clinical grade that runs I to V.

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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:

This is a find-the-odd-one-out question, so the trick is to spot the value that is actually healthy. Three options are classic threats to a small child and one is a normal lab figure.

Focus on the maternal hemoglobin of 11 gm%. In pregnancy, anemia is defined as a hemoglobin below 11 gm/dL. At exactly 11 the mother sits at the normal threshold and is not anemic, so her baby is not put at risk on that account. That marks it as the exception the question wants.

The other three are well known dangers for children under five. Poor nutrition

slows growth and cripples immune defenses. Being born too small carries the highest early risk of death and disease. Repeated infections, especially chest and gut infections, are the top killers in this age group worldwide.

Therefore the factor that does not damage the child is the normal maternal hemoglobin.

Maternal Hb of 11 gm%

Quick Tip: Which option is actually a normal value, not a risk?

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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:

Anchor on one rule of pediatrics: a hypertensive child almost always has a secondary cause, and at seven years a primary cause is unlikely. So the task is to pick the commonest secondary cause.

Among the kidney, vessel and endocrine causes of childhood hypertension, renal causes dominate, and within those the parenchymal disorders (chronic glomerulonephritis, reflux nephropathy, post-infectious scarring) form the largest single group. A normal urinalysis is reassuring but does not exclude them, because long-standing scarred kidneys often produce clear urine while still driving the pressure up.

Run through the distractors. True essential hypertension would be expected later, in teenagers, not at age seven. Polycystic kidney disease is only one entity and usually announces itself with flank masses or a family history. Coarctation of the aorta would give upper-limb hypertension with diminished or delayed femoral pulses, a picture the question does not mention.

That leaves renal parenchymal disease as the best fit.

Renal parenchymal disease

Quick Tip: In young children most hypertension is secondary, and the commonest group is renal.

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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:

Read the imaging carefully. A swollen pelvis with thinned cortex but a completely normal ureter localizes the block to the pelviureteric junction. That single fact sets the diagnosis as PUJ obstruction.

The next question is whether to save or sacrifice the kidney. Split function sits at 19 percent, which is depressed but still meaningful. The accepted threshold for

abandoning a kidney is roughly 10 percent or less, so at 19 percent the organ deserves a reconstructive attempt rather than removal.

The operation that corrects PUJ obstruction and reconnects a wide, draining junction is the Anderson-Hynes dismembered pyeloplasty. It excises the stenotic segment and restores funnel-shaped drainage, giving the kidney its best chance to recover.

The alternatives fall away. Nephrectomy is for a kidney functioning under about 10 percent, not this one. A nephrostomy gives only temporary external drainage and cures nothing. Endopyelotomy performs poorly in children with grossly dilated systems. Pyeloplasty is therefore the definitive answer.

Pyeloplasty

Quick Tip: Dilated pelvis with a normal ureter equals PUJ obstruction, and 19 percent function is still worth saving.

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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:

The whole answer turns on two words: suspected perforation. The moment a gut perforation is on the table, the priorities flip to confirming free intraperitoneal air and to keeping any harmful contrast out of the belly.

The investigation that does both is a plain abdominal radiograph. An erect or left lateral decubitus film reveals air under the diaphragm or outlining the bowel wall, which confirms perforation. It is fast, requires no preparation and carries no risk to an unstable newborn, so it is the correct first step.

Now the contrast options. Barium is absolutely forbidden when perforation is feared, because barium spilling into the peritoneal cavity triggers a brutal chemical peritonitis with high mortality. That eliminates both the barium enema and the barium meal follow through. When contrast is genuinely required in this scenario a water-soluble dye is chosen instead.

Upper GI endoscopy simply does not diagnose neonatal volvulus or perforation and would be dangerous. So the plain X-ray wins.

Plain X-ray

Quick Tip: Suspected perforation means look for free air and never give barium.

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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:

Two findings carry this case: severe hypertension recorded in the arm and femoral pulses you cannot feel. When the pressure is high above and the pulses

vanish below, the aorta is narrowed somewhere between the two.

The classic congenital lesion that does this is coarctation of the aorta. The constriction lies just distal to the left subclavian artery, so the upper body runs hypertensive while the legs are underperfused, giving weak or absent femoral pulses and a radio-femoral delay. The malignant pressure of 200/140 can readily provoke a seizure, explaining the presentation.

Sort the others out. Takayasu disease can also cause a pulseless state, but it favors young adult women and a broad set of vessels, and it is not the textbook child answer here. Renal parenchymal disease raises blood pressure yet leaves femoral pulses intact. A grand mal seizure is merely the event itself and explains neither the pulse deficit nor the arm-to-leg pressure gap.

The unifying diagnosis is coarctation of the aorta.

Coarctation of aorta

Quick Tip: High arm BP plus absent femoral pulses equals a narrowed aorta.

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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:

Treat this as an enzyme-pathway puzzle. Epinephrine raises blood sugar by mobilizing liver glycogen, and the very last step of releasing glucose into the

blood is run by glucose-6-phosphatase. If giving epinephrine fails to lift the sugar, that final enzyme must be the one missing.

A deficiency of glucose-6-phosphatase is Von Gierke's disease, glycogen storage disease type I. Glycogen and glucose-6-phosphate pile up in the liver, so the organ enlarges, while the inability to free glucose leaves the child profoundly hypoglycemic during fasting. The non-response to epinephrine is the signature clue.

Discard the rest by their hallmark organs. Andersen's disease scars the liver into cirrhosis. Pompe's disease floods the heart and muscle, producing a thick, failing heart in infancy. McArdle's disease strikes skeletal muscle, giving cramps and fatigue on exertion. None reproduces the liver-plus-refractory-hypoglycemia combination.

So the answer is Von Gierke's disease.

Von Gierke's disease

Quick Tip: No sugar rise after epinephrine points to a glucose-6-phosphatase block.

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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:

The wording forces a careful distinction. After a febrile seizure two separate outcomes are tracked: another febrile seizure (recurrence) versus genuine epilepsy with unprovoked fits. This stem asks specifically about progressing to epilepsy.

The recognized predictors of later epilepsy are a triad: complex or atypical febrile seizures, an underlying neurodevelopmental abnormality, and a family history of epilepsy. Whenever one of these is present, the chance of true epilepsy climbs notably.

Early age of onset does not belong to that triad. A young age at the first febrile seizure predicts that febrile seizures will happen again, but it does not, on its own, push the child toward epilepsy. That mismatch makes it the exception the question is after, which is why option B is the answer.

The other three each carry a clear epilepsy link and therefore stay on the risk list. So the odd one out is early age of onset.

Early age of onset

Quick Tip: Separate the risk of recurrence from the risk of true epilepsy; early onset predicts recurrence.

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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:

Start by profiling the spell itself. We are told the staring episodes are short, recur several times daily, switch on and off suddenly, and leave the child fully normal in between with no warning sensation and no drowsiness afterward. That symptom signature points to one specific entity.

The classic disorder is childhood absence epilepsy, in which typical absence seizures produce momentary **3** to **10** second lapses of awareness. The child simply freezes mid activity, then resumes as if nothing happened. The electroencephalogram fingerprint is a generalized $\mathbf{3}\,\boldsymbol{Hz}$ spike and wave discharge, and forced hyperventilation in the clinic can reproduce an attack within seconds.

Now eliminate the rest. A grand mal event is dramatic and convulsive, with a fall, tonic stiffening, clonic jerks, and a sleepy confused recovery, so it cannot explain a quiet repeated stare. A complex partial seizure tends to run longer, is commonly preceded by an aura, often shows automatic movements such as chewing or fumbling, and ends in postictal confusion, all of which are explicitly absent here. Day dreaming is breakable by a touch or a call, does not begin abruptly as an automatic event, and would not occur dozens of times a day in a stereotyped way.

Because the spells are brief, abrupt in onset and offset, very frequent, and free of aura or postictal confusion, the only entity that fits is the typical absence seizure.

Absence seizures

Quick Tip: Brief, frequent staring with sudden onset and offset and no aura or postictal confusion equals 3 Hz spike and wave.

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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:

The trick in this question is to separate frequency from severity. It asks which agent causes the largest number of meningoencephalitis cases in children, so the answer should be whichever virus shows up most often in routine practice rather than the one with the worst prognosis.

That distinction matters because the most feared organism, herpes simplex virus, is actually the textbook answer for the commonest cause of sporadic fatal encephalitis and the one that demands urgent acyclovir. But its absolute case numbers are small. The everyday workhorse of aseptic meningitis and meningoencephalitis in kids is the non polio enterovirus group, namely the coxsackie and echo viruses. They spread by the faeco oral route, surge in late summer and early autumn, and make up the overwhelming majority of laboratory confirmed cases.

The remaining options are regional or historical. Arboviral disease such as Japanese encephalitis drives serious seasonal outbreaks in endemic zones but is not the global leader. Mumps once contributed meaningfully, yet widespread ***MMR*** immunization has pushed it down to a rare cause in vaccinated populations.

So when the metric is raw frequency rather than lethality, enterovirus wins.

Enterovirus

Quick Tip: Most common by frequency is enterovirus; HSV is the most common cause

of fatal sporadic encephalitis.

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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:

Picture the anatomy first. When there is a communication between the subarachnoid space and the air filled nose, sinuses, or middle ear, whether from a basilar skull fracture, surgery, or a congenital dural gap, the leaking cerebrospinal fluid betrays that a doorway has opened. Bacteria sitting in the upper airway can then march straight through that doorway again and again, which is why such patients suffer repeated bouts of meningitis.

The organism that exploits this route most often is ***Streptococcus pneumoniae***. Pneumococcus is a heavy coloniser of the nasopharynx, so whenever a CSF leak provides access, it becomes the predominant cause of recurrent post traumatic or anatomical meningitis.

Now weigh the others. Neisseria meningitidis drives community outbreaks and sporadic disease but is not the signature organism of leak related recurrence. Haemophilus influenzae type b was historically a major paediatric meningitis pathogen, yet conjugate vaccines have curtailed it and it does not own this particular scenario. Escherichia coli belongs to the neonatal period, where it is picked up around delivery, not to recurrent meningitis through a CSF fistula.

Putting mechanism and microbiology together, the recurrent leak setting points squarely at the pneumococcus.

Pneumococci

Quick Tip: CSF leak plus recurrent meningitis equals pneumococcus, the dominant nasopharyngeal coloniser.

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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:

Read the trajectory of this illness. A toddler has been running a purulent nose for a while, now spikes a fever, and develops a red, swollen, puffy eye while x rays light up the ethmoid sinus. The ethmoid air cells sit right against the medial wall of the orbit, separated only by a paper thin bone, so an ethmoid infection that pushes into the eye is the textbook setup for orbital cellulitis or a subperiosteal abscess.

The management hinge is anatomical. Is the swelling stuck in front of the orbital septum, or has pus tracked behind it into the orbit and possibly walled off as an abscess? You cannot answer that by looking at the eyelid alone, and a postseptal abscess may need urgent drainage.

That is exactly what a contrast ***CT*** of the orbit and sinuses delivers. It maps the sinus disease, distinguishes preseptal from orbital extension, and reveals any collection that the surgeon must evacuate, so it is the logical immediate next step.

The alternatives fall short. A urine culture is irrelevant to a sinus and orbit problem. A blood culture is reasonable supportive testing but is often sterile in a focal orbital infection and tells you nothing about whether an abscess exists. Repeating the conjunctival swab is pointless when the first grew nothing and the real pathology lies deep behind the eye, not on its surface.

CT scan

Quick Tip: Sinusitis with a swollen eye needs CT to tell preseptal from orbital spread and find an abscess.

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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:

Treat each clue as a vote. A young boy is the first hint, because the leading dystrophy of childhood is X linked and therefore strikes males. Proximal lower limb weakness plus a positive Gower's manoeuvre, where the child walks his hands up his thighs to push himself upright, tells us the pelvic girdle is weak. Bulky calves that feel firm are pseudohypertrophy from fat and fibrous tissue replacing muscle. Finally a creatine kinase soaring to around **10000** signals brisk

muscle fibre breakdown.

Every one of those votes lands on Duchenne muscular dystrophy, a defect of the dystrophin gene that presents in early boyhood with exactly this constellation and with CPK in the thousands.

Now dismiss the contenders. Spinal muscular atrophy is an anterior horn cell disease with floppiness, fasciculations, and tongue twitching, but it spares the calves from pseudohypertrophy and keeps CPK near normal. Myotonia congenita is a chloride channel disorder producing stiffness and trouble letting go after a grip, sometimes with a muscular build, yet without the relentless proximal weakness or the sky high enzyme. Myotonic dystrophy leans on myotonia, distal rather than proximal weakness, facial wasting, cataracts, and frontal balding, with only modest CPK rise, none of which matches a small boy with hypertrophied calves and a CPK of **10000**.

Duchenne muscular dystrophy

Quick Tip: Boy plus calf pseudohypertrophy plus Gower's sign plus very high CPK equals Duchenne.

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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:

Anchor the answer in what vitamin C actually does. Ascorbic acid powers the hydroxylation of proline and lysine residues, a step that lets collagen fibres cross

link and gain tensile strength. Strip away vitamin C and collagen production falters everywhere collagen is needed.

Bone is one such place. The osteoid, the unmineralized organic scaffold that osteoblasts secrete before calcium is deposited, is built chiefly from type I collagen. In scurvy that scaffold cannot be assembled properly, so the fundamental bone lesion is failure to form normal osteoid matrix. The mineral handling machinery is not the problem here, the protein framework is.

This is precisely where scurvy parts ways with rickets and osteomalacia. In those vitamin D or phosphate disorders the osteoid is manufactured fine but never gets calcified, so the defect is decreased mineralization, the mirror image of scurvy. Increased resorption is the language of parathyroid excess, not vitamin C lack. And the description of low bone mass with otherwise normal mineral and matrix simply restates osteoporosis, a quantity problem rather than the matrix quality problem of scurvy.

So the defining metabolic bone defect in scurvy is the breakdown of osteoid matrix formation.

Decreased osteoid matrix formation

Quick Tip: Vitamin C builds collagen, so scurvy fails to form osteoid matrix; rickets instead fails to mineralize.

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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:

The question wants the odd one out, the drug that has no place in juvenile myoclonic epilepsy and may even make it worse. To choose well, remember the core teaching point about generalized epilepsies.

JME is an idiopathic generalized epilepsy of teenagers, marked by jerky myoclonus soon after waking, generalized tonic clonic seizures, and occasional absences. The golden rule is that sodium channel blocking drugs designed for focal epilepsy can paradoxically aggravate generalized seizures, particularly myoclonic jerks and absences.

Run through the list. Valproate is the broad spectrum mainstay and first choice. Topiramate and zonisamide are also broad spectrum and are accepted options. Carbamazepine, by contrast, is a classic narrow spectrum sodium channel blocker, and it is notorious for worsening myoclonus and absences, which is why it sits on the avoid list for JME alongside phenytoin and similar agents.

That makes carbamazepine the drug not used.

Carbamazepine

Quick Tip: Sodium channel blockers like carbamazepine worsen myoclonus; valproate is the JME drug of choice.

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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:

Three signals are on the table: seizures, learning disability, and pale patches of skin. Together they describe a neurocutaneous disorder, and the specific colour of the skin lesion is the deciding clue.

Pale, depigmented macules, the so called ash leaf spots that fluoresce under a Wood's lamp, are the earliest cutaneous marker of tuberous sclerosis. That condition classically links seizures, intellectual impairment, and skin findings, later adding facial angiofibromas, a shagreen patch, periungual fibromas, brain cortical tubers, and renal angiomyolipomas. The hypopigmentation is the key that unlocks the answer.

The competitors all break on the skin finding. Neurofibromatosis gives hyperpigmented cafe au lait spots and neurofibromas, the reverse of pale macules. Sturge Weber announces itself with a port wine facial stain along the trigeminal nerve and underlying meningeal angiomas, not white patches. Linear epidermal nevus syndrome produces raised linear warty streaks rather than flat hypopigmented macules.

With hypopigmented macules driving the diagnosis, the picture is tuberous sclerosis.

Tuberous sclerosis

Quick Tip: Seizures plus mental retardation plus hypopigmented ash leaf macules equals tuberous sclerosis.

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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:

Think about what each duct needs to either form or regress. The Wolffian duct needs testosterone acting through a working androgen receptor to grow into the male internal tract. The Mullerian duct needs anti-Mullerian hormone (AMH) from Sertoli cells to make it shrink away.

In complete androgen insensitivity, the patient is 46,XY with intra-abdominal testes. Those testes do two things: they pour out AMH and they pour out testosterone. The AMH does its job, so the Mullerian duct disappears. But the androgen receptor is broken, so even high testosterone cannot reach the Wolffian duct, which then never matures and is absent. Result, neither set of internal ducts is present.

Now scan the distractors. If AMH alone were missing, the testes still produce androgens, so the Wolffian system would be present and the Mullerian system would persist, the opposite of what is asked. An FSH receptor defect hits ovarian follicle response, not duct anatomy. An ovotesticular disorder mixes ovarian and testicular tissue, giving variable duct development rather than total absence. Only androgen insensitivity wipes out both duct lines.

Mullerian gone via AMH + Wolffian gone via receptor failure

$$\boxed{\textit{Androgen insensitivity syndrome}}$$

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:

Solve this by separating the two clues. Clue one is timing, the periods stopped right after an abortion. Clue two is the FSH of 6 mIU/ml, which is squarely in the normal range.

Start with the FSH because it is a fast filter. A failing ovary loses its negative feedback and the pituitary drives FSH sky high, typically far above 40. A failing pituitary cannot secrete gonadotropins, so FSH drops to very low levels. A value of 6 sits in neither camp, so both the ovary and the pituitary are functioning normally. That eliminates options A and B in one stroke.

Now the history takes over. Instrumentation of the uterus during evacuation can strip the basal endometrium and let raw surfaces adhere, sealing the cavity. The hormones still cycle, but there is no responsive lining and no outflow, so menstruation stops. This is Asherman syndrome, also called intrauterine synechiae. Fresh pregnancy could in theory cause amenorrhea, but the stem is built around a normal FSH after a curettage, which is the textbook setup for adhesions, and a beta-hCG would quickly exclude pregnancy.

Normal FSH + post curettage amenorrhea ⇒ intrauterine adhesions

$$\boxed{\textit{Uterine synechiae}}$$

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:

Frame the choices as predictive versus retrospective and direct versus indirect.

Basal body temperature is the classic trap. The small temperature step up happens because progesterone is thermogenic, and progesterone only rises after the corpus luteum forms, so the chart tells you ovulation has already occurred. Useful for confirmation, useless for advance warning.

The ferning pattern and the Spinnbarkeit thread test both read cervical mucus, which thins and stretches under rising estrogen in the days before ovulation. They hint that ovulation is near, but they are subjective, depend on the examiner, and only give a rough window.

Hormonal study is the precise tool. The luteinizing hormone surge is the actual switch that releases the egg, and ovulation reliably follows it within about a day and a half. Measuring serum or urinary LH, alongside estradiol, lets you forecast the exact ovulatory day, which is why ovulation predictor kits are built around LH. Among the four, the objective hormone assay is the strongest predictor.

LH surge → ovulation in 24 to 36 hours

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:

Use the pubic hair as the decisive feature, since two options share the absent uterus plus normal breasts picture.

An absent uterus tells you the Mullerian ducts regressed, which occurs either in Mullerian agenesis or when AMH-secreting testes are present. Normal breasts tell you estrogen is working on the breast tissue. Those two clues alone fit both Mullerian agenesis and androgen insensitivity, so they cannot settle the answer by themselves.

The tie breaker is sexual hair. Pubic and axillary hair are androgen dependent. In androgen insensitivity, the receptor cannot read testosterone, so this hair is scanty or missing even though estrogen keeps the breasts normal. That single mismatch, good breasts but poor hair, is the textbook signature of androgen insensitivity.

In Mullerian agenesis the patient has functioning ovaries and intact androgen response, so pubic hair is normal, which the stem specifically denies. Turner syndrome keeps the uterus but gives streak gonads, short stature and poor

breasts, and Noonan syndrome does not produce this triad at all.

$$\textit{Absent uterus} + \textit{normal breasts} + \textit{scant hair} \Rightarrow \textit{blocked androgen action}$$

$$\boxed{\textit{Androgen insensitivity syndrome}}$$

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:

The question is about frequency, so think in terms of which type shows up most often in cervical cancer specimens.

Cervical carcinoma is fundamentally an HPV disease, specifically the high-risk oncogenic types. When you tally cases globally, two types dominate, type 16 and type 18, and between them they cover roughly seventy percent of all cervical cancers. The crucial detail is that type 16 alone accounts for about half of all cases, making it the clear single leader, while type 18 sits second and is more tied to the glandular adenocarcinoma subtype.

Mechanistically these high-risk types matter because their E6 and E7 proteins knock out the p53 and Rb tumor suppressor pathways, unleashing uncontrolled cell division. Types 33 and 35 belong to the high-risk family too, but they appear in only a small fraction of tumors, so neither comes close to type 16.

$$HPV\ 16 + HPV\ 18 \approx 70\%,\ with\ 16\ leading$$

$$\boxed{HPV\ 16}$$

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:

The leak is constant and started soon after pelvic surgery, so a fistula between the urinary tract and vagina is the working diagnosis. The first job is to prove the fistula and figure out where it opens.

The neatest bedside tool for this is the three swab dye test. You pack three swabs along the vagina from the top to the introitus, fill the bladder with a coloured dye, and then read which swab gets wet or stained. A blue stained upper or mid swab tells you the dye crossed from bladder into vagina, confirming a vesicovaginal fistula. If instead the topmost swab is soaked with clear, undyed urine, the leak is coming from above the bladder, pointing to a ureterovaginal fistula. Staining near the introitus suggests a urethral cause or stress leakage. So one cheap test confirms and localises at once.

The alternatives play supporting roles. A urine culture only addresses the dysuria

and possible infection. Cystoscopy directly visualises the fistula tract and is valuable when planning surgical repair, but it is invasive and comes later. An IVP is reserved for checking the ureters when a ureteric injury is suspected. None of these is the right first diagnostic step.

$$\textit{Dye in bladder} + \textit{swab staining pattern} \Rightarrow \textit{fistula confirmed and located}$$

$$\boxed{\textit{Triple swab test}}$$

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:

This is a find the wrong statement question, so verify each claim against partial mole facts and the odd one out is the answer.

Genetics first. A partial mole is classically triploid, carrying 69 chromosomes because two sperm fertilise one egg or a diploid sperm fertilises it. So the triploidy statement is correct.

Behaviour next. Compared with a complete mole, a partial mole carries a much smaller chance of turning into persistent gestational trophoblastic neoplasia, only a few percent. So the rare progression statement is correct.

Presentation next. Because there is fetal tissue and only patchy villous swelling, a

partial mole rarely looks like the dramatic grape cluster and instead usually turns up as a missed or incomplete miscarriage. So that statement is also correct.

That leaves the ultrasound claim. In reality the early sonographic signs of a partial mole are subtle and easily overlooked, so it cannot be reliably diagnosed on early scan, and the firm diagnosis usually comes from histopathology after evacuation. That makes the imaging statement the false one.

Triploid + low GTN risk + missed abortion are true; reliable early USG is not

Can be reliably diagnosed by USG in early gestation

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:

The stem lists four findings and asks which one does NOT flag progression of a complete mole to an invasive mole, so confirm the three real markers and the leftover is the answer.

The cleanest marker is the hCG trend. After a mole is evacuated, the hormone should fall steadily to zero. If the level stalls and plateaus across several weekly readings, trophoblastic tissue is still active and invading, so a plateau in hCG is a true warning sign.

Uterine size is the second marker. A uterus that stays enlarged or grows again after evacuation reflects retained, invasive trophoblast rather than normal involution, so this too signals conversion.

Theca lutein cysts are the third marker. These ovarian cysts are driven by high hCG, so their persistence means hCG remains elevated and disease continues, again pointing to invasive mole.

That leaves the suburethral nodule, which is not a standard indicator of a complete mole turning invasive. It is therefore the exception the question wants.

Plateau hCG + persistent uterine enlargement + persistent theca lutein cysts indicate conversion; the nodule does not

$$\boxed{\textit{Suburethral nodule}}$$

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:

Start from what the smear actually proves. A PAP report of CIN III / HSIL is a flag from a screening test, not a final diagnosis. The lesion could be purely

intraepithelial or could already harbour early invasion, and a smear cannot distinguish these. So the management chain is: abnormal smear, then a diagnostic confirmation, then treatment.

The confirmation step in cervical disease is always colposcopy. Under magnification the clinician applies acetic acid and Lugol iodine, identifies the worst area, and obtains tissue. For a high grade lesion the most efficient way to do this is an excisional biopsy that also functions as treatment, which is LEEP. The loop cuts out the transformation zone and yields an intact specimen that the pathologist can examine for any invasive focus.

Why not the others? Removing the whole uterus for a lesion that has not invaded is over-treatment and discards the very tissue you need to read. Cryotherapy freezes the surface and destroys it without producing a specimen, which is unacceptable when you must exclude invasion in HSIL. Cold knife conization is the answer only in special situations such as a lesion running up the endocervical canal, an unsatisfactory colposcopy, or suspected microinvasion, so it is not the default next move.

Hence the immediate, single best step that both diagnoses and treats is colposcopic evaluation followed by LEEP.

Colposcopy and LEEP

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:

The whole point of a sentinel node biopsy is to find the single node that a tumour drains to first. Sample only that node: if it is clean, you spare the patient an extensive nodal clearance and the complications that follow. The technique works best where lymphatic drainage is orderly, accessible, and predictable.

Among gynaecological cancers, vulvar carcinoma fits this description best. The vulva drains to the inguinofemoral nodes, which are superficial and easy to map with patent blue dye plus a radioactive tracer. The alternative, a complete inguinofemoral lymphadenectomy, is notorious for wound dehiscence, infection, and chronic lower limb lymphoedema. Therefore a method that lets the surgeon avoid the full dissection in node negative cases offers a large benefit, and for early, single focus vulvar cancer the sentinel approach is validated and recommended.

The remaining options are less suited. Endometrial cancer has deeper pelvic and para-aortic drainage that is harder to predict, so sentinel mapping became routine only after this exam era. The cervix and vagina have rich, multidirectional lymphatics, which lowers the accuracy of identifying one true sentinel node.

So the malignancy where sentinel node biopsy is classically and most usefully applied is carcinoma of the vulva.

Carcinoma vulva

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:

Frame PCOS by its dominant endocrine state: women do not ovulate regularly, so they make plenty of oestrogen from follicles but little or no progesterone. The result is high androgens and oestrogen that is never opposed by progesterone. Almost every long term complication of PCOS flows from this.

Continuous oestrogen without progesterone keeps the endometrium proliferating, which is exactly the setup for endometrial hyperplasia and cancer, so endometrial carcinoma belongs to PCOS. The syndrome is also a textbook cause of insulin resistance and the metabolic syndrome, and the high insulin feeds back to raise ovarian androgen output. A weaker but recognised link to ovarian carcinoma is also described.

Now look at osteoporosis. Bone loss of the osteoporotic type is driven by a lack of oestrogen, which is why it appears after menopause and in hypothalamic amenorrhoea. PCOS patients sit at the opposite end: their oestrogen is normal or high. With oestrogen present, the bone is protected, so osteoporosis is not part of the PCOS spectrum.

The item that does not fit is therefore osteoporosis.

Osteoporosis

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:

The trick here is the word specific. Two markers point toward an open neural tube defect, but they do not perform the same job. Alpha fetoprotein is the screening marker: when the fetal neural tube is open, AFP spills into amniotic fluid and maternal serum, so maternal serum AFP goes up. The problem is that AFP also rises for many unrelated reasons, such as gastroschisis, twins, miscalculated dates, or fetal death, so a high value cannot prove an NTD.

Acetylcholinesterase is the confirmatory marker. It is an enzyme of nervous tissue, so it leaks into the amniotic fluid only when neural tissue is genuinely exposed, as in an open spina bifida or anencephaly. Because it is tied specifically to exposed neural tissue, detecting it in amniotic fluid is much more specific for an open neural tube defect than AFP. In practice, a raised maternal AFP is followed up by an amniotic AChE assay to confirm.

The other two choices do not belong. Pseudocholinesterase reflects liver synthesis and drug metabolism, not neural defects. HCG is a placental hormone used in Down syndrome screening, with no role in confirming NTDs.

Therefore the most specific marker is acetylcholinesterase.

Acetylcholinesterase

Quick Tip: AFP screens, but which neural enzyme in amniotic fluid actually confirms an open NTD?

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229. AFP (alpha fetoprotein) is raised in:

(A) Teratoma

(B) Yolk sac tumour

(C) Choriocarcinoma

(D) Dysgerminoma

Correct Answer: (B) Yolk sac tumour

SOLUTION:

Solve this by pairing germ cell tumours with the protein each makes. Yolk sac tissue makes alpha fetoprotein. Trophoblast tissue makes beta-HCG. Once you hold those two facts, the options sort themselves out.

The yolk sac tumour, also called the endodermal sinus tumour, is by definition built from yolk sac differentiation, and its histological signature, the Schiller-Duval body, mirrors that. Naturally, then, it pours out AFP, and clinicians use serum AFP to confirm the diagnosis and to track response to treatment.

Run the others through the same rule. Choriocarcinoma is trophoblastic, so its marker is beta-HCG, not AFP. Dysgerminoma, the ovarian counterpart of seminoma, typically raises LDH and sometimes placental alkaline phosphatase, but it does not raise AFP. A mature teratoma on its own does not secrete AFP unless it harbours yolk sac elements, so it is not the clean single answer the question wants.

That leaves the yolk sac tumour as the AFP producing tumour.

Yolk sac tumour

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:

The recommended amount of weight a woman should gain in pregnancy is built around a handful of maternal characteristics. The anchor is where she started: prepregnancy weight or BMI sets the target range, with underweight women advised to gain more and overweight women advised to gain less. So that option clearly belongs.

Two more background factors shape the picture. Socioeconomic status influences diet quality, food security, and the actual weight a woman puts on. Ethnicity feeds into body build and the population specific ranges used in guidelines. Both are standard determinants of gestational weight gain.

Smoking is different in kind. Its main effect in pregnancy is to restrict fetal growth and reduce birth weight; it is a harmful exposure, not a planning variable that the recommended maternal weight gain is calculated from. It does not feature in the list of factors that set the weight gain target, so within this question it is the exception.

Hence the factor that does not determine gestational weight gain here is smoking.

Smoking

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:

Tackle this by going to the dividing membrane on ultrasound, since that is where chorionicity is decided. Look at the spot where the membrane attaches to the placenta. If placental tissue pushes up into the membrane and forms a triangular tongue, that is the twin peak or lambda sign, and it tells you the pregnancy is dichorionic. A flat, right angle junction with no wedge is the T sign of a monochorionic pregnancy. So the statement that the twin peak sign indicates dichorionicity is correct.

Now test the rest. Same gender fetuses do not rule out dichorionicity, because dizygotic twins of the same sex are common and even monozygotic twins can be dichorionic if splitting happened early, so that statement fails. A thick separating membrane signals dichorionicity, since it is made of two chorions plus two amnions, whereas a monochorionic membrane is thin, so calling it a monochorionic feature is wrong. And chorionicity is assessed most accurately in the first trimester around 10 to 14 weeks, not only after 16 weeks, so that statement is also false.

Only one statement survives, and it is the twin peak sign in dichorionicity.

Twin peak sign is seen in dichorionicity

Quick Tip: Lambda or twin peak sign means a wedge of placenta, which means two chorions.

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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:

The picture is a pregnant woman in late gestation with severe itching, classically of the palms and soles, and a blood test showing raised bile acids. This is intrahepatic cholestasis of pregnancy, and the real concern is not the mother's discomfort but the fetal risk from high bile acid levels. So the ideal drug is one that brings bile acids down.

Ursodeoxycholic acid does exactly that. It promotes bile flow and displaces the toxic, hydrophobic bile acids with a more harmless one, which relieves the itch, improves the liver enzymes, and is regarded as safe to use in pregnancy. For all these reasons it is the accepted first line and treatment of choice.

The other options fall short. Cholestyramine only mops up bile acids in the gut, helps the itch less reliably, and can interfere with fat and vitamin K absorption, raising bleeding risk, so it is a backup at best. Corticosteroids and antihistamines may blunt symptoms a little but do not correct the underlying bile acid load, so

neither is the drug of choice.

Therefore the treatment of choice is ursodeoxycholic acid.

Ursodiol (ursodeoxycholic acid)

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:

Group the choices by the kind of risk each one poses. Three of them are structural or vascular problems that cannot tolerate the volume and output rise of pregnancy, and one is simply a rhythm disorder.

Eisenmenger physiology means a reversed shunt driven by pulmonary hypertension; when systemic resistance drops in pregnancy, more deoxygenated blood crosses right to left and the mother becomes profoundly hypoxic. Severe pulmonary hypertension on its own behaves the same way because the lung vasculature is fixed and cannot offload the extra blood volume. Both sit at the top of the maternal mortality tables, near 30 to 50 percent, so pregnancy is strongly advised against.

Coarctation of the aorta is a mechanical obstruction; the surge in cardiac output and the hormonal softening of the aortic wall raise the danger of dissection or rupture, so it too is listed as a contraindication.

WPW is different in nature. It is an accessory-pathway tachyarrhythmia, not a fixed plumbing defect. It responds to antiarrhythmics or catheter ablation and does not cap the heart's ability to raise output, so pregnancy is permitted.

The one that is not a contraindication is therefore WPW syndrome.

WPW syndrome

Quick Tip: Three options fix or strain cardiac output; one is just an arrhythmia.

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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:

Think of the problem in two filters: safety in pregnancy first, then convenience and cure rate.

Safety filter: the tetracyclines (tetracycline, doxycycline) are dropped at once because they deposit in fetal teeth and bone and are banned in pregnancy. Penicillin, when it appears, is irrelevant here because it does not reliably clear the

intracellular Chlamydia organism. That leaves the macrolides as the safe choices.

Convenience and cure filter: erythromycin works and is safe, but the course is long and the stomach upset is bad, so women often stop it early. Azithromycin clears Chlamydia with a single 1 g dose, which is far easier to take and gives high cure rates, so guidelines name it the drug of choice whenever it is on the list.

So in the set that includes azithromycin, azithromycin wins. In the set that swaps azithromycin out for penicillin, the safe and effective remaining macrolide, erythromycin, is the answer instead.

Azithromycin (Erythromycin in the no-azithromycin version)

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:

The key idea is the fetal swallowing pump. The fetus normally drinks large volumes of amniotic fluid every day; anything that blocks the mouth-to-gut route lets fluid pile up and gives polyhydramnios, while anything that cuts urine output dries the sac and gives oligohydramnios.

Run each option through this rule. Posterior urethral valves block the urinary tract, so less urine reaches the sac, which means low fluid, the opposite of what is asked. Bladder exstrophy is a surface defect of the bladder wall and does not

interfere with swallowing or with the bulk of urine production, so it is not tied to high fluid.

A diaphragmatic hernia can sometimes raise fluid by kinking the displaced stomach, but it is not the cleanest match among the choices. Cleft palate directly disrupts the swallowing mechanism: the fetus cannot clear the fluid it should be drinking, and the volume rises. That is the textbook link with polyhydramnios.

So the condition tied to polyhydramnios here is cleft palate.

Cleft palate

Quick Tip: Polyhydramnios follows impaired fetal swallowing; oligohydramnios follows poor urine output.

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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:

Nuchal translucency is the thin layer of fluid behind the fetal neck measured in the late first trimester. When that layer is thick, the test is flagging a higher chance of aneuploidy, and the diagnosis examiners pair it with most often is trisomy 21.

Why Down's syndrome: increased NT is the cornerstone ultrasound component of the combined first-trimester screen for trisomy 21, used together with PAPP-A and free beta-hCG. It is the single most frequently tested association.

Turner's syndrome does enlarge the neck fluid, but it tends to do so as a frank cystic hygroma, a different and bigger picture than a simple raised NT, so it is not the cleanest fit. Hydrocephalus is a later-pregnancy finding of enlarged ventricles, and skeletal dysplasias are recognised by short or abnormal bones rather than by NT, so neither belongs to the standard NT marker list.

The expected answer is therefore Down's syndrome.

Down’s syndrome

Quick Tip: Increased nuchal translucency is the classic first-trimester marker of trisomy 21.

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237. Non-immune hydrops fetalis is caused by:

(A) CMV

(B) Parvovirus

(C) HSV

(D) HIV

Correct Answer: (B) Parvovirus

SOLUTION:

The clue is the word non-immune, which removes Rh and other blood-group antibody causes and points toward infection, cardiac, or chromosomal causes. Among the four viruses listed, only one has a direct mechanism that produces hydrops, and that is Parvovirus B19.

Mechanism in plain terms: Parvovirus B19 has a strong tropism for the red-cell precursors in the fetal bone marrow. It shuts down their division, the fetus stops making red cells, and a deep anaemia develops. The struggling fetal heart then pumps harder to compensate, tips into high-output failure, and fluid leaks into

the skin, abdomen, and chest, producing hydrops.

The other viruses do not fit. CMV harms the fetus mainly through brain calcifications, small head, and poor growth, not through this red-cell wipeout. HSV and HIV are not standard causes of non-immune hydrops at all.

So the answer is Parvovirus.

Parvovirus

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:

Sort the four statements into proven indications versus the one that the evidence rejects. Three describe situations where antibiotics genuinely help, and one describes blanket prophylaxis that trials have shown to be useless.

The odd one out is giving antibiotics simply because a woman has threatened preterm labour with her membranes still intact. The ORACLE II study tested exactly this and found no improvement in outcomes, plus a hint of long-term harm to the children. Routine prophylaxis with unruptured membranes is therefore not recommended, which makes that statement incorrect.

The other three are accepted practice. Bacterial vaginosis, even without symptoms, is linked to preterm birth, so metronidazole is appropriate. Asymptomatic bacteriuria in pregnancy is always treated because it can progress to pyelonephritis and trigger preterm labour. And once the membranes rupture early (PPROM), antibiotics such as erythromycin prolong the pregnancy and cut infection rates.

So the statement that is not correct is the one advising prophylaxis with unruptured membranes.

Antibiotic prophylaxis even with unruptured membranes

Quick Tip: Recall ORACLE II: prophylaxis with intact membranes gives no benefit.

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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:

The single most discriminating detail is meconium-stained liquor at only 34 weeks. Meconium passage is uncommon this early, and when it accompanies ruptured membranes and infection it points the examiner toward Listeria monocytogenes.

Listeria reaches the pregnant woman through food such as soft cheeses, unpasteurised milk, and cold cuts. It then crosses to the fetus and placenta, setting off chorioamnionitis and preterm labour, and it is specifically associated

with that early meconium staining of the amniotic fluid.

The alternatives do not match an acute membrane-rupture presentation. Toxoplasmosis and CMV are congenital infections recognised by brain calcification, eye lesions, and growth restriction discovered over time, not by sudden meconium-stained PROM. Herpes infects the newborn mainly during vaginal delivery, causing skin, eye, and neurological disease rather than this antenatal picture.

Putting the food-borne route, the chorioamnionitis, and the tell-tale early meconium together, the organism is Listeria monocytogenes.

Listeria monocytogenes

Quick Tip: Early meconium-stained liquor with membrane rupture is the classic Listeria clue.

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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:

Picture the standard AMTSL bundle and check each option against it. The accepted bundle is: give a uterotonic such as oxytocin right after birth, deliver the placenta with controlled cord traction while guarding the uterus, and then keep the uterus firm with gentle massage. Those three appear in the list as options A, D, and C and are all correct steps.

The one that no longer belongs is immediate clamping, cutting, and ligation of the cord. Current WHO practice favours delayed cord clamping, waiting one to three minutes, because it improves the newborn's iron stores and lowers the risk of anaemia. Routine immediate clamping is therefore not part of recommended active management, which makes option B the answer for part (a).

In the second recall, the listed elements oxytocin, controlled cord traction, and uterine massage are all genuine AMTSL steps; the routine separate ergometrine injection is the relative odd one out in that framing.

So the intervention not recommended in part (a) is immediate clamping and ligation of the cord.

Immediate clamping, cutting and ligation of cord

Quick Tip: Modern AMTSL favours delayed, not immediate, cord clamping.

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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:

Shoulder dystocia happens when the baby's head is born but the front shoulder catches on the mother's pubic bone, so the rest of the body will not follow with gentle pulling. Doctors run through a set drill to release that shoulder. The first move is McRoberts, where the mother's legs are bent sharply up toward her chest; this tilts the pelvis and opens space. Next, an assistant presses just above the pubic bone (suprapubic pressure) to nudge the trapped shoulder sideways and down. If that fails, the Woods corkscrew is tried, where the operator places fingers behind the baby's shoulders and rotates the body like a screw to bring a shoulder out from under the bone. All three of these belong to the shoulder dystocia toolkit. The odd one out is the Mauriceau-Smellie-Veit maneuver. That technique is for a breech birth: the baby comes bottom first and the head is the last part to deliver, so the operator supports the body on the forearm and applies controlled flexion to the after-coming head. It has nothing to do with a stuck shoulder in a head-first birth. Therefore the maneuver NOT used in shoulder dystocia is the Mauriceau-Smellie-Veit maneuver.

Mauriceau-Smellie-Veit maneuver

Quick Tip: Which listed maneuver actually belongs to vaginal breech delivery of the after-coming head?

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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:

A baby can catch HIV from its mother during pregnancy, at delivery, or through breastfeeding, and the prevention strategy targets each of those windows. The cornerstone is treating the mother with combination antiretroviral drugs (HAART) so her blood carries as little virus as possible; a suppressed viral load is what protects the child most. When the mother's virus level stays high or is not known near term, a planned Caesarean before labour starts keeps the infant from travelling through the birth canal and limits its contact with maternal blood, which lowers the chance of transmission. Around the time of birth, giving the mother Zidovudine through a vein, plus dosing the newborn afterward, adds another layer of protection during the most risky hours. All three of these are genuine PMTCT steps. The choice that does not fit is "avoid ergometrine in the third stage of labour." Ergometrine is simply a drug to tighten the womb and stop bleeding after delivery; skipping it does nothing to stop the virus from reaching the baby. Any caution around ergometrine in HIV care is about a drug interaction with protease inhibitors, not about transmission. So the intervention that is NOT part of preventing mother-to-child HIV spread is avoiding ergometrine.

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.

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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:

Contact lens wearers are a classic group for Pseudomonas keratitis because this bug loves moist plastic surfaces. The key word in the question is mechanism of resistance, so we must explain why the organism shrugs off several antibiotics, not why the patient caught it. Pseudomonas aeruginosa is famous for building biofilms: it secretes a sticky sugar-rich slime and lives inside it as a community, and the lens and corneal surface give it a perfect scaffold. Once that slime layer forms, antibiotic molecules cannot diffuse in well, the cells inside slow their metabolism, and a hardy fraction simply rides out the drug. The net result is a population that behaves as resistant to many agents together, which is exactly what multidrug resistance looks like clinically. Now compare the other choices. Poor lens hygiene and heavy use of topical antibiotics may explain how the infection started or what selected for tougher bugs, but neither is the structural mechanism that makes the strain resist drugs. Picking up genes from neighbouring commensals is a recognised way bacteria gain resistance in general, yet it is not the signature, lens-linked Pseudomonas mechanism being tested here. The biofilm is the unifying answer for why this organism is hard to kill on a contact lens. So the best explanation is the bacterium's ability to make biofilms.

Ability of Pseudomonas to produce biofilms

Quick Tip: Think about the slimy matrix this organism builds on plastic lens surfaces.

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244. Endophthalmitis involves inflammation of all of the following, except:

(A) Sclera

(B) Uvea

(C) Retina

(D) Vitreous

Correct Answer: (A) Sclera

SOLUTION:

The trick in this question is the precise meaning of endophthalmitis versus panophthalmitis. Endophthalmitis is inflammation confined to the inner chambers and tissues of the globe, typically driven by infection that has reached inside the eye. That means the inflammation hits the vitreous gel, the aqueous, the uveal tract, and the neighbouring retina, all of which are intraocular structures. The sclera, however, is the tough white outer shell of the eyeball, an outer coat rather than an inner content. When the inflammatory process spreads outward to take in the sclera and the tissues around the eye, doctors no longer call it endophthalmitis; they call it panophthalmitis, which signals a far more severe, all-coat involvement. So among the listed structures, the uvea, retina, and vitreous all participate in endophthalmitis, but the sclera does not. The structure NOT involved in endophthalmitis is therefore the sclera.

$$\boxed{\text{Sclera}}$$

Quick Tip: Once the outer white coat is involved, the term changes to panophthalmitis.

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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:

This question lines up the main stromal corneal dystrophies and asks which is seen least often. A simple way to rank them is by frequency and by what they deposit. Granular dystrophy tops the list as the most common; it lays down crumb-like hyaline material in the stroma that lights up with Masson trichrome. Lattice dystrophy comes next; here the deposits are amyloid, arranged as fine branching lines that stain with Congo red, and type I is a familiar inherited pattern. Macular dystrophy sits at the bottom for frequency. It is inherited in an autosomal recessive manner, deposits abnormal mucopolysaccharides (glycosaminoglycans) that take up Alcian blue and colloidal iron, and clouds the cornea diffusely all the way to the edge, making it the most severe yet the rarest of the trio. Putting the choices in order, granular is commonest, lattice is in the middle, and macular dystrophy is the rare one. So the least common corneal dystrophy among the options is macular dystrophy.

Macular dystrophy

Quick Tip: Rank granular, lattice, and macular by frequency; the rarest deposits mucopolysaccharide.

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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:

The cherry-red spot is a retinal sign that appears when storage material piles up inside the retinal ganglion cells around the fovea. Those swollen, lipid-filled cells turn the surrounding retina milky and pale, but the very centre of the macula has almost no ganglion cells, so its thin floor lets the red colour of the choroid show through. The contrast creates a red dot ringed by a whitish halo. This sign is the hallmark of the sphingolipidoses that load ganglion cells with lipid. Tay-Sachs, a GM2 gangliosidosis, is the textbook example. GM1 gangliosidosis does the same by storing GM1 ganglioside in those cells. Niemann-Pick disease, particularly the acute neuronopathic type A, fills ganglion cells with sphingomyelin and likewise shows the cherry-red spot. The outlier here is Gaucher's disease. Although it is a lysosomal storage disorder, the deficient enzyme is glucocerebrosidase, and the glucocerebroside collects mainly in macrophages of the spleen, liver, marrow, and bone, sparing the retinal ganglion cells; so it does not produce a classic macular cherry-red spot. The printed key of "None" is wrong; the correct exception is Gaucher's disease.

Gaucher's disease

Quick Tip: Which lysosomal storage disorder loads macrophages of spleen and marrow, not retinal ganglion cells?

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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:

To answer this, recall how the pupillary light reflex is wired. Light entering an eye is sensed by the retina and sent up the optic nerve; the signal then crosses over so that shining a light in either eye normally makes both pupils shrink by the same amount. This sensing pathway is the afferent (input) side of the reflex, while the efferent (output) side travels in the oculomotor nerve to constrict the pupil. A relative afferent pupillary defect is purely an input-side problem, and it is brought out by swinging a torch back and forth between the two eyes. If one optic nerve is diseased, say from optic neuritis, that eye delivers a weaker light message. When the torch swings onto the bad eye, the brain registers less light, so instead of constricting, both pupils drift wider; that paradoxical dilation is the RAPD. Damage to the oculomotor nerve cannot cause this, because that nerve is the output cable; it produces a fixed, dilated pupil on the affected side rather than a relative afferent defect. Lesions further back, in the optic tract or lateral geniculate body, mostly create field cuts but, because of heavy fibre crossing, do not give the clean RAPD typical of optic nerve disease. The characteristic site for RAPD is therefore the optic nerve.

Optic nerve

Quick Tip: RAPD is an input-side defect found with the swinging flashlight test; which structure carries that afferent signal?

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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:

The whole question hinges on one unusual link: an eyelid that normally droops but pops open the moment the patient works the jaw. That ties together two different cranial nerves. The trigeminal nerve drives the chewing muscles (the pterygoids), and a branch of the oculomotor nerve drives the levator palpebrae, the muscle that raises the upper lid. In Marcus Gunn jaw-winking, there is an abnormal congenital cross-wiring so that whenever the jaw muscles fire, say on chewing or moving the jaw to the opposite side, the levator fires along with them and the droopy lid jumps up. This trigemino-oculomotor synkinesis is exactly what the vignette describes. The other options do not fit. Aberrant regeneration of the third nerve produces odd lid or pupil movements linked to eye movements, not to chewing. An abducent nerve palsy simply weakens outward gaze. A plain oculomotor palsy gives a drooping lid with a down-and-out, dilated eye, but the lid does not retract when the patient eats. The only condition where chewing lifts the ptotic lid is Marcus Gunn jaw-winking syndrome.

Marcus Gunn jaw-winking syndrome

Quick Tip: A trigeminal-oculomotor cross-wiring makes the drooping lid lift during jaw movement.

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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:

Otoacoustic emissions are echoes that the ear itself makes. A clinician places a small probe in the ear canal, sends in a click or tone, and a microphone picks up a

faint sound coming back out a few milliseconds later. The question is which cells create that returning sound.

The cochlea uses inner hair cells to convert vibration into nerve impulses, and these are the receptors for hearing in the strict sense. The outer hair cells do something different. They physically change their length in step with the incoming sound, pushing on the basilar membrane and sharpening the cochlear response. This active movement is what makes the human ear so sensitive and frequency selective.

Because outer hair cells move, they spill mechanical energy back toward the middle ear and out through the ear canal. That energy is recorded as the otoacoustic emission. The test therefore checks whether outer hair cells are alive and working, and it is the basis of automated newborn hearing screening since it needs no patient cooperation.

Inner hair cells feed the auditory nerve but do not generate the emission. The organ of Corti is the umbrella structure, not the precise source. A combined inner-plus-outer answer is incorrect because the emission is purely an outer hair cell phenomenon.

Outer hair cells

Quick Tip: Think about which cochlear cells are motile and act as the cochlear amplifier, the basis of newborn hearing screening.

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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:

Corneal transplantation succeeds far more often than other transplants, and the reasons trace back to two facts: the cornea has no blood vessels and the graft survives only if its endothelial pump is intact. These two facts let us sort the four options quickly.

The endothelium is a single layer that keeps the cornea dehydrated and clear. Before a graft is used, eye banks look at this layer with a specular microscope, which photographs the endothelial cells and gives a count and a measure of cell shape. A low count predicts graft failure, so this is the central quality check. That makes the specular microscopy statement the true one.

The idea that the entire globe is stored in tissue culture is incorrect. Eye banks remove a corneoscleral button and keep it in storage media; the intact eyeball is only used for short-term moist chamber preservation. The age limit statement is also false, since corneas from donors over 60 are routinely transplanted as long as the endothelial count is adequate. Finally, HLA matching is not mandatory for a standard graft because the avascular, immune-privileged cornea tolerates mismatched tissue; matching is reserved for vascularised high-risk beds.

So the only accurate statement concerns endothelial cell counting by specular microscopy.

Specular microscopy assesses 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:

This is a negative stem, so the task is to find the one wrong claim about sodium fluoride therapy in otosclerosis. Start from what the drug is meant to do. Active otosclerosis is a phase of bone breakdown and immature spongy bone formation around the stapes and cochlea. Fluoride is used to calm this activity down and harden the bone.

The first option says it inhibits proteolytic enzymes in the cochlea. That is correct; part of the rationale for fluoride is suppressing the lytic enzyme activity that drives otospongiosis. The third option says it should be avoided in chronic nephritis; that is also correct because fluoride clears through the kidneys and accumulates in renal impairment, so it is contraindicated there and in pregnancy. The fourth option ties treatment to a positive Schwartze sign, the pink flush over the promontory that marks active hyperaemic disease; since fluoride is given precisely for active otosclerosis, this too is true.

That leaves the claim that fluoride works by inhibiting osteoblastic activity. This is the false statement. Fluoride encourages bone formation and maturation rather than blocking the bone-forming osteoblasts; its inhibitory action is directed at the resorptive, osteoclastic and enzymatic side of the disease. Because the question asks for the exception, this wrong mechanism is the answer.

Acts by inhibiting osteoblastic activity (false)

Quick Tip: Sodium fluoride hardens bone and curbs resorption, so a statement that it blocks bone formation is the odd one out.

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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:

The Arden Index is a specific number that comes out of one electrophysiology test, so the trick is to recall which test reports a light-to-dark ratio. The electrooculogram is built around the standing potential between the front and back of the eye. When the patient sits in the dark this potential falls to a trough, and after a period in light it rises to a peak. Dividing the light peak value by the dark trough value and multiplying by a hundred gives the Arden ratio.

A healthy eye gives a ratio around 185 percent or higher. When the ratio drops well below this, it signals dysfunction at the level of the retinal pigment epithelium, which is the classic finding in Best vitelliform dystrophy where the EOG is markedly abnormal even when the ERG is normal.

The other choices do not generate this index. The electroretinogram describes retinal responses with a and b waves after a light flash. The visual evoked response measures signals at the visual cortex and is used in optic nerve disease. Perimetry is simply a map of the field of vision and involves no electrical potential ratio. So the Arden Index belongs only to the EOG.

EOG (Electrooculogram)

Quick Tip: It is a light peak to dark trough ratio of the standing corneo-retinal potential.

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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:

The stem asks which CSF leak statement is wrong, so each line must be checked for accuracy. The first claim places the commonest leak at the anterior skull base near the cribriform plate and fovea ethmoidalis. This holds true, since the bone there is paper thin and tightly fused to dura, making it the usual failure point in both spontaneous and post-traumatic leaks.

The second claim relies on beta-2 transferrin. This protein is essentially unique to cerebrospinal fluid, so finding it in a sample of nasal discharge confirms a CSF leak with very high specificity, and the statement is correct. The third claim uses intrathecal fluorescein, injected through a lumbar puncture so that the CSF turns fluorescent and the leak can be pinpointed under the endoscope; this is an accepted localisation technique and is also true.

The remaining option says gadolinium enhanced T1 MRI is the best test to localise the leak. This is the inaccurate statement. Bony defects are demonstrated best by high resolution CT of the skull base, and the fluid pathway by CT cisternography or heavily T2 weighted MR cisternography. Contrast enhanced T1 sequences are not the imaging of choice for tracing a CSF leak. Since the question wants the exception, this is the answer.

Gadolinium T1 MRI is best for localising the leak (false)

Quick Tip: For finding the leak site, think thin-section CT and cisternography, not contrast T1 MRI.

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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:

Hereditary retinoblastoma is the classic example of a germline tumour suppressor loss. Because one defective RB1 allele is present in every cell, only a single further mutation is needed to seed a tumour, so these individuals stay at risk for new cancers throughout life even after the eye disease is cured. The question asks which second cancer they are most likely to face.

The well established answer is osteosarcoma. Bone is the leading site of second primary malignancy in retinoblastoma survivors, and the risk climbs higher if the orbit was irradiated, although osteosarcoma also appears outside any radiation field. This consistent association is what makes option B correct.

Pinealoblastoma is a real RB1 link, seen as the third tumour in trilateral retinoblastoma, but it shows up in young children essentially together with the ocular tumours rather than as a later second cancer in a survivor, and it is far rarer than bone sarcoma. Renal cell carcinoma points to the von Hippel-Lindau

pathway and is unrelated to RB1. Chondrosarcoma is not a recognised RB1 second tumour. Weighing these together, osteosarcoma carries the highest risk.

Osteosarcoma

Quick Tip: RB1 carriers face lifelong second cancers, and the commonest one arises in bone.

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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:

The label acoustic neuroma is somewhat misleading because the lesion is neither truly acoustic in origin nor a neuroma. It is a schwannoma, a tumour of Schwann cells, and it forms on the vestibular component of the eighth cranial nerve. That is why the preferred name is vestibular schwannoma.

The eighth nerve splits into a cochlear part for hearing and a vestibular part for balance, the latter having superior and inferior divisions. Tumour growth nearly always begins on the vestibular side rather than the cochlear side. Within the vestibular nerves, the superior division is the site most often cited as the point of origin, related to the region where peripheral Schwann cell myelin meets central glial myelin and Schwann cells proliferate most readily.

Patients still present first with hearing loss and tinnitus, because the enlarging mass compresses the neighbouring cochlear fibres inside the narrow internal auditory canal, but this is a secondary effect rather than the source. The inferior

vestibular nerve can give rise to these tumours too, just less commonly, and the facial nerve is pushed aside rather than being the parent nerve. Putting this together, the superior vestibular nerve is the most frequent origin.

Superior vestibular nerve

Quick Tip: It is a vestibular schwannoma, and the upper vestibular division is the classic site of origin.

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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:

This negative stem asks for the one untrue claim about nasopharyngeal carcinoma. The disease starts high in the nasopharynx, classically in the fossa of Rosenmuller, and carries strong ties to Epstein-Barr virus and to dietary and ethnic factors seen in southern China and Southeast Asia.

Looking at the options, the bimodal age pattern is genuine; there is an early peak in young people and a larger later peak in middle age, so that statement stands. The presence of IgA antibodies to EBV is also real and clinically useful, since elevated anti-VCA and anti-EA IgA titres help detect and monitor the tumour. The histology statement is correct as well, because the tumour is fundamentally squamous, grouped by the WHO into keratinising, non-keratinising and undifferentiated forms, with the undifferentiated variety dominating in endemic regions.

The statement that fails is the one about surgery. Because the nasopharynx sits deep at the skull base where wide excision is impractical, and because the tumour responds extremely well to irradiation, radiotherapy is the mainstay, with concurrent chemotherapy for advanced stages. A radical nasopharyngectomy with neck dissection is not the primary treatment; surgery on the neck is only for nodal disease that persists after radiation. Therefore this surgical claim is the exception.

Nasopharyngectomy with radical neck dissection is the treatment of choice (false)

Quick Tip: The nasopharynx is deep and the tumour is radiosensitive, so the primary treatment is not surgery.

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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:

The question asks us to spot the one structure that does not belong to the extrinsic group of laryngeal attachments.

Think of the larynx as a box of cartilages. Anything that ties this box to a neighbour (the hyoid bone above or the trachea below) is extrinsic. Anything that ties two cartilages of the box to each other is intrinsic.

The thyrohyoid membrane reaches up from the thyroid cartilage to the hyoid bone, so it is extrinsic. The hyoepiglottic ligament holds the epiglottis to the hyoid, again reaching outside the box, so extrinsic. The cricotracheal ligament

drops from the cricoid down to the trachea, so extrinsic as well.

The cricothyroid membrane sits entirely between the cricoid and the thyroid cartilage. Both ends are laryngeal cartilages, so it connects the box to itself. That makes it intrinsic, and it is the odd one out.

Cricothyroid

Quick Tip: Extrinsic ties the larynx to the hyoid or trachea; a membrane between two laryngeal cartilages is intrinsic.

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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:

A bronchoscope is essentially a camera that travels inside the breathing tube. So the rule is simple: if a structure faces the airway lumen, the camera sees it; if it sits in the tissue outside the tube, the camera does not.

Going down the path, the cords guard the entrance to the larynx and are clearly visible. The trachea forms the corridor the scope travels through, so it is seen along its whole length. The carina splits the airway, and from there the scope reaches the lobar bronchi and the first segmental branches, all of which are luminal landmarks.

Subcarinal lymph nodes are a different matter. They lie buried in the

mediastinum, behind the wall of the airway, just under the carina. The light of the scope never reaches them. The only clue on plain bronchoscopy is a splayed or widened carina if the nodes are enlarged. To actually see or sample them, an ultrasound probe or a mediastinal approach is required.

Subcarinal lymph nodes

Quick Tip: The scope sees only the lumen. Nodes outside the airway wall stay hidden.

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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:

The clinical story is a textbook one. A toddler develops a seal like barking cough, noisy breathing on inspiration and respiratory distress over two or three days, with low grade fever. This is croup, or acute laryngotracheobronchitis, a viral illness usually driven by parainfluenza.

Now check each statement. The obstruction in croup sits at the subglottis, which is the tightest segment of a child airway, so saying symptoms come mainly from subglottic involvement is true. On imaging this subglottic swelling produces the classic steeple sign, and the airway above can balloon out as the hypopharynx dilates, so the radiology statement is true too. Epidemiology shows the disease favours boys, so that statement holds as well.

The trap is the treatment. Since a virus is to blame, an antibiotic does nothing for the swelling. Real treatment is supportive: keep the child calm, give humidified oxygen, use nebulised adrenaline when distress is significant, and give a single dose of dexamethasone to shrink the oedema. Antibiotics enter only if a bacterial complication is proven. So the claim that antibiotics are the mainstay is the wrong statement.

Antibiotics form the mainstay of treatment

Quick Tip: Barking cough and steeple sign mean viral croup. Steroids and adrenaline, not antibiotics, are the mainstay.

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260. Drug of choice in laryngeal stenosis is:

(A) Cyclophosphamide

(B) Doxorubicin

(C) Adriamycin

(D) Mitomycin C

Correct Answer: (D) Mitomycin C

SOLUTION:

The core issue in laryngeal stenosis is scar. After the airway is dilated or the scar is cut, fibroblasts rush back and lay down fresh collagen, so the narrowing returns. The ideal drug therefore is one that locally blocks fibroblasts.

Mitomycin C fits exactly. It is a streptomyces derived agent that alkylates DNA and powerfully inhibits fibroblast growth. Surgeons apply it topically on a soaked pledget to the freshly treated stenotic segment, which slows down scar reformation and improves the durability of dilation. That is its accepted role as the adjunct of choice in airway stenosis.

The other three options are systemic anticancer drugs. Cyclophosphamide is used for lymphomas and autoimmune disease, while doxorubicin and adriamycin are two names for the same anthracycline used in many solid and haematological cancers. None of them is placed topically to prevent airway scarring.

So the agent matched to the mechanism of stenosis is the one that locally curbs fibroblasts.

Mitomycin C

Quick Tip: Stenosis is a scarring problem. Pick the topical antifibroblast agent.

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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:

For surgery on the vocal cords the surgeon wants a beam that cuts cleanly and barely burns the tissue around the cut, because the larynx is tiny and the voice depends on intact mucosa.

The carbon dioxide laser wins on this. Its long **10,600** nm wavelength is soaked up almost entirely by tissue water, so the energy stops at the surface. The result is a precise incision with very little spread of heat sideways. Mounted on the operating microscope with a micromanipulator, it lets the surgeon vaporise papillomas, treat early cord cancers and divide webs with great control. That is

why it became the routine workhorse for laryngeal procedures.

The competitors serve other niches. The Nd:YAG beam goes deep into tissue, which suits debulking large tumours lower in the airway but is too aggressive for cords. Argon is mostly an eye and vascular laser. KTP handles vascular lesions and finds some use in phonosurgery, yet it has not displaced CO2 as the default.

So for everyday laryngeal surgery the answer is the deeply water absorbed, shallow cutting beam.

$\boxed{\text{CO2 laser}}$

Quick Tip: Water absorbs it strongly, so the cut is precise and shallow, perfect for vocal cords.

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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:

The way to settle this is to remember that the medial and lateral menisci behave oppositely because of how they are tied down.

The medial meniscus is locked to the joint by its firm attachment to the medial collateral ligament and the capsule. A structure that is held tight cannot move much, so it is the less mobile of the two. That immediately marks the claim that the medial meniscus is more mobile as untrue.

The flip side of being fixed is that it cannot dodge sudden twisting loads, so it tears more often. That confirms the medial meniscus is more commonly injured. The lateral meniscus, being almost a full circle and loosely held, slides freely and also drapes over a greater share of its side of the tibial plateau, so the coverage statement is correct. Finally, like all fibrocartilage, menisci are built mostly from type I collagen, so that statement is correct as well.

Only the mobility claim breaks the pattern, so it is the false one.

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.

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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:

The trick in this question is to read aging strictly, not to mix it up with osteoarthritis.

In an old but otherwise healthy joint, the cartilage cells slow down. They make fewer proteoglycans, so synthesis is down and the matrix ends up with less total proteoglycan. Those two statements describe aging correctly.

Proteoglycans are what pull water into cartilage, because their negative charges attract it. When their amount drops, the matrix can no longer hold as much fluid, so the water content of aged cartilage falls and the tissue becomes drier and stiffer. That makes the water statement true as well. Note this is the reverse of what happens in early osteoarthritis, where the matrix swells with extra water.

That leaves the claim about a rise in enzymatic degradation. Active enzymatic chewing of proteoglycans by metalloproteinases is the signature of osteoarthritis, a true catabolic disease, not of plain aging. In aging the deficit comes from making less, not from breaking down more. So this is the statement that does not belong.

Enzymatic degradation of proteoglycans is increased

Quick Tip: Separate aging from osteoarthritis. Surging enzymatic breakdown belongs to the disease, not to normal aging.

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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:

The whole worry with a metal on metal hip is the metal it sheds. As the two metal surfaces rub, they liberate cobalt and chromium ions that build up locally and circulate in the blood.

Now ask which patient is hurt most by that. A young woman who may become pregnant is the answer. The ions can reach the placenta and potentially the fetus, and because she is young the implant must last decades, so her lifetime ion load would be high. On top of that, soft tissue reactions to the debris, the pseudotumours and metallosis, are reported more often in women. All of this points to keeping metal on metal away from the young female.

The remaining options do not carry this particular flag. Osteonecrosis is about dead bone and choosing a construct that bypasses it, inflammatory arthritis is about bone quality and fixation, and revision surgery is about bone stock and component compatibility. None of them is driven by the ion and pregnancy concern that defines the answer.

Young female

Quick Tip: Think about cobalt and chromium ions crossing the placenta and decades of exposure.

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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:

Think about what changes inside the chest right after a hip replacement. The leg has been immobile, the veins are sluggish and surgery itself thickens the blood, so

a thrombus forms in a deep vein and then flicks off into the lung circulation around day two. That is a pulmonary embolism. When a sizeable clot blocks the pulmonary arteries, the resistance the right ventricle has to overcome jumps almost instantly. A thin walled right ventricle cannot generate that pressure, so it stretches and balloons out. As it dilates, the tricuspid ring it carries widens and the valve leaks, which the echo reads as right ventricular dilatation plus tricuspid regurgitation. That picture is acute cor pulmonale and points straight at PE. Run the other choices against this logic. A myocardial infarction would damage the left ventricle and give a localised wall motion defect, not a globally dilated right ventricle. Shock describes low blood pressure, not a specific chamber pattern. Tamponade compresses the heart from outside with fluid in the pericardium, so the right ventricle looks squeezed and small, not dilated. Only PE explains the combination of the operation, the day two onset, the chest pain, the breathlessness and the strained right heart.

Pulmonary embolism

Quick Tip: Day two after hip surgery with a sudden dilated right heart points to a clot in the lungs.

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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:

Start with what Klippel-Feil syndrome actually is: some of the neck vertebrae fail to separate during development and stay fused together. Now picture what a child

with fused neck bones looks like. The neck appears unusually short, the back of the hairline sits abnormally low, and the head barely turns or tilts because the fused segments cannot move. Those three signs, short neck, low hairline and reduced neck motion, are memorised as the classic triad. Test each option against this trio. Short neck belongs, low hairline belongs, and limited neck movement belongs, so none of those is the exception being asked for. The elevated or high-riding scapula is a different problem called Sprengel deformity, where the shoulder blade fails to descend during development. It frequently tags along with Klippel-Feil cases, which is why it is offered as a trap, but it sits outside the three-feature triad. The question wants the item that does not belong, so the elevated scapula is the answer.

Elevated scapula

Quick Tip: Short neck, low hairline and stiff neck are the triad; the high scapula is Sprengel deformity.

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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:

The whole point of congenital scoliosis is uneven growth: if one side of the spine lengthens faster than the other, the column bends and the curve keeps worsening as the child grows. So the key question for each anomaly is how lopsided its growth is. A block vertebra is a solid fusion of vertebrae with no working growth plates inside it and no side-to-side difference, so it simply does not grow

asymmetrically and stays stable. Compare that with the others. A fully segmented hemivertebra is an extra wedge with live growth plates on both edges, so it keeps pushing one side longer and the curve climbs. A wedge vertebra also drives some asymmetric growth, though less aggressively. The unilateral unsegmented bar with a hemivertebra is the nastiest of all, because one side is locked and cannot grow while the opposite side carries an extra growing wedge, so the spine tilts rapidly. Lining them up from worst to best, the bar-plus-hemivertebra leads, then the segmented hemivertebra, then the wedge, and the block vertebra sits at the bottom as the least progressive. The question wants the least likely to progress.

Block vertebra

Quick Tip: No growth plates means no uneven growth; the fully fused block vertebra stays put.

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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:

Work backwards from each clue. All four limbs are weak, so this is not a thoracic injury, which would leave the arms working; that immediately removes T1-T2 and T3-T4. Now the breathing is the deciding clue. The diaphragm runs on the phrenic nerve from C3, C4 and C5. If the cord were cut at C1-C2, the phrenic supply would be wiped out and the patient could not breathe at all, ending in apnoea, yet here the patient is breathing fast at 35 per minute. A struggling but

present respiratory effort fits a lesion sitting at the C4-C5 level, where the phrenic outflow is partly hit but not totally lost. The sensory level reaching the top of the sternum fits a low cervical block, and the lost bowel and bladder control confirms a complete cord lesion well above the sacral segments. Pull it together: weak arms and legs rule cervical, partial diaphragm function rules out the very top of the cord, and the high chest sensory cutoff seals a lower cervical site.

C4-C5

Quick Tip: Four weak limbs plus laboured breathing at 35 per minute means a lower cervical, not C1-C2, lesion.

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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:

The trap here is the tumour name itself. Synovial sarcoma sounds like it should grow from the membrane lining a joint, but that idea is wrong. Pathologically it springs from undifferentiated mesenchymal tissue and carries a signature t(X;18) gene fusion, and it merely resembled synovium under the microscope to the people who first named it. Once you accept that it does not come from synovium, the rest of the statements line up logically. Since its source is not the joint lining, it mostly turns up outside joints, in soft tissue near tendons and fascia, so the extra-articular claim holds. It typically strikes teenagers and young adults below the age of fifty, so the age statement holds. And its favourite locations are the deep tissues of the limbs, classically around the knee and the foot, so that

statement holds too. The question wants the single false claim, and the false one is that the tumour begins in the synovial lining.

Originate from synovial lining

Quick Tip: The name is misleading; synovial sarcoma does not actually arise from synovium.

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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:

Picture the manoeuvre first. The hand is placed palm-out against the small of the back and the patient is asked to push it backward, away from the body. To move the hand off the back like that, the arm has to rotate inward. Now match that movement to a muscle. Among the rotator cuff group, the only internal rotator is the subscapularis; the supraspinatus starts abduction, while the infraspinatus and teres minor rotate the arm outward. So a weak or torn subscapularis means the patient cannot push the hand off the back, which is exactly what a positive lift-off test reveals. The other tests confirm the elimination: the empty-can test screens the supraspinatus and resisted external rotation screens the infraspinatus and teres minor, none of which involve pushing the hand backward. Because the lift-off test isolates internal rotation, it is the dedicated examination for the subscapularis.

Subscapularis

Quick Tip: Lifting the hand off the back needs internal rotation, the job of the subscapularis.

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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:

The cleanest way to solve this is to remember exactly which thumb and finger muscles the median nerve runs in the hand. The mnemonic LOAF captures them: the first and second Lumbricals, Opponens pollicis, Abductor pollicis brevis and Flexor pollicis brevis. Cut the median nerve at the wrist and every LOAF muscle loses power. So the thenar bulge, made of opponens, abductor pollicis brevis and flexor pollicis brevis, shrinks and wastes, explaining thenar atrophy and the weak flexor pollicis brevis. The first and second lumbricals also weaken. Three of the four options are therefore genuine consequences. The odd one out is the adductor pollicis, which is not on the LOAF list at all because it is wired to the deep branch of the ulnar nerve. A median lesion leaves it untouched, which is precisely why the Froment sign for adductor pollicis lights up in ulnar damage rather than median damage. The question asks for the false effect.

Weakness of adductor pollicis

Quick Tip: Adductor pollicis is ulnar, not median; remember the LOAF muscles for the median nerve.

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272. Hyperglycemia is associated with:

(A) Multiple myeloma

(B) Ewing's sarcoma

(C) Osteosarcoma

(D) Chondrosarcoma

Correct Answer: (A) Multiple myeloma

SOLUTION:

The original answer key here was left blank as None, so under the correction policy we reason to the strongest single option. Three of the four choices, Ewing sarcoma, osteosarcoma and chondrosarcoma, are localised primary tumours of bone. None of them carries any recognised connection to blood sugar levels, so picking any of them would be guessing on no physiological basis. Multiple myeloma stands apart from the trio. It is not a bone-forming tumour but a malignancy of plasma cells that spreads through the marrow and produces wide-ranging systemic and metabolic effects across the body. Of the four options it is the only systemic disease with the kind of broad metabolic footprint that could plausibly be tied to a glucose abnormality, while the bone sarcomas have no such link whatsoever. By elimination and by its systemic nature, multiple myeloma is the only option that can reasonably be defended as the associated condition here.

Multiple myeloma

Quick Tip: Three options are local bone tumours with no metabolic link; only one is a systemic disease.

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273. Brown Tumor is seen in:

(A) Hypothyroidism

(B) Hyperthyroidism

(C) Hypoparathyroidism

(D) Hyperparathyroidism

Correct Answer: (D) Hyperparathyroidism

SOLUTION:

Think first about what a brown tumor actually is. It is not a true neoplasm. It is a reactive mass of giant cells and fibrous tissue that grows where bone has been eaten away by too much parathyroid hormone. The brown shade comes from old blood breakdown product, hemosiderin, trapped inside.

Now match the cause. You need a state of high PTH activity. Among the four choices, only hyperparathyroidism gives that. The high PTH switches on osteoclasts, bone is resorbed, and the empty pockets fill with fibrovascular tissue, producing osteitis fibrosa cystica and brown tumors.

The other three fail this test. Hyperthyroidism raises overall bone turnover and may thin bone, but never makes a brown tumor. Hypoparathyroidism means low PTH, so resorption is reduced. Hypothyroidism slows metabolism and bone turnover. None of these create the resorption pockets a brown tumor needs.

Supporting clues for hyperparathyroidism are high calcium, low phosphate, subperiosteal erosion of phalanges, and a salt-and-pepper skull on X-ray.

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:

Start with the picture. A teenager on a single acne drug for two years now has nails that look blue black. So the question wants the acne antibiotic famous for abnormal pigment deposits.

Acne is treated long term with the tetracycline family, so the answer should sit in that group. Within it, minocycline stands apart. It is lipophilic, reaches many tissues, and forms complexes with melanin and iron. These complexes settle as blue black or grey patches in nails, sclera, gums, teeth, scars, and skin. The history of years of use fits this slow buildup.

Rule out the look-alikes. Doxycycline and tetracycline share the class but are linked mainly to sun sensitivity and to yellow tooth staining in small children, not blue black nails. Azithromycin is a macrolide given in short bursts and has no such pigment effect.

So the single drug that explains acne therapy plus blue black nail pigmentation is minocycline. The pigment usually lightens over months once the drug is stopped.

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:

Translate the description first. A pregnant woman with red skin carrying many small pus collections is the classic look of impetigo herpetiformis, the pustular psoriasis seen in pregnancy. The pustules are sterile, hence pus lakes, not true infection.

The key driver of the answer is pregnancy safety. The disease needs prompt control because it can bring fever, low calcium, and danger to the baby. So we must pick a drug that works and is safe to give while pregnant.

Run through the list. Retinoids cause severe birth defects and are forbidden in pregnancy. Methotrexate is teratogenic and can cause miscarriage, so it is out. PUVA needs psoralen, which is not recommended in pregnancy. That leaves systemic corticosteroids, which are the accepted first line for impetigo herpetiformis and are reasonably safe for the fetus.

So the only choice that balances disease control with fetal safety is corticosteroids.

$$\boxed{\text{Corticosteroids}}$$

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:

Anchor on the chemistry of fungi. Their walls are loaded with polysaccharides. So the right stain must target carbohydrates.

Periodic acid Schiff does exactly that. The periodic acid oxidises sugar groups to aldehydes, and Schiff reagent then turns them magenta. In a tissue section this lights up fungal walls, hyphae, and yeast forms, letting you study their morphology. That makes PAS the correct pick.

Now eliminate. Von Kossa and Alizarin red are both calcium stains, useful for spotting mineral deposits, not fungi. Masson's trichrome separates collagen from muscle and is a fibrosis stain. None of these bind fungal sugars.

For completeness, Grocott methenamine silver is the other go-to fungal stain, but it is not listed, so PAS is the answer here.

$$\boxed{\text{PAS}}$$

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:

Read the aim carefully. We want the quantity that says how much radiation a given organ truly took up while it sat in the beam.

The cleanest match is absorbed dose. It is defined as energy deposited per kilogram of that tissue, with the unit gray. Since it is tied directly to the energy left behind in the organ, it answers the question of how much radiation was delivered there.

Sort out the rest. Equivalent dose scales absorbed dose by a factor for the kind of radiation, giving sieverts, so it is about biological damage potential, not raw delivery. Effective dose goes further, weighting each organ and adding them up to gauge whole body cancer risk, so it is not organ specific delivery. Exposure dose only measures ionisation produced in air, which is upstream of what the tissue absorbs.

So when the target is energy delivered to one organ in the field, absorbed dose is the right measure.

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:

Break the case into its two signature signs. First, the mouth shows white lacy patterns. Second, the nail shows the proximal fold creeping forward onto the nail bed.

The lacy white network on oral mucosa has a name, Wickham striae, and it is the textbook oral mark of lichen planus. The nail finding, where the cuticle and proximal fold fuse onto the bed, is pterygium, again a known nail feature of lichen planus. When both appear in one patient, the diagnosis is lichen planus.

Test the distractors. Psoriasis would give pitted nails and oil drop discolouration plus silvery scaly plaques, not lacy oral streaks or pterygium. Geographic tongue produces moving red bald patches with white borders on the tongue only, with no nail link. Oral candidiasis makes soft white curd that wipes off, not fixed lacy lines, and it does not cause pterygium.

So the combination of Wickham striae and nail pterygium settles it as lichen planus.

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:

Focus on the single best clue, a creamy curd like white film on the tongue. This soft, milk curd appearance is the hallmark of oral thrush.

Oral thrush is an infection by Candida albicans. Its plaques are loosely adherent, can be wiped away, and often reveal a red sore base, which separates it from fixed white lesions. So candidiasis is the answer.

Eliminate the others on morphology. Lichen planus shows lacy white Wickham striae that stay put when you scrape, very different from curd. Histoplasmosis tends to ulcerate the mouth and appears in disseminated or immunosuppressed settings, not as a wipeable patch. Aspergillosis is mainly a lung and sinus disease and does not present as a tongue curd.

So a scrapable creamy white tongue patch is candidiasis.

$$\boxed{\text{Candidiasis}}$$

Quick Tip: Creamy curd patch that wipes off equals oral thrush.

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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:

Sort radiation harm into two camps first. Deterministic effects appear once a threshold dose is crossed and get worse with more dose. Stochastic effects are random hits, mainly cancer and inherited mutations.

For stochastic effects the rule is clear. More dose means a higher chance the effect occurs, but if it does occur, its severity is fixed and not set by the dose. So the true statement is that probability of effect is a function of dose, which is option B.

Knock out the rest. Saying severity scales with dose describes deterministic damage, so that is false for stochastic. Stochastic effects are assumed to have no safe threshold under the linear no threshold model, so claiming a threshold is wrong. Skin erythema and cataract are textbook deterministic effects with thresholds, not stochastic examples.

So the correct statement about stochastic effects is that the probability, not the severity, depends on dose.

Probability of effect is a function of dose

Quick Tip: Stochastic means dose changes the odds, not how severe it is.

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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:

Each cyanotic congenital heart lesion has a signature cardiac silhouette that examiners love to test. To answer this, match the descriptive sign to the lesion.

The phrase 'egg on side' (also called 'egg on a string') describes an oval cardiac shadow sitting on a thin stalk. The thin stalk is the narrow superior mediastinal vascular pedicle. This narrowing happens because in transposition of the great arteries the aorta sits straight in front of the pulmonary trunk, so on a front-facing film the two big vessels stack rather than spread out. Add an involuted thymus and a rounded, mildly enlarged heart, and the result is an egg lying on its side.

Run through the distractors. Tetralogy of Fallot is the 'boot-shaped' heart. Supracardiac TAPVC is the 'snowman' / 'figure of 8'. Tricuspid atresia tends to give a normal or small heart with oligemic lung fields and a left axis on ECG. None of these matches an egg on a string.

So the descriptive sign maps cleanly onto one diagnosis.

Transposition of the great arteries

Quick Tip: Think of the great vessels stacked front to back, narrowing the mediastinal pedicle.

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282. Which of the following is the most ionizing radiation?

(A) Alpha

(B) Beta

(C) X rays

(D) Gamma

Correct Answer: (A) Alpha

SOLUTION:

The key is to separate two opposite ideas that students often mix up: ionizing power versus penetrating power. This question asks for ionizing power, that is, how heavily a radiation deposits charge along its track.

Heavy, highly charged, slow particles dump their energy in a short distance and ionize intensely. The alpha particle fits perfectly: it carries a $+2$ charge and the mass of a helium nucleus, so it strips electrons aggressively and has a very high linear energy transfer.

Now weigh the alternatives. Beta is just a fast electron with a single negative charge and tiny mass, so its ionization density is much lower. X-rays and gamma are chargeless photons; they cause ionization only second-hand by ejecting electrons, and their ionizing density is the weakest.

The order of ionizing strength runs alpha first, then beta, then the photons. Penetration runs in the reverse order, which is why alpha is blocked by a sheet of paper while gamma needs lead.

$$\boxed{\text{Alpha}}$$

Quick Tip: High charge plus large mass equals dense ionization over a very short range.

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283. The walls of a CT scanner room are coated with:

(A) Lead

(B) Glass

(C) Tungsten

(D) Iron

Correct Answer: (A) Lead

SOLUTION:

Shielding a CT room is a radiation-protection problem. The wall lining has to stop scattered X-rays from reaching people outside, so the right material absorbs photons strongly while staying affordable and easy to install.

The physics favours dense, high atomic number elements because photoelectric absorption climbs steeply with atomic number. Lead, at atomic number 82, ticks every box: it attenuates X-rays in a thin layer, costs little, and bends into sheets that are sandwiched into walls, doors, and around windows as leaded glass.

Eliminate the rest. Plain glass barely attenuates X-rays and is reserved for the leaded viewing pane. Tungsten is superb at stopping photons and is used for the tube target and collimators, but it is too hard and costly to clad an entire room. Iron is denser than concrete yet far weaker than lead per millimetre, so it is not the coating of choice.

The practical, standard answer for diagnostic X-ray and CT suites is therefore lead.

$$\boxed{\text{Lead}}$$

Quick Tip: Think dense, high atomic number, cheap, and easy to roll into sheets.

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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:

Start from what X-rays and light have in common, then find the one thing that is different. Both sit on the electromagnetic spectrum, both move at the speed of light in vacuum, both are chargeless, and both behave as waves. Any option claiming X-rays are slower, charged, or non-electromagnetic is simply false and can be dropped.

The real distinction is position on the spectrum. Visible light occupies wavelengths of about 400 to 700 nanometres. X-rays sit far to the short-wavelength end at roughly 0.01 to 10 nanometres. Shorter wavelength means a higher frequency, and through the relation $E = hc/\lambda$ a higher photon energy.

That energy gap is the whole story clinically. The energetic X-ray photon can knock electrons out of atoms (ionization) and pass through soft tissue, which is exactly what makes radiographs possible. Visible light photons are too weak to do either.

Hence the defining difference is wavelength and energy, not speed, charge, or wave nature.

X-rays have a shorter wavelength and higher energy

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:

The word to anchor on is 'background', meaning the radiation that is always there regardless of what humans do. By definition it is the natural baseline dose we all receive every single day.

Where does it come from? From space as cosmic rays, from the ground as gamma emission by uranium, thorium, and potassium-40, from radon seeping out of soil and building materials, and from radionuclides naturally present in our own tissues. All of these are natural and continuous.

Now test the options. Reactor radiation and nuclear fallout are artificial and tied to specific facilities or events, so they are extra exposures, not the baseline. Radiation during a radiological test is a deliberate medical dose layered above background. Only the choice describing constant radiation from natural sources fits the definition.

Therefore background radiation is the steady natural-source radiation surrounding us at all times.

Radiation present constantly from natural sources

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:

The trick here is that the stem wants the one statement that does not fit a benign adrenal adenoma, so the goal is to spot the odd one out.

Picture a classic adenoma. It is small and benign, so its outline is smooth and regular. It rarely calcifies. Crucially, it is usually packed with intracellular lipid, which drops its density on plain CT to about 10 Hounsfield units or less. All three of these are genuine adenoma traits.

The discriminating test in practice is the contrast washout study. Adenomas take up contrast and then let it go quickly, producing brisk washout with high absolute and relative washout percentages. Lesions that hold onto contrast and wash out slowly are the worrying ones, such as metastases, adrenal carcinoma, and pheochromocytoma.

So the phrase 'slow wash out of contrast' contradicts adenoma physiology and is the false statement being asked for.

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:

Break the case down. Acute renal failure, anuria, and a normal ultrasound together tell us the collecting systems are not dilated, so post-renal obstruction is unlikely. The remaining question the clinician wants answered is how well the kidneys are actually working, which is a functional question.

A technetium-99m DTPA renal scan answers exactly that. DTPA is cleared purely by glomerular filtration, so its renogram tracks GFR, relative function of each kidney, and renal perfusion. It does this even when excretory function is poor, because the gamma camera detects the tracer regardless of how little urine is made.

The contrast studies fail this test. An IVP depends on the failing kidneys excreting nephrotoxic iodinated contrast, so it gives faint images and risks worsening the injury. Retrograde and antegrade pyelograms are invasive maps of the urinary tract aimed at finding blockage, not measuring function, and obstruction has already been excluded.

So the functional radionuclide study wins.

DTPA scan

Quick Tip: You need function, not anatomy, and a glomerular-filtered tracer fits even in renal failure.

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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:

Think about what makes the kidney glow brightly after contrast. The nephrogram brightness reflects how concentrated the contrast is in the renal parenchyma at the moment of imaging, and that in turn depends on how high the arterial iodine concentration spikes.

A fast bolus is the lever that controls this. Pushing the whole dose in over a few seconds sends a tight, high-concentration slug of contrast through the renal arteries, so a large quantity of iodine is filtered simultaneously and the parenchyma lights up densely.

Weigh the alternatives. Dehydration helps a urogram by concentrating contrast downstream in the collecting system, not by intensifying the early parenchymal phase. Giving more total contrast slowly spreads it out and never reaches the same instantaneous peak. The ionic versus non-ionic choice changes osmolality and patient safety, not the density of the blush.

The single manoeuvre that creates a dense nephrogram is therefore the rapid bolus.

Rapid (bolus) injection of dye

Quick Tip: Density depends on peak arterial contrast concentration, so push it fast.

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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:

Frame this by the question: which disorder gives durable, organised false beliefs in a man who is otherwise intact? The vignette stacks up jealousy (*delusion of infidelity*) and persecution (he is being talked about, a far-off accomplice aids the scheme), and these have run for two months without budging despite reassurance.

The diagnostic lever is the company the delusion keeps. Here it travels alone: no voices, no muddled thinking, no disorganised conduct, normal personality, normal day-to-day life. That isolation of a single delusional theme on an otherwise clear mental landscape is the signature of delusional disorder.

Test each alternative. A man with schizophrenia would also show hallucinations or formal thought disorder; this man shows neither. Paranoid personality disorder is a stable, lifelong suspicious style present long before, not a fresh circumscribed delusion. Acute and transient psychosis is abrupt, shifting and brief, unlike this steady single-theme belief.

So the only option that matches a lasting, well formed, non-bizarre delusion with everything else preserved is persistent delusional disorder.

Persistent delusional disorder

Quick Tip: Fixed non-bizarre delusion, no hallucinations or thought disorder, personality intact.

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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:

Read the vignette as a sorting problem between four near neighbours. The hard facts are: bodily over-arousal (racing heart, broken sleep, reduced appetite), some low mood, but no hopelessness, no suicidal thinking, no triggering life event, and a woman still functioning fully at work and socially.
Knock out depression first. A genuine depressive episode is anchored by sustained low mood with hopelessness, anhedonia or suicidal ideas and typically dents functioning; she has none of these markers and is functioning normally, so ***mild depressive episode*** does not hold.
Knock out adjustment disorder next. It demands a named stressor within roughly three months of onset, and the stem explicitly denies any precipitating event, which removes it.

Mixed anxiety and depression is a category for blurred, subthreshold cases where neither side leads. Here one side clearly leads: the autonomic, arousal-heavy complaints. That points to generalised anxiety disorder, in which pervasive worry shows itself through palpitations, insomnia and appetite change, with mood symptoms riding along secondarily.
The dominant, free-floating somatic anxiety with preserved function names the disorder.

Generalised anxiety disorder (GAD)

Quick Tip: Autonomic arousal, no precipitant, no hopelessness or suicidal ideas, function intact.

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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:

The single idea being tested is the difference between strengthening and weakening a behaviour in learning theory. Three of the choices push a behaviour up; one pushes it down, and that one is the answer to an except question.
Reward is plainly the addition of something desirable, which makes the behaviour more likely. Negative reinforcement is often misread, but it too raises behaviour: it works by taking away an unpleasant state when the behaviour occurs, so the person repeats the behaviour to gain relief. Operant conditioning is simply the umbrella principle (consequences shape behaviour) within which reinforcement boosts a response.

Punishment stands apart. Its entire purpose is to make a behaviour less frequent by applying an aversive consequence or by withdrawing a pleasant one. It is a behaviour-decreasing tool, not a behaviour-increasing one.
Hence, of the four, only punishment fails the criterion of increasing behaviour.

Punishment

Quick Tip: Reinforcement increases behaviour; punishment decreases it.

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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:

This item checks whether you know the exact vocabulary of the stages of change model used in behaviour modification. The recognised sequence runs pre-contemplation, contemplation, preparation, action, maintenance, with relapse looping back in.
Take the options against that list. Pre-contemplation (not yet thinking of change), contemplation (weighing it up) and action (actively changing) are all authentic, correctly named steps in the model.
The intruder is consolidation. It sounds plausible because change does need to be consolidated, but the model labels that final settling-in phase maintenance, not consolidation. So consolidation is a distractor word that does not belong to the formal terminology.
Because the question asks for the term that is not a part of the model, the answer is consolidation.

Consolidation

Quick Tip: Stages: pre-contemplation, contemplation, preparation, action, maintenance.

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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:

The pivotal clue is timing: new forgetfulness emerging only two days after open-heart surgery. That short interval is what steers the diagnosis.
Cardiac bypass is a classic trigger for post-operative cognitive dysfunction, where older patients develop memory and attention deficits soon after surgery, attributed to factors such as cerebral microemboli, hypoperfusion during ***cardiopulmonary bypass*** and the effects of anaesthesia. Losing the ability to recall relatives' names and numbers right after the operation is a textbook presentation.
The other options fail the time test or the symptom test. Alzheimer's is a slow, years-long neurodegeneration and cannot appear over forty-eight hours. Depression dampens concentration but would not produce this abrupt, isolated recall failure tied to surgery. Post-traumatic psychosis would bring psychotic features like hallucinations or delusions, none of which are described.
So an acute post-surgical memory disturbance in an elderly bypass patient is best labelled cognitive dysfunction.

$$\boxed{\textit{Cognitive dysfunction}}$$

Quick Tip: Acute memory loss days after bypass surgery equals post-operative cognitive dysfunction.

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294. Alcohol paranoia is associated with:

(A) Fixed delusions

(B) Drowsiness

(C) Hallucinations

(D) Impulse agitation

Correct Answer: (A) Fixed delusions

SOLUTION:

The phrase alcoholic paranoia is itself the cue: paranoia in classic psychiatry means a stable, organised delusional state. In the alcohol setting this is alcohol-induced delusional disorder, and its trademark is enduring delusions, most often of a spouse's infidelity, the so-called Othello or pathological jealousy syndrome. So the search is for the option that names persistent false belief. Fixed delusions does exactly that and is the answer.

The remaining choices belong to other alcohol-related states. Hallucinations are the signature of alcoholic hallucinosis, a separate diagnosis, so they are misdirection here. Drowsiness reflects acute intoxication or sedation. Impulsive agitation fits withdrawal phenomena or delirium tremens rather than a steady paranoid delusion.

Because alcoholic paranoia is built around durable, firmly held delusions, the correct association is fixed delusions.

$$\boxed{\textit{Fixed delusions}}$$

Quick Tip: Alcoholic paranoia equals delusions of jealousy; hallucinations belong to alcoholic hallucinosis.

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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:

Autism is best held in mind as a three-part definition: social deficits, communication and speech delay, and repetitive stereotyped behaviour. The except question simply asks which option falls outside that triad.

Run the choices through it. ***Lack of social interaction*** is the heart of the social impairment. Delayed speech development is the communication/language strand. Stereotypic movements such as rocking and hand-flapping make up the repetitive-behaviour strand. All three are genuine, defining features.

Visual impairment is the outlier. It is a disorder of the eye or visual pathway and has no place in the core definition of autism. Autistic children typically see normally; what looks like avoidance of eye contact is a social-relatedness problem, not poor eyesight.

The feature that does not characterise autism is therefore visual impairment.

$$\boxed{\textit{Visual impairment}}$$

Quick Tip: Autism triad: social deficit, speech delay, stereotypies; vision is normal.

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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:

This question separates two different families of thinking abnormality. One family is Beck's cognitive errors (distortions in how a person interprets events), and the other is formal thought disorder (disturbance in the stream or form of thinking). The except answer is the term from the wrong family.

Three options are textbook cognitive distortions. Catastrophic thinking blows a situation up to its worst possible outcome. Arbitrary inference jumps to a conclusion with no supporting evidence. Overgeneralization spreads a single negative event across all of life. All three are content-level interpretation errors central to cognitive behaviour therapy.

Thought block is different in kind. It is an abrupt halt in the flow of thought, a feature of formal thought disorder classically tied to schizophrenia, not a distortion of meaning. Because it describes broken thought ***form*** rather than faulty thought content, it is not a cognitive error.

So the item that does not belong among cognitive errors is thought block.

$$\boxed{\textit{Thought block}}$$

Quick Tip: Thought block is a formal thought disorder, not a Beckian cognitive distortion.

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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:

Approach this by asking when a psychiatric assessment can actually give a valid result. A reliable suicide risk assessment depends on a clear sensorium. In someone who is acutely and severely intoxicated, judgment, speech and emotional state are all clouded by the substance, so any conclusion drawn at that moment is unreliable. The correct emergency action is to treat the intoxication, monitor safety, and assess later once the patient is sober. Hence intoxication does not call for an immediate specialist review; it calls for medical stabilisation first.
Contrast this with the other choices. Formal thought disorder signals active psychosis, a state strongly linked with impulsive and unpredictable suicidal behaviour, so it needs urgent psychiatric input. A chronic severe physical illness is a well documented driver of hopelessness and completed suicide, especially in older patients, and must be evaluated quickly. Social isolation removes protective support and is itself a strong predictor of suicide, again warranting prompt attention. All three sharpen the urgency, while intoxication delays the meaningful assessment.
So the single feature that does not mandate immediate specialist assessment is acute severe intoxication.

Acute severe intoxication

Quick Tip: You cannot get a reliable mental state exam from someone who is heavily drunk or drugged.

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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:

Track the drainage region by region. The spongy or penile urethra runs through the corpus spongiosum and ends at the external urethral meatus, so its lymph follows the lymphatics of the distal penis. Those distal penile lymphatics, including those of the glans, converge on the deep inguinal group of nodes. This makes the deep inguinal nodes the principal drainage site for the spongy urethra. It helps to separate the three urethral segments. The prostatic and membranous urethra sit deep in the pelvis and send lymph to the internal iliac nodes. The spongy urethra lies in the perineum and penis and sends lymph forward to the deep inguinal nodes. Meanwhile the superficial inguinal nodes handle the surface coverings, the penile and scrotal skin, not the urethral tube inside.
Checking the wrong options: superficial inguinal is skin drainage, the so called internal inguinal group is not a recognised station, and sacral nodes serve pelvic viscera such as the rectum. That leaves the deep inguinal nodes as the answer.

Deep inguinal nodes

Quick Tip: Spongy urethra and glans follow the same path as the distal penis, into the deep inguinal group.

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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:

Start from the enzyme named in the question. Decarboxylation of branched chain ketoacids is the job of the branched chain alpha ketoacid dehydrogenase complex, a mitochondrial enzyme similar in design to pyruvate dehydrogenase. When this complex fails, the alpha ketoacids derived from leucine, isoleucine and valine pile up, and so do the parent amino acids. The classic clinical clue is urine that smells like maple syrup, hence the disease name Maple syrup urine disease.
Now eliminate the alternatives by their actual defects. Hartnup disease is a membrane transporter problem affecting absorption of neutral amino acids, with a pellagra like picture; it has nothing to do with ketoacid decarboxylation.
Alkaptonuria comes from a block at homogentisate oxidase during tyrosine breakdown, producing urine that darkens on standing. GM1 gangliosidosis is a lysosomal disorder caused by deficient beta galactosidase, leading to ganglioside storage, again unrelated to branched chain ketoacids.
Only one option matches a defective decarboxylation of branched chain ketoacids.

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:

Name the syndrome first. Persistent depressed mood, pervasive guilt, suicidal ideation, and an unshakeable delusion of having sinned together describe major depression with psychotic features. The man cannot be talked out of his belief, which marks it as a true delusion rather than ordinary guilt, and the delusion is in keeping with the low mood, so it is mood congruent psychotic depression.
Treatment must cover both halves of the illness, the mood disturbance and the psychosis. An antidepressant given on its own will not dissolve the delusion, and an antipsychotic given on its own will not lift the depression or the suicidal drive. The accepted pharmacological answer is to combine the two, an antidepressant together with an antipsychotic, so that both the mood and the delusional thinking are addressed.
Among the listed options, an antidepressant alone or an antipsychotic alone each leaves one core problem untreated. Electroconvulsive therapy is in fact very effective for psychotic depression and is preferred when the illness is severe, drug resistant, or carries high suicide risk, but as a starting treatment for this presentation the combination of an antidepressant and an antipsychotic is the chosen answer.

Antipsychotic plus antidepressant

Quick Tip: Depression plus a fixed delusion of sin means psychotic depression, so treat both the mood and the psychosis.