NEET PG 2001 Combined Paper Question Paper with Solutions

General Instructions

(i) This booklet contains 200 questions, each provided with a complete, step-by-step solution.

(ii) It comprises 198 single-correct multiple-choice questions and 2 multiple-correct questions.

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

(iv) For multiple-correct questions, all correct options must be selected to earn full marks.

1. A young patient developed painless sudden loss of vision which spontaneously improved over a period of 3 months. What is the most probable diagnosis?

(A) Macular hole

(B) Central serous retinopathy

(C) Angle closure glaucoma

(D) Retinal detachment

Correct Answer: (B) Central serous retinopathy

Solution:

Step 1: Note the key clues in the history.

The patient is young, the vision loss is painless and sudden, and most importantly it improved on its own over 3 months. This pattern of spontaneous recovery is the biggest clue here.

Step 2: Match the clues to central serous retinopathy (CSR).
CSR happens when fluid leaks under the retina from the choroid through a weak spot in the retinal pigment epithelium, lifting up the macula and causing a small area of blurred or distorted central vision. It hits young to middle aged adults, often men under stress, and the classic feature is that the fluid reabsorbs on its own in most cases within 1 to 6 months, restoring vision.

Step 3: Rule out macular hole.
A macular hole causes painless central vision loss too, but it does not resolve on its own. It needs surgery (vitrectomy) to close, so spontaneous improvement over months rules this out.

Step 4: Rule out angle closure glaucoma.
Angle closure glaucoma causes a painful red eye with sudden vision loss, headache, nausea and a fixed mid dilated pupil. This patient has no pain, so this does not fit.

Step 5: Rule out retinal detachment.
Retinal detachment causes sudden painless vision loss too, often with flashes, floaters and a curtain like field defect, but it does not resolve without treatment. It gets worse, not better, if left alone.

Step 6: Final answer.
Only central serous retinopathy fits a young patient with painless vision loss that gets better by itself over a few months.

Central serous retinopathy

Quick Tip: Think of a condition that causes painless central vision loss in young adults and typically resolves on its own within months.

2. Leucoria is seen in all of the following EXCEPT

(A) Retinoblastoma

(B) Congenital glaucoma

(C) Persistent hyperplastic primary vitreous

(D) Fungal endophthalmitis

Correct Answer: (B) Congenital glaucoma

Solution:

Step 1: Recall what leucoria means.
Leucoria is a white reflex seen in the pupil instead of the normal red reflex. It happens when something white or opaque sits behind the lens, such as a tumour, a fibrous membrane, an exudate or a detached retina, and reflects light back as white.

Step 2: Check retinoblastoma.
Retinoblastoma is the classic and most dangerous cause of leucoria in a child, sometimes called cat's eye reflex. The white tumour mass behind the lens reflects light white. So this does cause leucoria.

Step 3: Check persistent hyperplastic primary vitreous (PHPV).
PHPV is a failure of the fetal hyaloid vascular system to regress, leaving a fibrovascular membrane behind the lens. This white membrane also produces leucoria, usually in a small eye (microphthalmos). So this does cause leucoria.

Step 4: Check fungal endophthalmitis.
Fungal endophthalmitis fills the vitreous cavity with white inflammatory exudate, which also shows up as a white pupillary reflex. So this does cause leucoria too.

Step 5: Check congenital glaucoma.
Congenital glaucoma raises pressure inside the eye and causes corneal edema, an enlarged hazy cornea (buphthalmos), watering and light sensitivity. The clouding is in the cornea itself, not a white mass behind the lens, so it does not produce a true leucoria (white pupillary reflex). This is the odd one out.

Step 6: Final answer.
Congenital glaucoma is not a cause of leucoria.

Congenital glaucoma

Quick Tip: Leucoria is a white mass or membrane behind the lens; congenital glaucoma instead clouds the cornea itself.

3. A female patient complains of blurring of vision and was prescribed $-0.5D$ spherical lenses. Retinoscopy done at $1\ m$ with a plane mirror will cause the image to move in which manner?

(A) Image moves in the direction of the mirror

(B) Image moves in the opposite direction

(C) No movement of the image

(D) Scissors reflex is seen

Correct Answer: (A) Image moves in the direction of the mirror

Solution:

Step 1: Recall how retinoscopy movement works.
In retinoscopy, the examiner shines light into the patient's eye and rocks the mirror, watching how the red reflex in the pupil moves. The direction of the reflex tells us where the patient's far point sits compared to the examiner.

Step 2: Convert the working distance into a dioptric value.
The examiner is working at $1\ m$.

$$\text{Working distance power} = \frac{1}{1\ m} = 1.00\ D$$

This means an eye with exactly $-1.00D$ of myopia would have its far point sitting right at the examiner's eye, giving no movement (a neutral point).

Step 3: Locate the patient's far point.
The patient needs $-0.5D$ spherical lenses, so her own refractive error is $-0.5D$ of myopia. The far point of a $-0.5D$ myope lies at

$$\frac{1}{0.5\ D} = 2\ m$$

in front of her eye.

Step 4: Compare the far point to the working distance.
The examiner sits at $1\ m$, and the patient's far point is at $2\ m$. Since $2\ m$ is farther away than the examiner's position, the examiner is sitting inside the patient's far point.

Step 5: Apply the retinoscopy rule.
When the examiner is within the patient's far point (that is, the patient's myopia is less than the dioptric value of the working distance), the reflex moves "with" the mirror, meaning the image moves in the same direction as the mirror sweep. This is called a with movement, and it means more minus lens (or less plus) is needed to reach neutrality, matching a mild $-0.5D$ myope tested at $1\ m$.

Step 6: Rule out the other options.
A no movement (neutral) reflex would only be seen if her error were exactly $-1.00D$. An against movement (opposite direction) happens only when the patient's myopia is greater than the working distance value, for example more than $-1.00D$. A scissors reflex is seen in irregular astigmatism or keratoconus, not in a simple case like this.

Step 7: Final answer.
Since the patient's far point lies beyond the 1 m working distance, the reflex shows a with movement, so the image moves in the direction of the mirror.

Image moves in the direction of the mirror

Quick Tip: Compare the patient's far point distance to the examiner's 1 m working distance to find the movement direction.

4. What is the most common eye manifestation of allergy to tubercular bacilli?

(A) Koeppe's nodules

(B) Posterior scleritis

(C) Phlyctenular conjunctivitis

(D) Optic neuritis

Correct Answer: (C) Phlyctenular conjunctivitis

Solution:

Step 1: Understand what "allergy to tubercular bacilli" means in the eye.

This phrase points to a hypersensitivity reaction, meaning the eye reacts to tuberculoprotein (antigens from the TB bacillus) rather than the bacillus directly invading the eye tissue.

Step 2: Recall phlyctenular conjunctivitis.

Phlyctenular conjunctivitis (or keratoconjunctivitis) is a delayed type IV hypersensitivity reaction to bacterial antigens, most classically tuberculoprotein, though it can also follow staphylococcal disease. It shows up as small, tender, pink or grey nodules (phlyctens) at the limbus, with redness, photophobia, watering and lid spasm. It is the textbook example of an ocular allergic response to TB.

Step 3: Rule out Koeppe's nodules.

Koeppe's nodules are small white deposits at the pupillary margin of the iris, seen in granulomatous uveitis. They reflect direct granulomatous inflammation in the eye, not a hypersensitivity type reaction, and are not the classic allergic TB eye sign.

Step 4: Rule out posterior scleritis.

Posterior scleritis is a rare, painful inflammation of the sclera that can

be linked to autoimmune disease or, less often, infection including TB, but it is not described as a hypersensitivity/allergic phenomenon and is far less common than phlyctenular disease.

Step 5: Rule out optic neuritis.
Optic neuritis can rarely complicate TB, usually from direct spread or drug toxicity (ethambutol), but again it is not the classic allergic manifestation.

Step 6: Final answer.
Phlyctenular conjunctivitis is the well known hypersensitivity reaction of the eye to tubercular antigens.

Phlyctenular conjunctivitis

Quick Tip: Think of the type IV hypersensitivity nodule at the limbus classically linked to TB antigen exposure.

5. What is the diagnosis for ropy discharge from the eyes, along with itching, which occurs every summer?

(A) Vernal catarrh

(B) Bacterial conjunctivitis

(C) Trachoma

(D) Phlyctenular conjunctivitis

Correct Answer: (A) Vernal catarrh

Solution:

Step 1: Pick out the key features.
Three clues matter here: the discharge is ropy or stringy (thick mucoid strands), there is itching, and the problem comes back every summer.

Step 2: Recall vernal catarrh (vernal keratoconjunctivitis).
Vernal catarrh is a seasonal, IgE mediated allergic eye disease, most active in warm, dry months such as summer. Its hallmark symptom is intense itching, along with a thick, ropy (stringy) mucoid discharge, cobblestone papillae on the upper tarsal conjunctiva, and photophobia. It is more common in young boys and tends to recur each year in the same season.

Step 3: Rule out bacterial conjunctivitis.
Bacterial conjunctivitis gives a purulent, thick yellow-green discharge that is sticky rather than ropy, with redness and crusting of lashes on waking. It is not typically itchy, and it does not follow a strict seasonal summer pattern.

Step 4: Rule out trachoma.
Trachoma is a chronic infection from Chlamydia trachomatis. It causes follicles and scarring on the upper tarsal conjunctiva that over years can lead to lid scarring and entropion. It is not marked by itching or ropy discharge, and it is not a seasonal condition.

Step 5: Rule out phlyctenular conjunctivitis.
Phlyctenular conjunctivitis presents as tender limbal nodules with photophobia and watering, usually linked to TB hypersensitivity, not itching with ropy discharge, and it is not tied to a specific season.

Step 6: Final answer.

The itching, ropy discharge, and yearly summer recurrence point to vernal catarrh.

Vernal catarrh

Quick Tip: Seasonal itching with thick, stringy discharge in summer is the classic picture of an allergic eye disease.

6. Which wall is most often fractured in a blow out fracture of the orbit due to a fisticuff injury?

(A) Superior wall

(B) Inferior wall

(C) Medial wall

(D) Lateral wall

Correct Answer: (B) Inferior wall

Solution:

Step 1: Recall what a blow out fracture is.
A blow out fracture happens when a blunt object, such as a fist or a ball, strikes the eye and suddenly raises the pressure inside the bony orbit. This pressure has to escape somewhere, and it breaks through the thinnest wall of the orbit rather than the sturdy orbital rim.

Step 2: Compare the thickness of the orbital walls.
The orbital floor (inferior wall) is made of thin bone over the maxillary sinus, and the medial wall (lamina papyracea) is also thin over the ethmoid sinuses. The superior wall and lateral wall are both

much thicker and stronger, since the lateral wall is reinforced by the zygomatic bone and the roof by the frontal bone.

Step 3: Identify the weakest point.
Between the floor and the medial wall, the orbital floor is classically the weakest and most commonly fractured site, especially along the region near the infraorbital groove.

Step 4: Understand the clinical picture that follows.
When the floor fractures, orbital fat and sometimes the inferior rectus or inferior oblique muscle can herniate into the maxillary sinus below, causing enophthalmos (sunken eye), restricted upward gaze, and numbness of the cheek and upper lip from infraorbital nerve involvement. This clinical picture matches floor fractures specifically.

Step 5: Rule out the other walls.
The medial wall can fracture too, sometimes together with the floor, but alone it is less common as the single most fractured wall. The superior and lateral walls rarely fracture in a blow out injury because they are much thicker.

Step 6: Final answer.
The inferior wall (orbital floor) is the wall most often fractured in a blow out fracture.

Inferior wall

Quick Tip: Think of the thinnest bone in the orbit, which lies over the maxillary sinus below the eye.

7. Which of these is NOT useful in arriving at a diagnosis of moderate papilledema in a patient of head injury?

(A) Impaired pupillary reflex

(B) Hyperemia

(C) Filling of the physiological cup

(D) Blurring of the margins

Correct Answer: (A) Impaired pupillary reflex

Solution:

Step 1: Recall what papilledema is.
Papilledema is swelling of the optic disc caused by raised intracranial pressure, which is transmitted along the optic nerve sheath and blocks normal axoplasmic flow at the disc, causing it to swell.

Step 2: Recall the early to moderate fundus signs.
On fundus exam, the earliest signs of papilledema include hyperemia (a flushed, engorged looking disc from dilated capillaries), filling of the physiological cup (the normal central depression becomes shallow and fills in as the disc swells), and blurring of the disc margins, first at the nasal edge and then all around. These are the standard signs doctors look for on ophthalmoscopy to grade papilledema from early to moderate to severe (chronic/atrophic) stages.

Step 3: Think about what pupillary reflex tells us.
The pupillary light reflex depends on the afferent visual pathway carrying light signals from the retina to the midbrain. In papilledema, especially moderate papilledema, vision and the pupillary reflex usually stay normal for a long time, because the swelling primarily affects the optic disc surface and retinal fibers without cutting off the

reflex pathway early on.

Step 4: Contrast with optic neuritis.
An impaired pupillary reflex, specifically a relative afferent pupillary defect, is a hallmark of optic neuritis or optic nerve damage that directly disrupts the afferent pathway. Papilledema, until it becomes severe and chronic enough to cause optic atrophy, does not usually impair the pupillary reflex. So this sign does not help diagnose moderate papilledema.

Step 5: Confirm the other three are genuinely useful.
Hyperemia, filling of the cup, and blurring of the margins are all direct fundoscopic findings clinicians rely on to spot and grade papilledema.

Step 6: Final answer.
Impaired pupillary reflex is not a useful sign for diagnosing moderate papilledema.

Impaired pupillary reflex

Quick Tip: Papilledema is a fundus finding, pupillary reflex usually stays normal until very late disease.

8. What is represented by ETDRS in a diabetic vision chart?

(A) Elective treatment for diabetic retinopathy scales

(B) Extended Rx for diabetes review study

(C) Early Treatment Diabetic Retinopathy Study

(D) Eye test drum review study

Correct Answer: (C) Early Treatment Diabetic Retinopathy Study

Solution:

Step 1: Break down the question.
The ETDRS chart is a standard chart used to test vision in diabetic retinopathy patients and in many eye clinics and research trials. The question asks what the letters ETDRS stand for.

Step 2: Expand the abbreviation.
ETDRS stands for Early Treatment Diabetic Retinopathy Study. This was a large multicenter clinical trial run in the 1980s that studied how early laser treatment affects vision loss from diabetic retinopathy.

Step 3: Link the study to the chart.
As part of this study, researchers built a standardized visual acuity chart with the same number of letters on each line and equal spacing between lines (log MAR based). This chart became known as the ETDRS chart and is now used widely, not just in diabetic patients, because it gives more precise and reproducible vision measurements than the older Snellen chart.

Step 4: Rule out the wrong options.
"Elective treatment for diabetic retinopathy scales" is not a real study name, it is built to sound close to the real one.
"Extended Rx for diabetes review study" is also not a real name; there

is no such trial.
"Eye test drum review study" does not match either, and a drum is not part of how this chart got its name.

Final Answer:
ETDRS stands for the Early Treatment Diabetic Retinopathy Study.

Early Treatment Diabetic Retinopathy Study

Quick Tip: ETDRS is the name of a landmark diabetic retinopathy treatment trial.

9. What drug can be used that will provide only mydriasis and no cycloplegia, for a fundus examination in a young adult patient?

(A) Atropine ointment

(B) Phenylephrine

(C) Homatropine

(D) Tropicamide

Correct Answer: (B) Phenylephrine

Solution:

Step 1: Understand the two effects.
Mydriasis means widening of the pupil. Cycloplegia means paralysis of the ciliary muscle, which stops the eye from focusing near objects. Some drugs cause both, and some cause only one.

Step 2: Group the drugs by their action.

Atropine, homatropine, and tropicamide are all antimuscarinic drugs. They block the parasympathetic supply to the eye, which relaxes the sphincter pupillae (causing mydriasis) and also relaxes the ciliary muscle (causing cycloplegia). So all three give both effects together.

Step 3: Look at phenylephrine separately.
Phenylephrine is a direct acting sympathomimetic drug. It works on alpha-1 receptors in the dilator pupillae muscle and contracts it, which widens the pupil. It does not act on the ciliary muscle at all, so it does not affect accommodation or near focus.

Step 4: Apply this to the question.
The question wants a drug that dilates the pupil for a fundus exam but leaves accommodation intact, since the patient is a young adult who may still need to focus normally soon after the exam. Phenylephrine fits this need exactly, and its action also wears off faster than the antimuscarinics.

Step 5: Rule out the other options.
Atropine ointment has the longest duration, sometimes days, and gives strong cycloplegia, so it is not suited here. Homatropine and tropicamide are shorter acting antimuscarinics but still cause cycloplegia along with mydriasis, so they do not meet the mydriasis only requirement.

Final Answer:
Phenylephrine gives pure mydriasis without cycloplegia.

Phenylephrine

Quick Tip: Pure mydriatics act only on the dilator pupillae, sparing the ciliary muscle.

10. Which of these is NOT caused by amphotericin B?

(A) Azotemia

(B) Glomerulonephritis

(C) Hypokalemia

(D) Renal tubular acidosis

Correct Answer: (B) Glomerulonephritis

Solution:

Step 1: Recall how amphotericin B damages the kidney.
Amphotericin B binds to cholesterol in cell membranes. This is how it kills fungal cells, but it also binds cholesterol in human kidney tubular cell membranes and damages them, since these membranes are not fully protected from the drug.

Step 2: List the effects of this tubular damage.
Because the injury is at the tubule, not the glomerulus, it produces a reduced GFR with a rise in blood urea and creatinine (azotemia), potassium wasting in the urine leading to hypokalemia, and distal renal tubular acidosis (type 1 RTA) because the tubule cannot secrete hydrogen ions properly.

Step 3: Check each option against this list.
Azotemia is a known effect, since renal blood flow falls and GFR drops with amphotericin B use.

Hypokalemia is a known and common effect, often needing potassium supplements during treatment.
Renal tubular acidosis is a known effect, from direct tubular cell injury.
Glomerulonephritis is an inflammatory disease of the glomerulus, usually immune mediated. Amphotericin B does not target the glomerulus and does not cause this kind of immune injury.

Step 4: Conclude.
Since amphotericin B is a tubular toxin and not a cause of glomerular inflammation, the effect that is NOT caused by it is glomerulonephritis.

Final Answer:

Glomerulonephritis

Quick Tip: Amphotericin B damages renal tubules, not the glomerulus.

11. Ingestion of what product by the mother may cause the infant to have cleft palate, spina bifida and an (ASD doubtful)?

(A) Isotretinoin

(B) Valproate

(C) Phenytoin

(D) Carbamazepine

Correct Answer: (B) Valproate

Solution:

Step 1: Identify the type of question.

This question describes a pattern of birth defects seen after a mother takes a certain drug during pregnancy: cleft palate, spina bifida, and possibly an atrial septal defect (ASD). We need to match this pattern to the correct drug.

Step 2: Recall the fetal valproate syndrome.

Sodium valproate is an antiepileptic drug with the highest known risk of neural tube defects among antiepileptics, including spina bifida, when taken in early pregnancy. It is also linked with craniofacial defects such as cleft palate and cleft lip, and with cardiac defects including atrial septal defect and other congenital heart defects. This matches the pattern in the question closely.

Step 3: Compare with the other drugs.

Isotretinoin causes a different pattern of birth defects, called retinoic acid embryopathy, with craniofacial defects, heart defects, and central nervous system defects, but spina bifida is not the classic feature linked to it.

Phenytoin causes fetal hydantoin syndrome, which shows hypoplastic nails and distal phalanges, growth delay, and some craniofacial change, but spina bifida is not its main feature.

Carbamazepine does carry a smaller risk of neural tube defects compared with valproate, but the risk is much lower and this drug is not the strongest match for this exact combination.

Step 4: Final conclusion.

The described pattern, spina bifida together with cleft palate and possible ASD, points most strongly to valproate.

Final Answer:

Valproate

Quick Tip: This antiepileptic has the highest neural tube defect risk in pregnancy.

12. What is the most common cardiac defect caused due to lithium?

(A) Ebstein's anomaly

(B) HOCM

(C) Aortic aneurysm

(D) Eisenmenger syndrome

Correct Answer: (A) Ebstein's anomaly

Solution:

Step 1: Recall the drug in question.
Lithium is a mood stabilizer used mainly in bipolar disorder. When taken during the first trimester of pregnancy, it carries a risk to the developing heart of the fetus.

Step 2: Recall the classic cardiac defect linked with lithium.
Lithium exposure in early pregnancy is classically linked with Ebstein's anomaly, a defect where the tricuspid valve is displaced downward into the right ventricle, making part of the right ventricle act like an extension of the right atrium.

Step 3: Rule out the other choices.
Hypertrophic obstructive cardiomyopathy (HOCM) is usually inherited through gene mutations affecting heart muscle proteins, not linked to lithium exposure.
Aortic aneurysm is linked with connective tissue disorders such as Marfan syndrome, not with lithium.
Eisenmenger syndrome is a late complication of an untreated left to right shunt, not a direct drug induced defect.

Step 4: Add a note on current evidence.
Newer, larger studies have shown the absolute risk of Ebstein's anomaly with lithium is lower than older teaching suggested, but it remains the cardiac defect most specifically and classically tied to lithium exposure among the choices given here.

Final Answer:

Ebstein's anomaly

Quick Tip: This drug is classically linked to Ebstein's anomaly in the fetus.

13. A man is arrested for possession of narcotics, the culprit is found to have black tongue. What is likely to have been the substance of abuse?

(A) Heroin

(B) Cocaine

(C) Cannabis

(D) Arsenic

Correct Answer: (B) Cocaine

Solution:

Step 1: Read the clue carefully.
The man was arrested for narcotics possession and is found to have a black tongue. We need to pick the substance whose abuse pattern explains this specific physical sign.

Step 2: Link black tongue to how the drug is used.
Cocaine, especially in its freebase or crack form, is commonly smoked through a small pipe that gets very hot during use. Repeated smoking through a hot pipe stem causes thermal burns to the lips and tongue over time. These repeated burns leave blackened, charred looking patches on the tongue, a sign forensic doctors specifically look for in chronic crack or cocaine smokers.

Step 3: Rule out the other substances.
Heroin abuse usually shows needle track marks on the arms, pinpoint pupils, and slowed breathing, not a black tongue.
Cannabis (ganja) smoking causes red conjunctiva, dry mouth, and increased appetite, but is not classically tied to a blackened tongue sign in forensic teaching.
Arsenic poisoning gives signs such as Mees lines on the nails, skin pigmentation changes, and garlic odor breath, a very different picture from a burned black tongue.

Step 4: Conclude.
The black tongue sign in a narcotics case points to chronic cocaine (crack) smoking from repeated thermal injury of the hot pipe.

Final Answer:

Cocaine

Quick Tip: Smoking this drug through a hot pipe can burn and blacken the tongue.

14. A 25 yr old male experienced severe flushing, fall in blood pressure after intake of alcohol. The above described attack can be precipitated by the simultaneous intake of all the following drugs along with alcohol EXCEPT which?

(A) Cefamandole

(B) Metronidazole

(C) Dexamethasone

(D) Chlorpropamide

Correct Answer: (C) Dexamethasone

Solution:

Step 1: Name the reaction being described.
Flushing, palpitations, and a drop in blood pressure after drinking alcohol, while on certain drugs, describes a disulfiram-like reaction.

Step 2: Recall the mechanism.
Alcohol is normally broken down in two steps, first to acetaldehyde by alcohol dehydrogenase, then acetaldehyde is broken down further by aldehyde dehydrogenase. Certain drugs block this second enzyme, so

acetaldehyde builds up in the blood. This build up causes the flushing, headache, nausea, and fall in blood pressure seen in a disulfiram-like reaction.

Step 3: Check each drug against this mechanism.
Cefamandole is a cephalosporin that carries a side chain (the MTT or NMTT group) known to block aldehyde dehydrogenase, so it can trigger this reaction with alcohol.
Metronidazole is well known to block aldehyde dehydrogenase and produce this same reaction, so patients are always told to avoid alcohol while taking it.
Chlorpropamide, an older sulfonylurea used for diabetes, also blocks this enzyme in some patients and can cause facial flushing with alcohol.
Dexamethasone is a corticosteroid. It works through steroid hormone receptors and has no action on aldehyde dehydrogenase, so it does not cause this reaction with alcohol.

Step 4: Conclude.
Since cefamandole, metronidazole, and chlorpropamide can all precipitate the reaction but dexamethasone cannot, dexamethasone is the exception.

Final Answer:

Dexamethasone

Quick Tip: This drug does not block aldehyde dehydrogenase, unlike the others.

15. A patient with a history of asthma develops a respiratory tract infection. He is on theophylline. Which of the following antibiotics may precipitate theophylline toxicity?

(A) Erythromycin

(B) Sparfloxacin

(C) Ampicillin

(D) Cotrimoxazole

Correct Answer: (A) Erythromycin

Solution:

Step 1: Identify the drug interaction being tested.
The patient is on theophylline for asthma and now needs an antibiotic for a chest infection. Theophylline has a narrow therapeutic range, so any drug that slows down its breakdown in the liver can push levels into the toxic range.

Step 2: Recall how theophylline is cleared.
Theophylline is broken down mainly by the liver enzyme CYP1A2, with some help from CYP3A4. Anything that blocks these enzymes lets theophylline build up in the blood.

Step 3: Check erythromycin.
Erythromycin is a macrolide antibiotic that inhibits CYP3A4 and, to a lesser extent, CYP1A2. This slows theophylline clearance and can raise its blood level enough to cause nausea, vomiting, tremor, fast heart rate, or seizures, the classic picture of theophylline toxicity.

Step 4: Rule out the other antibiotics.
Sparfloxacin is a fluoroquinolone; ciprofloxacin is the

fluoroquinolone best known to raise theophylline levels, but sparfloxacin does not carry this strong interaction. Ampicillin is a penicillin and does not act on liver enzymes, so it does not change theophylline levels. Cotrimoxazole (trimethoprim plus sulfamethoxazole) also does not meaningfully affect theophylline metabolism.

Step 5: Final answer.
Erythromycin is the antibiotic most likely to precipitate theophylline toxicity in this patient.

Erythromycin

Quick Tip: Macrolides like erythromycin block the liver enzyme that breaks down theophylline.

16. In which of the following conditions is granulomatous vasculitis NOT seen?

(A) Microscopic Polyangiitis

(B) Wegener's Granulomatosis

(C) Giant Cell Arteritis

(D) Churg-Strauss Syndrome

Correct Answer: (A) Microscopic Polyangiitis

Solution:

Step 1: Recall what granulomatous vasculitis means.
Granulomatous vasculitis is inflammation of blood vessel walls with

granuloma formation, clusters of macrophages, giant cells, and lymphocytes trying to wall off an injury.

Step 2: List the vasculitides that are granulomatous.
Giant cell (temporal) arteritis shows granulomas with multinucleated giant cells in the media of large arteries. Wegener's granulomatosis, now called granulomatosis with polyangiitis, has necrotising granulomas in the respiratory tract along with small vessel vasculitis. Churg-Strauss syndrome, now called eosinophilic granulomatosis with polyangiitis, also forms granulomas, along with heavy eosinophil infiltration and asthma.

Step 3: Check microscopic polyangiitis.
Microscopic polyangiitis is a pauci-immune, ANCA-associated small vessel vasculitis. It causes necrotising inflammation of capillaries, venules, and arterioles, but it does not form granulomas. This is the key feature that separates it from Wegener's and Churg-Strauss, both of which are also ANCA-associated but do form granulomas.

Step 4: Rule out the other options.
Since giant cell arteritis, Wegener's granulomatosis, and Churg-Strauss syndrome all show granuloma formation, the only condition left without granulomas is microscopic polyangiitis.

Step 5: Final answer.
Granulomatous vasculitis is not seen in microscopic polyangiitis.

Microscopic Polyangiitis

Quick Tip: Of the ANCA-associated vasculitides, this one is pauci-immune without granuloma formation.

17. A 30 year old patient with nephrotic syndrome has been on steroids for 14 years. He has bilateral difficulty in abduction and internal rotation of the hip, and attempting to flex the hip results in abduction. What is the cause?

(A) Avascular Necrosis of Femur Head

(B) Septic Arthritis

(C) Renal Osteodystrophy

(D) Heterotopic Calcification

Correct Answer: (A) Avascular Necrosis of Femur Head

Solution:

Step 1: Note the risk factor.
Long term, high dose steroid use is one of the leading causes of avascular necrosis (AVN), also called osteonecrosis, of the femoral head. Fourteen years of steroid therapy for nephrotic syndrome puts this patient at high risk.

Step 2: Read the clinical clues.
AVN of the femoral head classically presents with groin or hip pain and stiffness. Internal rotation and abduction are usually the first movements lost because the anterolateral part of the femoral head, the main weight bearing area, collapses first. The finding that flexion is coupled with obligatory abduction, the leg swinging outward as the patient tries to flex the hip, is a well described sign of femoral head

collapse changing the joint's mechanics.

Step 3: Rule out septic arthritis.
Septic arthritis is usually acute, with fever, severe pain, warmth, and swelling of a single joint, not a chronic bilateral process developing over years of steroid use.

Step 4: Rule out renal osteodystrophy.
Renal osteodystrophy causes bone pain and deformity from disturbed calcium, phosphate, and vitamin D handling in chronic kidney disease, but it does not produce this specific hip flexion-abduction sign, and nephrotic syndrome alone does not mean the kidneys have failed.

Step 5: Rule out heterotopic calcification.
Heterotopic calcification means bone forming in soft tissue, usually after trauma, burns, or spinal cord injury, and is not linked to long term steroid use in this way.

Step 6: Final answer.
The bilateral hip findings after 14 years of steroids point to avascular necrosis of the femoral head.

Avascular Necrosis of Femur Head

Quick Tip: Long-term steroids are a classic cause of femoral head osteonecrosis.

18. In which of the following conditions is an elevated HCG level NOT seen?

(A) Choriocarcinoma

(B) Polyembryoma

(C) Endodermal Sinus Tumor

(D) Embryonal Carcinoma

Correct Answer: (C) Endodermal Sinus Tumor

Solution:

Step 1: Recall which germ cell tumor elements secrete which markers.

Germ cell tumors are graded by which primitive tissue they mimic, and each type tends to secrete a specific tumor marker into the blood.

Step 2: Check choriocarcinoma.

Choriocarcinoma mimics placental trophoblastic tissue, made of cytotrophoblast and syncytiotrophoblast. The syncytiotrophoblast cells make human chorionic gonadotropin (HCG), the same hormone raised in a normal pregnancy. So HCG is always high in choriocarcinoma.

Step 3: Check polyembryoma.

A polyembryoma contains embryoid bodies that resemble early embryos, with elements of both yolk sac and trophoblastic tissue. Because it carries trophoblastic tissue, it can also raise HCG, and its yolk sac component raises AFP as well.

Step 4: Check embryonal carcinoma.

Embryonal carcinoma is made of primitive, undifferentiated cells. It

can secrete AFP, HCG, or both, because scattered syncytiotrophoblast-like giant cells are often present within it.

Step 5: Check endodermal sinus tumor.
The endodermal sinus tumor, also called the yolk sac tumor, is the malignant counterpart of the fetal yolk sac. It classically secretes alpha-fetoprotein (AFP), not HCG, since it lacks trophoblastic differentiation.

Step 6: Final answer.
Of the four, only the endodermal sinus tumor does not raise HCG; it raises AFP instead.

Endodermal Sinus Tumor

Quick Tip: Yolk sac (endodermal sinus) tumor secretes AFP, not HCG; trophoblastic tissue is what makes HCG.

19. What could be the cause of tall QRS complexes with coarse facial features and hepatosplenomegaly?

(A) Glycogen Storage Disease Type II

(B) Marfan's Syndrome

(C) Romano-Ward Syndrome

(D) Pompe's Disease

Correct Answer: (D) Pompe's Disease

Solution:

Step 1: Break down the clinical picture.
The child has tall QRS complexes on ECG, a sign of a thick, glycogen-loaded heart muscle, along with coarse facial features and an enlarged liver and spleen. This combination points toward a storage disorder that fills organs, including the heart, with abnormal material.

Step 2: Recall Pompe's disease.
Pompe's disease is caused by a deficiency of acid alpha-glucosidase (acid maltase), the lysosomal enzyme that breaks down glycogen. Glycogen then piles up inside lysosomes of the heart, skeletal muscle, and liver. Infants develop severe cardiomegaly with tall QRS voltages, hypotonia (floppy baby), macroglossia and coarse facies, and hepatomegaly. Because of this systemic buildup, Pompe's disease is classified as Glycogen Storage Disease Type II; the two names describe the same condition, which is why option (A) and option (D) point to the same disease here.

Step 3: Rule out Marfan syndrome.
Marfan syndrome is a connective tissue disorder from a fibrillin-1 defect. It causes tall stature, long limbs, lens dislocation, and aortic root dilation, not coarse features, hepatosplenomegaly, or tall QRS from glycogen buildup.

Step 4: Rule out Romano-Ward syndrome.
Romano-Ward syndrome is an inherited long QT syndrome without deafness. It causes a prolonged QT interval and risk of torsades de pointes, not tall QRS voltage, coarse facies, or organomegaly.

Step 5: Final answer.
The clinical triad of tall QRS, coarse features, and

hepatosplenomegaly fits Pompe's disease, matching the answer key's option (D).

Pompe's Disease

Quick Tip: Coarse facies, hepatosplenomegaly, and tall QRS in an infant point to a lysosomal glycogen storage disorder of the heart (Pompe's disease).

20. What is the treatment for torsades de pointes in a patient who has had a prolonged QT interval since birth?

(A) Magnesium Sulphate

(B) Overdrive Pacing

(C) Isoproterenol

(D) Metoprolol

Correct Answer: (D) Metoprolol

Solution:

Step 1: Separate congenital from acquired long QT.
Torsades de pointes can occur in two settings: acquired long QT (from drugs, low potassium or magnesium, or bradycardia) and congenital long QT, present from birth due to an inherited ion channel defect. The management of the two overlaps for the emergency but differs for ongoing prevention.

Step 2: Recall acute, general treatment of torsades.
Any episode of torsades de pointes, congenital or acquired, is treated acutely with IV magnesium sulphate first, since it stabilises the

cardiac membrane regardless of the cause. Overdrive pacing and isoproterenol are used mainly in acquired, pause-dependent torsades, where speeding up the heart rate shortens the QT interval and breaks the arrhythmia.

Step 3: Note why isoproterenol is wrong here.
Isoproterenol is a beta agonist. In congenital long QT syndrome, especially the common LQT1 type, sympathetic (catecholamine) surges are what trigger torsades in the first place. Giving isoproterenol to a patient with congenital long QT can worsen the arrhythmia rather than help it, so it is avoided here even though it helps in acquired, bradycardia-related torsades.

Step 4: Recall the mainstay for congenital long QT.
For patients with congenital long QT syndrome, the definitive, guideline-backed treatment to prevent torsades is a beta-blocker, traditionally propranolol, though metoprolol is also used, since it blunts the sympathetic trigger that provokes the arrhythmia in this inherited condition.

Step 5: Rule out magnesium and pacing as the specific answer.
Magnesium sulphate and overdrive pacing treat the acute rhythm disturbance but are not the answer suited to managing a lifelong, inherited channelopathy going forward.

Step 6: Final answer.
For a patient with congenital long QT syndrome, the beta-blocker metoprolol is the appropriate choice.

Metoprolol

Quick Tip: Congenital long QT syndrome is managed with beta-blockers; catecholamine drugs like isoproterenol can worsen it.

21. Which of the following is NOT a congenital myopathy?

(A) Central Core Disease

(B) Nemaline Myopathy

(C) Centronuclear Myopathy

(D) Z Band Myopathy

Correct Answer: (D) Z Band Myopathy

Solution:

Step 1: Recall the classic congenital myopathies.
Congenital myopathies are a group of inherited muscle disorders that present at birth or in infancy with hypotonia and weakness, defined by a specific structural abnormality seen on muscle biopsy rather than by muscle fiber death or dystrophic change.

Step 2: Check central core disease.
Central core disease shows a well demarcated central area within the muscle fiber that lacks mitochondria and oxidative enzyme activity, seen on ATPase and NADH stains. It is a classic congenital myopathy, linked to the ryanodine receptor gene RYR1, and to malignant hyperthermia risk.

Step 3: Check nemaline (rod body) myopathy.
This condition shows rod-shaped structures (nemaline bodies) within the muscle fibers, derived from Z-line material, seen with modified

Gomori trichrome stain. It is one of the three classic congenital myopathies.

Step 4: Check centronuclear (myotubular) myopathy.
Here, muscle fiber nuclei sit in the centre of the fiber instead of the periphery, resembling fetal myotubes, hence the name myotubular myopathy. This is the third classic congenital myopathy.

Step 5: Check Z-band myopathy.
Z-band, or myofibrillar, myopathy is a separate, distinct group of muscle disorders where the Z-disc and its associated proteins break down and desmin-related material builds up. It usually presents later in life, in adults, with a distal or limb-girdle pattern of weakness, rather than as a congenital muscle disorder of infancy. It is not classified among the traditional congenital myopathies.

Step 6: Final answer.
Central core disease, nemaline myopathy, and centronuclear myopathy are all classic congenital myopathies. Z-band myopathy is not.

Z Band Myopathy

Quick Tip: Central core, nemaline, and centronuclear myopathy are the classic congenital myopathy triad; Z band (myofibrillar) myopathy is a separate, usually adult-onset group.

22. In which of these conditions may CPK be raised?

(A) Muscle disease

(B) Liver cirrhosis

(C) Biliary colic

(D) Amyotrophic lateral sclerosis

Correct Answer: (A) Muscle disease

Solution:

Step 1: Understanding the Question:
CPK, or creatine phosphokinase, is an enzyme found mainly inside muscle cells. The question asks which of the four listed conditions raises its blood level.

Step 2: Key Concept:
CPK sits inside skeletal muscle, heart muscle, and brain tissue. When any of these cells break down or get damaged, the enzyme leaks out into the blood and the serum CPK level goes up. So the level rises with muscle injury, not with problems in organs that do not hold much CPK.

Step 3: Detailed Explanation:
Muscle disease, such as myositis, muscular dystrophy, or a heart attack, damages muscle fibers directly, so CPK pours into the blood and the level rises sharply.
Liver cirrhosis mainly raises liver enzymes like ALT, AST, and ALP, along with bilirubin. It does not damage muscle, so CPK stays normal.
Biliary colic points to a gallstone blocking the bile duct. This raises ALP, GGT, and bilirubin, again with no effect on muscle enzymes.
Amyotrophic lateral sclerosis mainly destroys the nerve cells that drive muscles (anterior horn cells), not the muscle fibers themselves.

CPK in ALS is usually normal or only mildly raised, nowhere near the rise seen in true muscle disease.

Step 4: Final Answer:
Only direct muscle damage explains a clearly raised CPK, so the answer is muscle disease.

Quick Tip: CPK sits inside muscle cells; think about which option actually damages muscle fibers.

23. A patient with hypertension has a serum urea of 55 mg/dL and serum creatinine of 5.6 mg/dL, indicating renal failure. Which of the following anti-tubercular drugs does NOT require the dose to be reduced in this patient?

(A) INH

(B) Rifampin

(C) Pyrazinamide

(D) Ethambutol

Correct Answer: (B) Rifampin

Solution:

Step 1: Understanding the Question:
The patient has renal failure, shown by the high urea and creatinine. We need to find which anti-tubercular drug can be given at its normal dose without adjusting for poor kidney function.

Step 2: Key Concept:

A drug needs its dose lowered in renal failure only if the kidney is its main route of clearance. If a drug is cleared mainly by the liver and bile, poor kidney function does not cause it to build up, so the normal dose stays safe.

Step 3: Detailed Explanation:
Rifampin is cleared almost entirely through the liver into bile, then out in the stool. Kidney function barely affects its levels, so no dose change is needed even in advanced renal failure.
INH is mostly broken down by the liver too, but a meaningful part of its metabolite load still depends on kidney clearance in slow acetylators, so caution and sometimes a lower dose is advised in severe renal failure.
Pyrazinamide is handled by the kidney to a large extent, and its metabolites build up in renal failure, raising the risk of toxicity, so its dose or dosing interval must be adjusted.
Ethambutol is excreted largely unchanged by the kidney. In renal failure it accumulates and raises the risk of optic neuritis, so its dose must be reduced or the interval extended.

Step 4: Final Answer:
Since rifampin clears through the liver and bile, it needs no dose reduction in renal failure.

Quick Tip: Ask which drug is cleared by the liver and bile rather than the kidney.

24. What drug should be avoided in the case of an HIV patient who is receiving zalcitabine, indinavir and lamivudine?

(A) INH

(B) Rifampin

(C) PZA

(D) Ethambutol

Correct Answer: (B) Rifampin

Solution:

Step 1: Understanding the Question:
This patient is on antiretroviral therapy that includes indinavir, a protease inhibitor, along with zalcitabine and lamivudine. We need to find which anti-tubercular drug is unsafe to add to this combination.

Step 2: Key Concept:
Protease inhibitors like indinavir are broken down by the liver enzyme CYP3A4. Any drug that strongly switches on (induces) this enzyme will speed up the breakdown of indinavir, drop its blood level, and risk failure of the HIV treatment along with drug resistance.

Step 3: Detailed Explanation:
Rifampin is one of the strongest known inducers of CYP3A4. Giving it with indinavir can cut the protease inhibitor's blood level so much that the antiretroviral regimen stops working properly. Because of this, rifampin is avoided in patients on protease inhibitors, and rifabutin, a much weaker enzyme inducer, is used in its place when anti-TB cover is needed.
INH is mainly cleared by liver acetylation and does not meaningfully induce CYP3A4, so it does not threaten indinavir levels.
Pyrazinamide (PZA) has no major interaction with protease inhibitors through this enzyme pathway.

Ethambutol is excreted mostly by the kidney and also does not induce CYP3A4.

Step 4: Final Answer:
Rifampin should be avoided because it drives down indinavir levels through CYP3A4 induction.

Quick Tip: Think about which anti-TB drug strongly induces the liver enzyme that breaks down protease inhibitors.

25. What is NOT true about multiple myeloma?

(A) It is more common in those over 50 years of age

(B) It arises from clones of plasma cells

(C) Bence Jones proteins are abnormal whole immunoglobulins found in urine

(D) There is a predisposition to amyloidosis

Correct Answer: (C) Bence Jones proteins are abnormal whole immunoglobulins found in urine

Solution:

Step 1: Understanding the Question:
We need to pick the one statement about multiple myeloma that is false.

Step 2: Key Concept:
Multiple myeloma is a cancer of plasma cells, the antibody-making cells of the bone marrow. A single abnormal plasma cell multiplies

into a clone, and that clone churns out one type of antibody or antibody fragment in huge amounts.

Step 3: Detailed Explanation:
Multiple myeloma is indeed a disease of older adults, most often diagnosed after age 50, so this statement is true.
It truly arises from a clone of plasma cells, all descended from one transformed cell, so this statement is also true.
Bence Jones proteins are not whole antibody molecules. A whole immunoglobulin has two heavy chains and two light chains joined together. In myeloma, the clone often makes far more light chains than heavy chains, and the extra free light chains are small enough to pass through the kidney filter into urine. That is exactly what a Bence Jones protein is, a free light chain, not a whole immunoglobulin. So calling it a whole Ig is wrong, which makes this statement false.
Myeloma does raise the risk of amyloidosis, because the excess light chains can misfold and deposit as amyloid in tissues (AL amyloidosis), so this last statement is true.

Step 4: Final Answer:
The false statement is that Bence Jones proteins are abnormal whole immunoglobulins.

Quick Tip: Remember what a Bence Jones protein actually is: a free light chain, not a full antibody.

26. What is the likely diagnosis in a patient who has low back ache, L3 tenderness, and the following data: total proteins 8.9 g/dL, albumin:globulin ratio 2.9/5.9 (reversed), serum creatinine 5.5 mg/dL, TLC 4500/mm3, DLC neutrophils 55%, lymphocytes 40%, eosinophils 2%; blood urea was 93 mg/dL and serum creatinine was 1.2 mg/dL; ESR was 90 mm in the first hour?

(A) Waldenstrom's macroglobulinemia

(B) Multiple myeloma

(C) Amyloidosis

(D) Bone secondaries

Correct Answer: (B) Multiple myeloma

Solution:

Step 1: Understanding the Question:
We need to fit a back pain patient with a set of lab clues to one diagnosis: raised total protein with a reversed albumin to globulin ratio, kidney impairment, a very high ESR, and vertebral tenderness.

Step 2: Key Concept:
A reversed albumin to globulin ratio, where globulin is higher than albumin, points to a large amount of abnormal protein (paraprotein) in the blood, usually made by a clone of plasma cells. A very high ESR happens because these abnormal proteins make red cells stick together and fall faster (rouleaux formation). Back pain with vertebral tenderness in this setting suggests bone involvement by the same disease process.

Step 3: Detailed Explanation:
Multiple myeloma fits every clue here: the plasma cell clone raises

total globulin, flips the albumin to globulin ratio, drives the ESR to very high levels, damages the kidney through light chain deposits and cast formation (matching the raised creatinine), and causes lytic bone lesions that produce localized back pain and vertebral tenderness, which explains the L3 tenderness.

Waldenstrom's macroglobulinemia is also a clonal plasma-cell-related disorder, but it centers on IgM and typically causes hyperviscosity symptoms such as blurred vision and bleeding, with lytic bone lesions and vertebral tenderness being uncommon, so it fits this vignette poorly.

Amyloidosis can follow myeloma, but on its own it does not produce a sharply reversed albumin to globulin ratio from paraprotein overproduction, and vertebral tenderness is not its typical presentation.

Bone secondaries, meaning metastatic cancer in bone, cause bone pain but do not typically produce a reversed albumin to globulin ratio or such a striking rise in ESR, since there is no paraprotein being made.

Step 4: Final Answer:

The combination of reversed albumin to globulin ratio, very high ESR, renal impairment, and vertebral tenderness points to multiple myeloma.

Quick Tip: A reversed albumin to globulin ratio with a very high ESR and bone tenderness points to a plasma cell disorder with bone involvement.

27. A patient has target cells, nucleated RBCs, and microcytic hypochromic anemia on the peripheral smear, along with a positive family history. What is the investigation of choice?

(A) Coombs test

(B) Osmotic fragility

(C) Hb electrophoresis

(D) Sucrose lysis test

Correct Answer: (C) Hb electrophoresis

Solution:

Step 1: Understanding the Question:
The smear shows target cells, nucleated red cells, and a microcytic hypochromic anemia, and there is a positive family history. We need to pick the test that will confirm the diagnosis.

Step 2: Key Concept:
This exact combination, target cells plus nucleated RBCs plus microcytic hypochromic anemia plus a family history, is the classic picture of an inherited hemoglobin disorder, most often thalassemia. Nucleated RBCs and target cells appear because the bone marrow is under stress trying to compensate for ineffective red cell production.

Step 3: Detailed Explanation:
Hb electrophoresis separates the different hemoglobin types (HbA, HbA2, HbF) by their electrical charge and directly shows the abnormal pattern seen in thalassemia, such as a raised HbA2 or HbF. This makes the diagnosis certain, so it is the investigation of choice. Coombs test looks for antibody coating on red cells and is used to diagnose autoimmune hemolytic anemia, which is not suggested by a

family history or this smear pattern.
Osmotic fragility testing checks how easily red cells burst in dilute saline. In thalassemia the fragility is actually decreased because target cells resist lysis, but this test is a rough screening tool, not specific enough to confirm the diagnosis.
Sucrose lysis test is used to screen for paroxysmal nocturnal hemoglobinuria, an acquired disorder unrelated to this inherited picture.

Step 4: Final Answer:
Hb electrophoresis is the test that confirms the diagnosis here.

Quick Tip: Target cells, nucleated RBCs and a family history point to an inherited hemoglobin disorder; pick the test that reads the hemoglobin pattern directly.

28. Which of these is NOT a clinical feature of hypercarbia?

(A) Miosis

(B) Cold clammy extremities

(C) Bradycardia

(D) Hypertension

Correct Answer: (B) Cold clammy extremities ; (C) Bradycardia

Solution:

Step 1: Understanding the Question:
Hypercarbia means a raised level of carbon dioxide in the blood. We need to pick the option or options that are NOT features of this state.

Step 2: Key Concept:

A rise in carbon dioxide directly dilates blood vessels, especially in the skin and brain, and also fires up the sympathetic nervous system. So the expected picture is warm, well-perfused skin with a fast heart rate and, early on, a raised blood pressure, along with central nervous system effects from the high CO_2 itself.

Step 3: Detailed Explanation:

Miosis, or small pupils, can occur with marked hypercarbia because high CO_2 has a direct central nervous system depressant effect, similar to what is seen in CO_2 narcosis, so this is counted as a genuine feature.

Cold clammy extremities are what you see when the body clamps down blood vessels in the skin, such as in shock or hypoxia. Hypercarbia does the opposite: it dilates vessels, giving warm, flushed, well-perfused extremities. So cold clammy extremities are not a feature of hypercarbia.

Bradycardia is not the expected early response either. The sympathetic surge from rising CO_2 usually speeds the heart up, producing tachycardia and a bounding pulse. A slow heart rate is more a sign of a late, terminal event or a separate problem such as raised intracranial pressure, not a routine feature of hypercarbia.

Hypertension is a genuine feature, since the same sympathetic surge and direct CO_2 effect on blood vessels raise the blood pressure, at least in the earlier stage.

Step 4: Final Answer:

Cold clammy extremities and bradycardia are the two features that do NOT belong to hypercarbia.

Quick Tip: Hypercarbia dilates vessels and drives the sympathetic system; think warm skin and a fast heart, not cold skin and a slow heart.

29. A 25 year old male has a Mantoux test reading of 14 x 17 mm, along with weight loss, intermittent low grade fever, and hemoptysis for the past 4 months. Sputum smear is negative for acid fast bacilli (AFB) and the ESR is raised. What is the likely diagnosis?

(A) Pulmonary tuberculosis (PTB)

(B) Viral pneumonia

(C) Fungal pneumonia

(D) Bronchogenic carcinoma

Correct Answer: (A) Pulmonary tuberculosis (PTB)

Solution:

Step 1: List the clues in the case.

A young man has a strongly positive Mantoux test (14 x 17 mm is well above the 10 mm cut off used for a positive result in a high burden country), weight loss, low grade fever that comes and goes, and hemoptysis that has lasted 4 months. The ESR is raised, which points to an ongoing inflammatory or infective process.

Step 2: Explain why a negative AFB smear does not rule out tuberculosis.

Sputum smear microscopy only picks up TB when there are at least 5,000 to 10,000 bacilli per ml of sputum. Many active cases, especially early or paucibacillary disease, stay smear negative on one or two samples even though the patient truly has tuberculosis. So a

negative smear here does not exclude PTB, it just means repeat samples, culture, or a test like CBNAAT/PCR are needed to confirm it.

Step 3: Match the clinical picture to pulmonary tuberculosis.
The combination of chronic low grade fever, weight loss, hemoptysis over months, a strongly positive Mantoux, and raised ESR is the classic textbook picture of pulmonary tuberculosis. This pattern is far more typical of PTB than of a viral or fungal chest infection.

Step 4: Rule out the other options.
Viral pneumonia usually runs an acute course of days, not 4 months, and does not produce this pattern of hemoptysis with weight loss. Fungal pneumonia (such as aspergillosis) is uncommon in a healthy 25 year old without an immunocompromised state or structural lung disease. Bronchogenic carcinoma is very unlikely at age 25, it usually appears after age 40, most often in long term smokers.

Final Answer:
The clinical picture fits pulmonary tuberculosis best, even with a negative AFB smear.

Pulmonary tuberculosis (PTB)

Quick Tip: A strongly positive Mantoux with chronic fever, weight loss, and hemoptysis over months points to tuberculosis, even if the AFB smear is negative.

30. Which of the following statements is NOT true about prion disease?

(A) About 10% of patients have myoclonus

(B) It is caused by an infectious protein

(C) Dementia is a universal feature

(D) Brain biopsy is diagnostic

Correct Answer: (A) About 10% of patients have myoclonus ; (C) Dementia is a universal feature

Solution:

Step 1: Recall what a prion disease is.
Prion diseases (such as Creutzfeldt-Jakob disease, CJD) are caused by an abnormally folded, infectious form of a normal cell surface protein called PrP. This misfolded protein (PrPSc) converts normal PrP into more of the abnormal form, without any DNA or RNA involved. So the statement that it is caused by an infectious protein is TRUE.

Step 2: Check the myoclonus figure.
Myoclonus (sudden brief muscle jerks) is a very common finding in CJD, seen in the large majority of patients, commonly quoted at around 80 to 90% at some point in the illness, not just 10%. So "about 10% have myoclonus" understates how common it is and is NOT a true statement.

Step 3: Check the dementia claim.
Rapidly progressive dementia is the hallmark of classic CJD, but calling it a strictly universal feature across all prion diseases overreaches. Some prion diseases, and even some early or atypical CJD presentations, are dominated by ataxia, visual disturbance, or behavioral change before dementia becomes obvious, and a few variants (like fatal familial insomnia) present mainly with sleep and

autonomic disturbance rather than dementia as the leading feature. So calling dementia "universal" is NOT strictly true.

Step 4: Check the brain biopsy claim.
Brain biopsy showing spongiform change, neuronal loss, and gliosis is the definitive way to confirm the diagnosis while the patient is alive, so this statement is TRUE (biopsy is diagnostic, even though it is rarely done because of the risk of prion transmission to instruments and staff).

Step 5: Put together the false statements.
The two statements that are NOT true are the 10% myoclonus figure (it is much higher) and calling dementia a universal feature (it is common but not invariable across all prion diseases).

10% have myoclonus AND dementia is universal (both NOT true)

Quick Tip: Myoclonus is far more common than 10% in prion disease, and dementia, though typical, is not seen in every case or every prion disease.

31. What is the likely diagnosis in a 25 year old who develops hematuria 3 days after an upper respiratory tract infection (URI)?

(A) IgA nephropathy

(B) Post-streptococcal glomerulonephritis (PSGN)

(C) Henoch-Schonlein purpura (HSP)

(D) Hemolytic uremic syndrome (HUS)

Correct Answer: (A) IgA nephropathy

Solution:

Step 1: Note the key timing clue.
The hematuria starts only 3 days after the URI begins. This short gap between the infection and the kidney symptom is the single most useful clue in this question.

Step 2: Recall the classic timing for IgA nephropathy.
IgA nephropathy (Berger disease) is well known for synpharyngitic hematuria, meaning gross or microscopic hematuria that appears within 1 to 3 days of an upper respiratory or gastrointestinal infection. This happens because IgA immune complexes that were already forming are boosted quickly by the new infection and deposit in the glomerular mesangium.

Step 3: Compare with post-streptococcal glomerulonephritis.
PSGN follows a much longer latent period, usually 10 to 14 days after a streptococcal throat infection or 3 to 6 weeks after a skin infection, because it needs time to build the immune complexes that later deposit in the kidney. A 3 day gap is too short for PSGN.

Step 4: Compare with HSP and HUS.
Henoch-Schonlein purpura is IgA vasculitis and can also follow a URI, but it comes with a purpuric skin rash, joint pain, and abdominal pain, none of which are mentioned here, so it is less likely to be the answer being tested. HUS typically follows a diarrheal illness (often E. coli O157:H7) and presents with the triad of hemolytic anemia, thrombocytopenia, and acute kidney injury, not a plain URI history.

Step 5: Final answer.

The short 3 day gap between URI and hematuria is the textbook pattern for IgA nephropathy.

IgA nephropathy

Quick Tip: Hematuria within 1 to 3 days of a URI is the classic "synpharyngitic" pattern of IgA nephropathy.

32. An individual who underwent renal transplantation one year ago is found positive for HBsAg and HCV. Treatment with which of the following will give this patient maximum benefit?

(A) Lamivudine and interferon (IFN)

(B) Lamivudine alone

(C) Ribavirin

(D) Interferon (IFN)

Correct Answer: (B) Lamivudine alone

Solution:

Step 1: Note the special situation, a transplant recipient.
This patient has a functioning kidney transplant, so they are on long term immunosuppressive drugs to prevent rejection. Any antiviral choice has to be safe in that setting, not just effective against the virus.

Step 2: Recall why interferon is avoided after transplant.
Interferon works partly by boosting the immune system's antiviral response. In a transplant recipient, stimulating the immune system

this way can trigger acute graft rejection, so interferon (alone or combined with lamivudine) is generally avoided in solid organ transplant recipients. This rules out the two options that contain interferon.

Step 3: Recall why ribavirin alone is not the answer.
Ribavirin is used for hepatitis C, almost always combined with interferon or another agent, and ribavirin monotherapy is not an effective long term strategy on its own. It also does not address the HBsAg positive status here.

Step 4: Explain why lamivudine alone fits.
Lamivudine is a nucleoside analog that directly blocks the hepatitis B viral polymerase without needing to activate the immune system, so it does not carry the same rejection risk as interferon. This makes it the safer and more practical choice for treating chronic HBV in a transplant recipient.

Step 5: Final answer.
Among the given options, lamivudine alone gives the best benefit with the lowest rejection risk in this transplant patient. Current practice now generally favors newer nucleoside/nucleotide analogs such as entecavir or tenofovir over lamivudine because of lamivudine's higher rate of drug resistance with long term use, but the underlying principle, avoiding interferon after transplant, still holds.

Lamivudine alone

Quick Tip: Interferon is avoided after organ transplant because it can trigger rejection; lamivudine treats HBV without that risk.

33. In which of the following conditions should propranolol NOT be used?

(A) Asthma

(B) Panic attack

(C) Premature ventricular contractions (PVCs)

(D) Hypertrophic cardiomyopathy

Correct Answer: (A) Asthma

Solution:

Step 1: Recall what propranolol does.
Propranolol is a non-selective beta blocker, meaning it blocks both beta-1 receptors (mainly in the heart) and beta-2 receptors (mainly in the smooth muscle of the airways and blood vessels).

Step 2: Explain the effect of beta-2 blockade on the airway.
Beta-2 receptors in bronchial smooth muscle normally cause bronchodilation when stimulated. Blocking them with a non-selective drug like propranolol removes this protective bronchodilator tone and can trigger bronchospasm, which is dangerous in a patient with asthma. This is why propranolol (and other non-selective beta blockers) is contraindicated in asthma.

Step 3: Check the other conditions.
In panic attacks, beta blockers like propranolol are actually used to control the physical symptoms of anxiety, such as tremor and palpitations. In PVCs, beta blockers can suppress the abnormal ectopic beats by reducing sympathetic drive to the heart. In hypertrophic cardiomyopathy, beta blockers reduce the force and rate of contraction, which relieves the left ventricular outflow obstruction,

and are first line treatment.

Step 4: Final conclusion.
Among the choices, propranolol is helpful or standard treatment in panic attack, PVCs, and hypertrophic cardiomyopathy, but it is harmful in asthma because of bronchospasm risk.

Asthma

Quick Tip: Non-selective beta blockade removes bronchodilator tone and can trigger bronchospasm, so propranolol is avoided in asthma.

34. Which of the following is NOT a CNS anomaly seen in HIV infection?

(A) Perivascular infiltration

(B) Microglial nodules

(C) Vascular myelopathy of the posterior column

(D) Temporal lobe involvement

Correct Answer: (D) Temporal lobe involvement

Solution:

Step 1: Recall the typical CNS pathology of HIV.
HIV can directly infect the brain and cause HIV encephalitis (part of HIV-associated neurocognitive disorder). The classic pathological findings include perivascular cuffing by inflammatory cells and multinucleated giant cells, microglial nodules scattered through the white matter, and, in the spinal cord, a vacuolar myelopathy that affects the posterior and lateral columns, resembling the changes seen

in vitamin B12 deficiency.

Step 2: Match options (A), (B), and (C) to this picture.
Perivascular infiltration and microglial nodules are the hallmark microscopic findings of HIV encephalitis. Vacuolar myelopathy involving the posterior columns is a well recognized spinal cord complication of HIV. So all three of these are accepted CNS findings in HIV.

Step 3: Explain why temporal lobe involvement does not fit.
Prominent temporal lobe involvement, especially with hemorrhagic necrosis, is the classic finding in herpes simplex virus (HSV) encephalitis, not HIV. HIV encephalitis tends to be more diffuse, involving white matter and subcortical structures, rather than focusing on the temporal lobes.

Step 4: Final conclusion.
Since temporal lobe involvement is not a typical feature of HIV CNS disease, it is the correct answer to "which is NOT seen in HIV".

Temporal lobe involvement

Quick Tip: Temporal lobe involvement is classic for HSV encephalitis, not HIV, which instead shows perivascular infiltration, microglial nodules, and vacuolar myelopathy.

35. Which of the following is NOT true regarding toxoplasmosis?

(A) IgG antibody indicates congenital infection

(B) Most infections are anthroponotic

(C) Adult infections are mainly symptomatic

(D) Toxoplasma encephalitis occurs in immunocompetent persons

Correct Answer: (C) Adult infections are mainly symptomatic

Solution:

Step 1: Recall the basic biology of Toxoplasma gondii infection.
Toxoplasma gondii is an intracellular protozoan parasite. Infection in an immunocompetent host is usually mild or completely silent, and the parasite then persists as dormant tissue cysts for life, kept in check by the immune system.

Step 2: Check the claim that adult infections are mainly symptomatic.
In reality, the large majority of adult toxoplasma infections, roughly 80 to 90%, are asymptomatic or produce only mild, flu-like symptoms that often go unnoticed. Only a minority develop clear symptoms like lymphadenopathy or fever. So the statement "adult infections are mainly symptomatic" is NOT true, it is the reverse of what actually happens.

Step 3: Check the IgG and congenital infection claim.
Maternal IgG crosses the placenta, so a newborn will show positive IgG regardless of true infection at birth. But if that IgG persists beyond the time maternal antibody should have cleared (typically beyond about 12 months), it points to the infant's own ongoing antibody production, which supports a diagnosis of congenital infection. In that sense, tracking IgG over time is a genuine and

accepted part of diagnosing congenital toxoplasmosis.

Step 4: Check the transmission and encephalitis claims.
Congenital toxoplasmosis passes from mother to fetus, so the transmission chain of the most clinically important form of the disease runs person to person rather than directly involving an animal reservoir at that step. Toxoplasma encephalitis is seen most often in immunocompromised patients such as those with AIDS, from reactivation of a dormant infection, but rare cases have also been reported in immunocompetent people, so this statement is accepted as true.

Step 5: Final conclusion.
The statement that fails is the one about symptoms, since most adult toxoplasma infections are actually silent, not symptomatic.

Adult infections are mainly symptomatic (this is false)

Quick Tip: Most adult toxoplasma infections are silent; only a small fraction cause noticeable symptoms.

36. What is NOT true regarding an adult hemophiliac who visits a dentist?

(A) Cryoprecipitate may be needed

(B) The dose of lidocaine required is increased

(C) HIV screening is required

(D) Monitored GA care needs to be given

Correct Answer: (B) The dose of lidocaine required is increased

Solution:

This question asks which statement is NOT true for an adult hemophiliac (a person with a genetic clotting factor deficiency, usually factor VIII or IX) who needs dental treatment. Dental work, even a simple extraction, can cause bleeding that a hemophiliac cannot stop on his own, so each statement needs to be checked against how these patients are actually managed.

1. **Cryoprecipitate may be needed**: This is true. Cryoprecipitate is rich in factor VIII, and it (or factor VIII concentrate/desmopressin) is given before extractions or block injections to raise clotting factor levels and cut the bleeding risk.
2. **The dose of lidocaine required is increased**: This is NOT true. Hemophiliacs do not need a higher dose of local anesthetic. The real concern is the injection technique, not the drug dose: a block injection (like an inferior alveolar nerve block) can tear a vessel in a deep tissue plane and cause a hematoma that is hard to control, so infiltration anesthesia is preferred over block anesthesia when possible.
3. **HIV screening is required**: This is true. Many older hemophiliacs received repeated transfusions of blood products before modern viral screening existed, so they carry a higher risk of blood-borne infections such as HIV and hepatitis. Screening and standard precautions matter before any invasive dental work.
4. **Monitored GA care needs to be given**: This is true. If general anesthesia is unavoidable, it should be given with careful monitoring and factor replacement cover, since intubation itself can cause bleeding in the airway.

The statement that fails is the one about lidocaine dose, since local anesthetic dosing is not altered in hemophilia; only the injection technique and factor cover are.

Let's summarize:

- Factor replacement (cryoprecipitate or factor concentrate) covers the bleeding risk of dental procedures.
- Infiltration is safer than block anesthesia in hemophiliacs; the anesthetic dose itself is not increased.

So the correct answer is that the dose of lidocaine required is increased, since this is the false statement among the four.

Quick Tip: Think about what changes for a hemophiliac at the dentist: factor cover, screening, GA precautions, and injection technique, not the local anesthetic dose itself.

37. What is NOT true about fibrolamellar cancer of the liver?

(A) Not more common in males

(B) Better prognosis

(C) AFP is raised over 1000

(D) Seen in a younger age group

Correct Answer: (C) AFP is raised over 1000

Solution:

Step 1: Understanding the Question:
We need the FALSE statement about fibrolamellar hepatocellular carcinoma (fibrolamellar HCC), a rare variant of liver cancer.

Step 2: Key Formula or Approach:
Compare fibrolamellar HCC with the usual (classic) HCC on four points: sex distribution, prognosis, AFP level, and age group, since these are the features that separate the two tumors.

Step 3: Detailed Explanation:
Unlike classic HCC, which is much more common in males, fibrolamellar HCC affects males and females about equally, so "not more in males" is a true statement.
Fibrolamellar HCC also behaves less aggressively than classic HCC and is more often resectable at diagnosis, giving it a better prognosis, so that statement is true too.
A key teaching point is that fibrolamellar HCC usually arises in a liver that is NOT cirrhotic, and its serum alpha-fetoprotein (AFP) is typically normal or only mildly raised, unlike classic HCC where AFP is often markedly high. So the claim that "AFP is raised over 1000" is false for fibrolamellar HCC.
Classic HCC is a disease of older patients with cirrhosis, while fibrolamellar HCC classically occurs in children and young adults with no underlying liver disease, so "seen in a younger age group" is true.

Step 4: Final Answer:
The false statement is that AFP is raised over 1000, since fibrolamellar HCC classically has a normal or only slightly raised AFP.

Quick Tip: Fibrolamellar HCC is the young, non-cirrhotic liver cancer with a near-normal AFP, unlike classic HCC.

38. Adult polycystic kidney disease (PCKD) is associated with which of these?

(A) Fusiform abdominal aortic aneurysm

(B) Berry aneurysm

(C) Saccular aneurysm of ascending aorta

(D) Aneurysm of the arch of aorta

Correct Answer: (B) Berry aneurysm

Solution:

Autosomal dominant polycystic kidney disease (ADPKD, the adult form of PCKD) is not just a kidney condition, it comes with several extrarenal associations, and one of the best known is a specific type of blood vessel abnormality in the brain.

1. **Fusiform abdominal aortic aneurysm**: Not the classic association. Aortic aneurysms are linked more to atherosclerosis and connective tissue disorders, not specifically to ADPKD.
2. **Berry aneurysm**: This is the well known association. ADPKD patients have a higher rate of saccular (berry) aneurysms in the circle of Willis at the base of the brain, because the vessel wall defect that weakens the kidney tubules also weakens cerebral artery walls. Rupture of one of these causes a subarachnoid hemorrhage, which is why ADPKD patients with a family history of such bleeds are screened with MR angiography.
3. **Saccular aneurysm of ascending aorta**: Not a recognized feature of ADPKD.
4. **Aneurysm of the arch of aorta**: Also not a recognized feature of ADPKD.

Cysts in ADPKD can also form in the liver, pancreas and spleen, and patients can have mitral valve prolapse and colonic diverticula, but the single best known vascular association tested here is the berry aneurysm.

Let's summarize:

- ADPKD weakens vessel walls generally, and this shows up most clearly as berry aneurysms in the brain.
- Aortic aneurysms (fusiform or saccular, ascending or arch) are not the classic ADPKD association.

So the correct answer is berry aneurysm.

Quick Tip: ADPKD's weak vessel walls show up most clearly as circle of Willis (berry) aneurysms, not aortic aneurysms.

39. An 18-year-old boy presents with massive hematemesis, and the spleen is evidently enlarged up to the umbilicus. He is then found to have varices on esophagoscopy. What could be the diagnosis?

(A) Budd-Chiari syndrome

(B) Veno-occlusive disease

(C) Non-cirrhotic portal fibrosis

(D) Cirrhosis of the liver

Correct Answer: (C) Non-cirrhotic portal fibrosis

Solution:

Step 1: Understanding the Question:

An 18-year-old boy has massive hematemesis (vomiting blood), a

spleen enlarged all the way to the umbilicus, and varices on esophagoscopy. We need the diagnosis that best fits this picture.

Step 2: Key Formula or Approach:
Massive splenomegaly plus esophageal varices in bleeding points to portal hypertension. The job here is to pick which cause of portal hypertension best fits a young patient with a huge spleen but no other clue pointing to liver failure.

Step 3: Detailed Explanation:
Budd-Chiari syndrome is caused by hepatic vein outflow obstruction and usually presents with painful hepatomegaly, ascites, and abdominal pain, not with a spleen this massive as the leading feature.
Veno-occlusive disease affects the small hepatic veins, often after bone marrow transplant or exposure to certain toxins, and again presents mainly with tender hepatomegaly and ascites rather than a dramatic splenomegaly.
Cirrhosis of the liver in an 18 year old would usually come with signs of chronic liver cell failure, such as jaundice, ascites, and a shrunken or nodular liver, none of which is mentioned here.
Non-cirrhotic portal fibrosis (NCPF) is a condition seen mostly in young people from the Indian subcontinent, where portal hypertension develops from fibrosis of the small portal vein branches while the liver cells themselves stay healthy. Patients present exactly like this: a massive, well tolerated splenomegaly, repeated bouts of hematemesis from esophageal varices, and normal liver function, because the liver parenchyma is spared.

Step 4: Final Answer:
The clinical picture of a young patient with a huge spleen, variceal

bleeding, and preserved liver function fits non-cirrhotic portal fibrosis best.

Quick Tip: Massive, well tolerated splenomegaly with normal liver function in a young patient points to non-cirrhotic portal fibrosis.

40. What is NOT true with regard to the adult polycystic kidney?

(A) Hematuria

(B) Hypertension is rare

(C) Autosomal dominant transmission

(D) Cysts are seen in the liver, spleen and pancreas

Correct Answer: (B) Hypertension is rare

Solution:

Adult (autosomal dominant) polycystic kidney disease has a well defined set of features, and this question wants the one statement that does not match them.

1. **Hematuria**: True. Cysts can bleed into the urinary tract or rupture, so blood in the urine is a common finding.
2. **Hypertension is rare**: False. Hypertension is actually very common in ADPKD, often appearing even before the kidneys start to fail, because the enlarging cysts activate the renin-angiotensin system.
3. **Autosomal dominant transmission**: True. This is the defining inheritance pattern, caused mainly by mutations in the PKD1 or PKD2 genes.

4. **Cysts are seen in the liver, spleen and pancreas**: True. ADPKD is a systemic disease of cyst formation, and cysts are commonly found in the liver, and less often in the spleen and pancreas, along with the kidneys.

Since hypertension is actually one of the most common and earliest features of ADPKD rather than a rare one, that statement is the false one.

Let's summarize:

- ADPKD commonly causes hematuria, hypertension, and cysts in other organs besides the kidney.
- Calling hypertension "rare" in ADPKD is incorrect; it is one of the earliest and most common findings.

So the correct answer is that hypertension is rare.

Quick Tip: Hypertension is one of the earliest and most common findings in ADPKD, not a rare one.

41. An 18-year-old presents with massive hematemesis and a history of fever for the past 14 days that was treated with drugs. A moderately enlarged spleen is present. What is the diagnosis?

(A) NSAID-induced duodenal ulcer

(B) Drug-induced gastritis

(C) Esophageal varices

(D) Portal hypertension

Correct Answer: (C) Esophageal varices

Solution:

Step 1: Understanding the Question:

An 18-year-old has massive hematemesis, a two-week history of fever treated with drugs, and a moderately enlarged spleen. We need to pick the diagnosis that explains the bleeding.

Step 2: Key Formula or Approach:

Splenomegaly plus hematemesis in a young patient should raise suspicion of a portal hypertensive source of bleeding rather than a simple mucosal ulcer, so weigh each option against that pattern.

Step 3: Detailed Explanation:

An NSAID-induced duodenal ulcer does not explain the splenomegaly, and there is no history of NSAID use given in the vignette, so this option does not fit the whole picture.

Drug-induced gastritis also fails to explain the splenomegaly, and gastritis rarely causes hematemesis this massive on its own.

Portal hypertension is the underlying state that raises pressure in the portal venous system, but it is not itself the visible bleeding lesion, it is the mechanism behind one.

Esophageal varices are the dilated submucosal veins at the lower esophagus that form because of portal hypertension, and they are the actual site that ruptures and bleeds massively. Given the splenomegaly (from portal hypertension, here likely following the febrile illness) and the massive hematemesis, the direct diagnosis for the bleeding source is esophageal varices.

Step 4: Final Answer:

The diagnosis is esophageal varices, which are the ruptured vessels

actually responsible for the massive hematemesis, with portal hypertension and splenomegaly as the setting behind them.

Quick Tip: Splenomegaly plus massive hematemesis in a young patient points to bleeding esophageal varices, the visible lesion, not just the underlying portal hypertension.

42. A 25-year-old male with no previous cardiac complaints presents with arrhythmia. He gives a recent history of binge drinking. What is the pathology likely in this patient?

(A) Atrial fibrillation

(B) Supraventricular tachycardia

(C) Ventricular ectopics

(D) Atrial flutter

Correct Answer: (A) Atrial fibrillation

Solution:

A young man with no earlier heart problems develops an arrhythmia right after a bout of heavy drinking. This clinical picture has a well known name, and the question wants the specific rhythm disturbance behind it.

1. **Atrial fibrillation**: This is the classic answer. Acute alcohol binges can trigger new-onset atrial fibrillation in people with an otherwise normal heart, a pattern well known as "holiday heart syndrome" because it is often seen after weekend or holiday binge drinking.

2. **Supraventricular tachycardia**: This can be triggered by many things, including caffeine or stress, but it is not the specific rhythm linked by name to binge drinking.
3. **Ventricular ectopics**: These are isolated extra beats from the ventricle and can occur with alcohol use, but they are not the classic, most likely finding in this setting.
4. **Atrial flutter**: This can occur with alcohol excess as well, but atrial fibrillation is the far more common and classic rhythm seen in this scenario.

The pairing of a healthy young heart, a recent binge drinking episode, and a new arrhythmia is the textbook description of holiday heart syndrome, which is caused by atrial fibrillation.

Let's summarize:

- Holiday heart syndrome is new-onset atrial fibrillation triggered by an acute alcohol binge in a person without prior heart disease.
- It usually resolves once the alcohol is cleared and no ongoing cardiac disease is found.

So the correct answer is atrial fibrillation.

Quick Tip: New arrhythmia after a binge drinking episode in a healthy young heart is classic "holiday heart syndrome."

43. Which condition may cause hypothalamic hypogonadism?

(A) Frohlich syndrome

(B) Foster Kennedy syndrome

(C) Kallmann syndrome

(D) Fragile X syndrome

Correct Answer: (C) Kallmann syndrome

Solution:

Step 1: Recall what hypothalamic hypogonadism means.
Hypothalamic hypogonadism (also called hypogonadotropic hypogonadism) happens when the hypothalamus fails to release enough gonadotropin releasing hormone (GnRH). Without GnRH, the pituitary does not make enough LH and FSH, so the gonads never get stimulated.

Step 2: Match each option to its actual defect.
Frohlich syndrome (adiposogenital dystrophy) does cause obesity with hypogonadism, but it comes from a general hypothalamic tumor or lesion, not a specific GnRH neuron defect.
Foster Kennedy syndrome is optic atrophy in one eye with papilledema in the other, caused by a frontal lobe tumor pressing on the olfactory groove. It has nothing to do with the reproductive axis.
Fragile X syndrome is an X linked repeat disorder that causes intellectual disability and large testes in males; it is not a cause of hypogonadism.

Step 3: Focus on Kallmann syndrome.
Kallmann syndrome happens when the neurons that should make GnRH fail to migrate from the olfactory placode into the hypothalamus during fetal development. This gives isolated

hypogonadotropic hypogonadism, classically together with anosmia or a poor sense of smell, since the olfactory bulbs also fail to form properly.
This is a true hypothalamic cause of hypogonadism, unlike the other three options.

Final Answer:
Kallmann syndrome is the condition that causes hypothalamic hypogonadism.

Kallmann syndrome

Quick Tip: Think of the syndrome with anosmia and failed GnRH neuron migration.

44. What is the next line of management for a 25 year old woman who develops acute pulmonary embolism?

(A) Thrombolysis
(B) LMW heparin
(C) Oral anticoagulants
(D) IVC filter

Correct Answer: (A) Thrombolysis

Solution:

Step 1: Note what the question is really asking.
The phrase "next line of management" points to a patient who already has a confirmed diagnosis of acute pulmonary embolism (PE) and

needs urgent, life saving treatment right now, not a routine follow up step.

Step 2: Recall how PE treatment is chosen.
Treatment depends on how sick the patient is. A stable patient, with normal blood pressure and no major right heart strain, is treated with anticoagulation, usually low molecular weight heparin (LMWH), followed later by oral anticoagulants. An unstable patient, with hypotension, shock, or severe right ventricular strain, needs the clot broken down fast, since the embolism itself is the immediate threat to life.

Step 3: Apply this to a severe, acute PE.
When a question frames PE as needing urgent "next" management, it is pointing at this high risk, massive PE picture, where the clot is large enough to obstruct blood flow and threaten the patient's life. In that setting, dissolving the clot takes priority over simply preventing new clot.
Thrombolysis, using drugs such as streptokinase, urokinase, or alteplase, dissolves the clot directly and restores blood flow through the pulmonary arteries quickly, something heparin cannot do since heparin only stops new clot from forming.

Step 4: Rule out the other options.
LMW heparin prevents the clot from growing further, and is right for a stable patient, but it will not rescue someone already in shock from a large pulmonary artery blockage.
Oral anticoagulants such as warfarin take several days to reach a useful blood level, so they are never used as urgent first line therapy.
An IVC filter only stops new clots travelling up from the leg veins, it does nothing to a clot that is already lodged in the pulmonary artery.

Final Answer:
For an acute, life threatening pulmonary embolism, thrombolysis is the immediate next step in management.

Thrombolysis

Quick Tip: A life threatening PE needs clot dissolving therapy, not just clot prevention.

45. A 30 year old male patient presented with history of dizziness, vertigo, diplopia, dysphagia, weakness on the right side of the body, along with Horner's syndrome on the same side. Loss of pain and temperature sensations on the left side was noted. The patient also has loss of memory. Which artery is most likely involved in the condition described above?

(A) Anterior inferior cerebellar

(B) Posterior inferior cerebellar

(C) Middle cerebral

(D) Superior cerebellar

Correct Answer: (B) Posterior inferior cerebellar

Solution:

Step 1: List the clinical features one by one.
Dizziness and vertigo point to the vestibular nuclei or their cerebellar connections.
Dysphagia points to the nucleus ambiguus, which supplies the pharynx and larynx.

Horner's syndrome (ptosis, miosis, anhidrosis) on the same side as the lesion points to interruption of the descending sympathetic fibers. Loss of pain and temperature sensation on the opposite side of the body points to the spinothalamic tract, which has already crossed to the other side lower down in the spinal cord.

Step 2: Put the pattern together.
Ipsilateral cranial nerve and cerebellar signs together with contralateral pain and temperature loss in the body is the hallmark of a brainstem stroke affecting one side of the medulla, while the more medially placed corticospinal tract is largely spared. This combination is called lateral medullary syndrome, also known as Wallenberg syndrome.

Step 3: Identify the artery responsible.
The lateral medulla is supplied by the posterior inferior cerebellar artery (PICA), a branch of the vertebral artery. Occlusion of PICA is the classic cause of Wallenberg syndrome, producing exactly this mix, ipsilateral Horner's syndrome, ipsilateral vertigo and ataxia, dysphagia from nucleus ambiguus damage, and contralateral pain and temperature loss.

Step 4: Rule out the other arteries.
The anterior inferior cerebellar artery (AICA) supplies the lateral pons and causes a similar but higher syndrome, usually adding ipsilateral facial weakness and hearing loss, which are not the core description here.
The middle cerebral artery supplies the cerebral cortex and causes contralateral hemiparesis with cortical signs such as aphasia, not this brainstem pattern.
The superior cerebellar artery mainly causes ataxia and a different,

less complete pattern, without the classic Horner's plus dysphagia combination.

Final Answer:
The clinical picture fits Wallenberg syndrome from a lateral medullary infarct, caused by the posterior inferior cerebellar artery.

Posterior inferior cerebellar artery

Quick Tip: Ipsilateral Horner's plus contralateral pain and temperature loss points to lateral medullary syndrome.

46. In which of the following conditions is plasmapheresis useful?

(A) Wegener's granulomatosis

(B) HSP (Henoch-Schonlein purpura)

(C) Goodpasture syndrome

(D) Guillain-Barre syndrome

Correct Answer: (C) Goodpasture syndrome

Solution:

Step 1: Recall what plasmapheresis does.
Plasmapheresis (plasma exchange) removes the liquid part of blood, along with whatever harmful antibodies or immune complexes are floating in it, then returns the blood cells with replacement fluid. It works best in diseases where a circulating antibody is directly causing the damage.

Step 2: Look at Goodpasture syndrome closely.

Goodpasture syndrome is caused by antibodies against type IV collagen in the basement membrane of the kidney glomeruli and the lung alveoli, called anti glomerular basement membrane (anti-GBM) antibodies. Since the disease is driven directly by a circulating antibody, physically removing that antibody with plasmapheresis is a core part of treatment, given together with steroids and immunosuppressants.

Step 3: Compare the other options.

Wegener's granulomatosis (granulomatosis with polyangiitis) is driven by ANCA antibodies plus vessel inflammation, and is mainly treated with steroids and cyclophosphamide or rituximab; plasmapheresis is kept for severe cases with lung hemorrhage or advanced kidney failure, not as the standard first answer.

Henoch-Schonlein purpura (HSP) is an IgA mediated vasculitis in children that is usually self limiting and managed with supportive care and sometimes steroids; plasmapheresis is not a standard treatment for it.

Guillain-Barre syndrome does also respond to plasmapheresis, since it removes antibodies against peripheral nerve myelin, but among these four choices, the disease where plasma exchange is most directly tied to the disease mechanism is Goodpasture syndrome, since the antibody itself is the entire cause of the tissue damage.

Final Answer:

Plasmapheresis is useful in Goodpasture syndrome, where it directly removes the disease causing anti-GBM antibody.

Goodpasture syndrome

Quick Tip: Think of the disease where a circulating antibody against basement membrane is the direct cause.

47. Which of these does NOT feature eye manifestations in association with a seronegative arthropathy?

(A) Psoriasis

(B) Rheumatoid arthritis

(C) Reiter's syndrome

(D) Ankylosing spondylitis

Correct Answer: (B) Rheumatoid arthritis

Solution:

Step 1: Recall what a seronegative arthropathy is.
Seronegative arthropathies (seronegative spondyloarthropathies) are a group of joint diseases that share a negative rheumatoid factor, a strong link to the HLA-B27 gene, involvement of the spine and sacroiliac joints, and frequent eye inflammation. The classic members are ankylosing spondylitis, psoriatic arthritis, reactive arthritis (Reiter's syndrome), and arthritis linked to inflammatory bowel disease.

Step 2: Check the eye involvement in each true seronegative arthropathy.
Ankylosing spondylitis commonly causes anterior uveitis (iritis), presenting as a painful red eye.
Psoriatic arthritis, part of the same family, can also cause uveitis and conjunctivitis.

Reiter's syndrome (reactive arthritis) is classically remembered by the triad of arthritis, urethritis, and conjunctivitis or uveitis, so eye involvement is one of its defining features.

Step 3: Examine rheumatoid arthritis.
Rheumatoid arthritis is a seropositive disease in most patients, meaning rheumatoid factor is usually present, and it is not part of the seronegative spondyloarthropathy family at all. It does have its own eye complications, such as scleritis, episcleritis, and dry eyes from secondary Sjogren syndrome, but these come from its own separate immune process, not from being a seronegative arthropathy.

Step 4: Conclude.
Since rheumatoid arthritis does not belong to the seronegative arthropathy group in the first place, it is the one that does not fit the eye disease pattern of that group.

Final Answer:
Rheumatoid arthritis is the condition that does NOT feature eye manifestations as part of a seronegative arthropathy.

Rheumatoid arthritis

Quick Tip: Rheumatoid arthritis is seropositive, not part of the seronegative arthropathy family.

48. Why is the sickle cell carrier state usually asymptomatic?

(A) The HbS carrier state has higher oxygen affinity

(B) Less than 50% saturation does not cause sickling

(C) There is allosteric binding of HbA to the HbS carrier state

(D) There is ample HbF to make up for the carrier state

Correct Answer: (B) Less than 50% saturation does not cause sickling

Solution:

Step 1: Recall the biology of sickle cell trait.
Sickle cell trait, the carrier state, happens when a person inherits one normal beta globin gene and one sickle (HbS) gene. Roughly 40 percent of their hemoglobin is HbS and the rest, around 60 percent, is normal HbA.

Step 2: Recall why HbS causes sickling.
HbS polymerizes and distorts the red cell into a sickle shape only when it is deoxygenated and present above a certain threshold concentration inside the cell. Sickling is a concentration dependent process, the higher the HbS share relative to HbA, the easier it is for HbS molecules to link into rigid polymers.

Step 3: Apply this to the carrier state.
In sickle cell trait, HbS makes up less than half the total hemoglobin, generally under 50 percent. Below this level, the HbA present dilutes the HbS enough that the red cells almost never reach the concentration needed for polymer formation and sickling under normal physiological oxygen levels. This is why carriers are usually asymptomatic, their HbS proportion simply stays under the threshold that triggers sickling.

Step 4: Rule out the other options.

HbS itself does not have a higher oxygen affinity than HbA, so that is not the reason.

There is no special allosteric binding of HbA to HbS, the two simply coexist inside the same red cell without any direct chemical binding between them.

HbF (fetal hemoglobin) is present at only trace levels in adults and is not what protects the carrier state, since the trait itself is defined by the HbA and HbS proportions, not by HbF.

Final Answer:

The carrier state stays symptom free because the HbS fraction remains below the roughly 50 percent threshold needed to trigger sickling.

HbS below 50% does not cause sickling

Quick Tip: Sickling needs a high enough proportion of HbS; carriers stay under that threshold.

49. An HIV patient is on treatment with didanosine, stavudine, and indinavir. He is diagnosed to have pulmonary tuberculosis. Which one of the following ATT drugs is to be avoided?

(A) INH

(B) Rifampin

(C) PZA

(D) Ethambutol

Correct Answer: (B) Rifampin

Solution:

Step 1: Note the patient's current ART regimen.
The patient is already on didanosine, stavudine, and indinavir.
Indinavir belongs to the protease inhibitor class of antiretroviral drugs.

Step 2: Recall how protease inhibitors are broken down.
Protease inhibitors such as indinavir are metabolized in the liver mainly through the cytochrome P450 3A4 (CYP3A4) enzyme system.
Anything that strongly induces this enzyme speeds up the breakdown of indinavir and lowers its blood level.

Step 3: Look at the standard anti-tubercular therapy (ATT) drugs.
INH (isoniazid) is a mild enzyme inhibitor and is generally safe to combine with protease inhibitors.
Rifampin is a very strong inducer of CYP3A4 and related liver enzymes. Giving rifampin along with indinavir causes a large drop in indinavir blood levels, enough to make the antiretroviral drug ineffective and to risk treatment failure and drug resistance.
Pyrazinamide (PZA) does not have this strong CYP3A4 induction effect and is usually continued.
Ethambutol is also not a significant enzyme inducer and can be safely continued.

Step 4: Decide the practical solution.
Because rifampin lowers protease inhibitor levels so strongly, standard practice is to either replace rifampin with rifabutin, which has much weaker enzyme induction, or adjust the antiretroviral

regimen if rifampin absolutely must be used. In a patient already established on indinavir, avoiding rifampin is the safer path.

Final Answer:
Rifampin should be avoided in this patient because it drastically lowers indinavir levels through strong enzyme induction.

Rifampin

Quick Tip: Rifampin strongly induces liver enzymes that break down protease inhibitors.

50. A lady with congenital heart disease underwent a dental extraction and developed endocarditis. Which is the organism most likely to have been involved in the pathogenesis?

(A) Staphylococcus aureus

(B) Streptococcus pneumoniae

(C) Streptococcus sanguis

(D) Staphylococcus epidermidis

Correct Answer: (C) Streptococcus sanguis

Solution:

Step 1: Read the clinical picture.
The woman already has congenital heart disease, so her heart valves are not normal. A dental extraction was done, and soon after she developed endocarditis (infection of the heart valve lining).

Step 2: Link dental work to bacteremia.
Dental extraction damages the gums and lets mouth bacteria enter the blood for a short time. This is called transient bacteremia. In a normal heart this bacteremia clears on its own, but in a heart with an existing defect the abnormal valve surface lets bacteria stick and grow.

Step 3: Identify the usual organism from the mouth.
The main bacteria living in the mouth and gums are the viridans group streptococci, which includes Streptococcus sanguis, Streptococcus mitis and Streptococcus mutans. These organisms are low virulence but stick well to damaged or abnormal valve tissue, which is exactly why they are the classic cause of subacute bacterial endocarditis after a dental procedure.

Step 4: Rule out the other organisms.
Staphylococcus aureus causes a fast, aggressive (acute) endocarditis, most often seen in intravenous drug users or after skin infections, not after dental work.
Streptococcus pneumoniae is a respiratory organism that causes pneumonia and meningitis, and is a rare cause of endocarditis.
Staphylococcus epidermidis lives on skin and is mainly linked to infection of prosthetic (artificial) heart valves and catheters, not native valve endocarditis after a dental extraction.

Final Answer:
The organism most likely involved is the viridans streptococcus from the mouth.

Streptococcus sanguis

Quick Tip: Mouth flora enter the blood during dental work; viridans streptococci are the classic culprit in endocarditis after dental extraction.

51. In Rheumatic heart disease the vegetations are seen along the line of closure of the mitral valve. These vegetations are likely to get lodged in any of the following sites EXCEPT ?

(A) Brain

(B) Lung

(C) Spleen

(D) Kidney

Correct Answer: (B) Lung

Solution:

Step 1: Locate where the vegetations form.
In rheumatic heart disease the small vegetations sit along the line of closure of the mitral valve. The mitral valve lies between the left atrium and the left ventricle, which is the left side of the heart.

Step 2: Trace where a piece breaking off would travel.
If a small piece of vegetation breaks off (an embolus), it leaves the left ventricle, enters the aorta, and then travels wherever the aorta's branches carry it. This means it enters the systemic arterial circulation, the same blood supply that feeds the brain, spleen, kidney, limbs and other organs.

Step 3: Explain why the lung is different.
The lung receives its blood supply from the right side of the heart

through the pulmonary artery, not from the left side. An embolus from a left-sided valve like the mitral valve cannot reach the lung directly, because it never enters the right heart or the pulmonary circulation. Only emboli starting on the right side of the heart (tricuspid or pulmonary valve) or arising in the systemic veins would end up in the lung.

Step 4: Confirm the organs that can be affected.
The brain, spleen and kidney are all supplied by branches of the aorta, so all three are classic sites for embolic infarcts from a mitral valve vegetation.

Final Answer:
Since mitral valve emboli travel through the systemic circulation and cannot reach the lung, the lung is the exception.

Lung

Quick Tip: Mitral valve is on the left side of the heart, so its emboli go into the systemic (not pulmonary) circulation.

52. Which of these is Hepatitis B infection most commonly associated with?

(A) PAN

(B) Cryoglobulinemia

(C) SLE

(D) Polymyositis

Correct Answer: (A) PAN

Solution:

Step 1: Recall what Hepatitis B virus does beyond the liver.
Hepatitis B virus (HBV) does not only damage the liver. Circulating HBV antigen-antibody immune complexes can deposit in blood vessel walls elsewhere in the body and trigger inflammation there. This is called an extrahepatic immune complex disease.

Step 2: Name the classic extrahepatic disease of HBV.
The best known extrahepatic association of Hepatitis B is polyarteritis nodosa (PAN), a vasculitis of medium-sized arteries. A sizable fraction of PAN cases carry HBsAg (Hepatitis B surface antigen) in the blood, and immune complexes containing HBsAg have been found in the inflamed vessel walls of these patients.

Step 3: Separate this from Hepatitis C's association.
Mixed cryoglobulinemia is instead the classic extrahepatic disease linked to Hepatitis C virus, not Hepatitis B. Cryoglobulins are abnormal proteins that clump together in the cold and deposit in small vessels, and this link is strongest with chronic HCV infection.

Step 4: Rule out SLE and polymyositis.
Systemic lupus erythematosus (SLE) is an autoimmune disease driven by antinuclear antibodies and has no established strong link to Hepatitis B. Polymyositis is an inflammatory muscle disease and is not a recognised HBV complication either.

Final Answer:
Hepatitis B is most commonly linked to polyarteritis nodosa.

PAN

Quick Tip: HBsAg immune complexes deposit in medium arteries, linking Hepatitis B to polyarteritis nodosa (PAN).

53. Which of these is NOT caused by enteroviruses?

(A) Herpangina

(B) Hemorrhagic fever

(C) Pleurodynia

(D) Aseptic meningitis

Correct Answer: (B) Hemorrhagic fever

Solution:

Step 1: Recall the enterovirus family.

Enteroviruses are a group of small RNA viruses that include poliovirus, coxsackievirus A and B, and echovirus. They spread by the fecal-oral route and mainly infect the gut, but they are named after the gut only because that is where they multiply, and they actually cause disease in many other organs.

Step 2: Match each option to an enterovirus disease.

Herpangina is caused by coxsackievirus A: it causes painful small blisters and ulcers at the back of the throat and soft palate, mainly in children.

Pleurodynia (also called Bornholm disease) is caused by coxsackievirus B: it causes sudden, severe chest and upper belly muscle pain due to inflammation of chest wall muscles.

Aseptic meningitis (viral meningitis with a clear, non-purulent spinal fluid) is one of the most common causes of meningitis worldwide, and enteroviruses, especially echovirus and coxsackievirus, are the leading cause.

Step 3: Check hemorrhagic fever.
Viral hemorrhagic fevers, such as dengue hemorrhagic fever, Ebola, Lassa fever and Crimean-Congo hemorrhagic fever, are caused by completely different virus families (flaviviruses, filoviruses, arenaviruses, bunyaviruses). None of these belong to the enterovirus group. Enteroviruses are not a recognised cause of a hemorrhagic fever syndrome.

Final Answer:
Herpangina, pleurodynia and aseptic meningitis are all classic enterovirus diseases, but hemorrhagic fever is not.

Hemorrhagic fever

Quick Tip: Enteroviruses cause herpangina, pleurodynia and aseptic meningitis; hemorrhagic fevers come from other virus families entirely.

54. Which of these is a calicivirus?

(A) HAV

(B) HBV

(C) HCV

(D) HEV

Correct Answer: (D) HEV

Solution:

Step 1: Sort the four hepatitis viruses by family.
Hepatitis A virus (HAV) belongs to the Picornaviridae family, in the genus Hepatovirus, and is a small non-enveloped RNA virus.
Hepatitis B virus (HBV) belongs to the Hepadnaviridae family, and is unusual because it is a DNA virus that replicates through an RNA intermediate.
Hepatitis C virus (HCV) belongs to the Flaviviridae family, the same family as dengue and yellow fever virus.

Step 2: Place Hepatitis E virus.
Hepatitis E virus (HEV) is a small, non-enveloped, single-stranded RNA virus spread by the fecal-oral route, much like Hepatitis A, and it was traditionally taught as a member of the Caliciviridae family because of its similar size and shape on electron microscopy. This classification is why textbooks from this exam's era list HEV as the calicivirus among the hepatitis viruses.

Step 3: Note the current classification (update).
Later molecular and genomic studies showed HEV's genome organisation is different enough from true caliciviruses that it has since been moved into its own family, Hepeviridae (genus Orthohepevirus), and it is no longer officially classified as a calicivirus. This is a reclassification, not a change in which virus causes Hepatitis E; the exam-era answer among these four options remains HEV, since none of HAV, HBV or HCV was ever grouped with caliciviruses.

Final Answer:
Among the given options, Hepatitis E virus is the one historically classified as a calicivirus.

$$\boxed{\text{HEV}}$$

Quick Tip: HEV was historically grouped with Caliciviridae due to its size and shape, though it is now placed in its own family, Hepeviridae.

55. From which area should a biopsy be taken in the case of a viral esophageal ulcer?

(A) Edges

(B) Surrounding mucosa

(C) Base

(D) Indurated area

Correct Answer: (A) Edges

Solution:

Step 1: Picture what a viral esophageal ulcer looks like.
Viral infections of the esophagus, most often herpes simplex virus (HSV), produce small, shallow, punched out ulcers on the lining of the food pipe.

Step 2: Understand where the virus actually lives in the ulcer.
HSV infects the squamous epithelial cells, the living cells that line the esophagus. These infected cells are found at the edges (margins) of the ulcer, where the epithelium is still present but shows viral

damage, such as multinucleated giant cells and Cowdry type A inclusion bodies on microscopy.

Step 3: Explain why the base and other sites give a lower yield.
The base (floor) of the ulcer is usually just necrotic debris, fibrin and inflammatory cells with no live infected epithelium left, so a biopsy from there often misses the diagnostic viral changes. The surrounding mucosa and any indurated (thickened) area are typically normal or non-specifically inflamed tissue that will not show the specific viral inclusions either.

Step 4: Note the CMV exception, for completeness.
This rule is specific to HSV-type viral ulcers. For cytomegalovirus (CMV) esophagitis, the virus instead infects endothelial and stromal cells found deeper, in the ulcer base, so CMV biopsies are best taken from the base rather than the edge. When a question simply says viral esophageal ulcer without naming CMV, the classic teaching point being tested is the HSV rule: biopsy the edge.

Final Answer:
The biopsy should be taken from the edges of the ulcer.

Edges

Quick Tip: HSV infects the epithelial cells at the ulcer margin, so biopsy the edges, not the necrotic base.

56. What is the investigation for an 8 yr old boy with a lesion on the back, featuring peripheral scaling and central scarring?

(A) Tzanck test

(B) KOH mount

(C) Skin biopsy

(D) Patch test

Correct Answer: (C) Skin biopsy

Solution:

Step 1: Read the description of the lesion.
An 8 year old boy has a skin lesion on the back that is scaly at the edges and scarred in the centre, meaning the lesion has been slowly spreading outward while healing (and leaving a scar) in the middle.

Step 2: Match this pattern to a diagnosis.
A slowly growing plaque that spreads at the edge while scarring in the middle is a classic description of lupus vulgaris, which is a chronic form of cutaneous tuberculosis caused by Mycobacterium tuberculosis reaching the skin. It typically appears as reddish-brown, soft (apple jelly) nodules that slowly enlarge outward and leave central scarring behind as they heal.

Step 3: Decide the correct investigation.
Because lupus vulgaris is a tissue-level, granulomatous disease, it needs to be confirmed by skin biopsy, which shows caseating (or sometimes non-caseating) epithelioid cell granulomas with Langhans giant cells, and can be paired with acid-fast staining or tuberculosis culture and PCR of the tissue for full confirmation.

Step 4: Rule out the other tests.
Tzanck test is used to look for multinucleated giant cells in

vesicular/blistering conditions such as herpes or pemphigus, and gives no information about a granulomatous plaque like this.
KOH mount is used to look for fungal hyphae in suspected fungal skin infections such as tinea (ringworm), which can also show peripheral scaling with some central clearing, but does not show central scarring in the way lupus vulgaris does, and would miss a mycobacterial diagnosis.
Patch test is used to identify the allergen in allergic contact dermatitis and has no role in diagnosing an infectious granulomatous skin lesion.

Final Answer:
The lesion described is lupus vulgaris, and it is best confirmed with a skin biopsy.

Skin biopsy

Quick Tip: Peripheral spread with central scarring points to lupus vulgaris (cutaneous TB), confirmed on skin biopsy.

57. What is the investigation of choice for invasive amebiasis?

(A) ELISA

(B) Biopsy

(C) IHA (Indirect Haemagglutination Assay)

(D) PCR

Correct Answer: (A) ELISA

Solution:

Step 1: Understanding the Question:
Invasive amebiasis means Entamoeba histolytica has moved out of the gut lumen into the bowel wall or a distant organ, most often the liver, to form an abscess. The question asks which test best confirms this invasive stage.

Step 2: Key Concept:
Once the parasite invades tissue, trophozoites are no longer shed freely in stool, so stool microscopy is often negative in invasive disease, even when the infection is active and severe. The body does mount a strong antibody response against the invading parasite, and this response is what the best test should pick up.

Step 3: Detailed Explanation:
ELISA for antiamebic antibodies is over 90 percent sensitive in invasive amebiasis, including amebic liver abscess, and becomes positive within a week of invasion, which makes it the current test of choice.
IHA also detects antibodies but is less sensitive than ELISA and stays positive for years after a past infection has already cleared, so it cannot tell a doctor whether the disease is currently active.
Biopsy, taken at colonoscopy, can show trophozoites invading the colon wall, but it is invasive, carries a risk of perforation in inflamed tissue, and is not practical for an abscess sitting deep in the liver.
PCR on stool is good for confirming E. histolytica in the gut and telling it apart from the harmless look alike E. dispar, but it is not the standard test once the disease has already become invasive and systemic.

Step 4: Final Answer:

Serology by ELISA is the most sensitive and practical test for invasive amebiasis, so the answer is ELISA.

Quick Tip: Stool exam is often negative once the amoeba invades tissue, so serology becomes the test of choice.

58. A 30 year old patient who had a laparotomy recently developed an intraperitoneal abscess (peritonitis). The causative organism is a Gram positive coccus that is resistant to vancomycin and to bacitracin, grows well in 6.5% NaCl broth, and shows a positive optochin reaction. Which organism is the likely cause?

(A) Enterococcus faecalis

(B) Streptococcus pneumoniae

(C) Staphylococcus aureus

(D) Peptostreptococcus

Correct Answer: (A) Enterococcus faecalis

Solution:

Step 1: Understanding the Question:
The clinical picture is a post laparotomy intraperitoneal abscess, and the lab clues describe a Gram positive coccus. The task is to match those biochemical clues to one organism among the four options.

Step 2: Key Concept:
Three simple bedside tests separate the common Gram positive cocci from each other: the bacitracin disc test, growth in 6.5% NaCl broth, and vancomycin susceptibility. Group A strep (Streptococcus

pyogenes) is bacitracin sensitive, while enterococci and most other streptococci are bacitracin resistant. Only enterococci reliably grow in 6.5% NaCl broth, a feature other streptococci and staphylococci do not share in the same way.

Step 3: Detailed Explanation:
Growth in 6.5% NaCl broth points straight to Enterococcus, since this high salt tolerance is a classic identifying feature of the genus and is not shared by Streptococcus pneumoniae or Peptostreptococcus.
Resistance to bacitracin fits Enterococcus as well, since bacitracin resistance is expected for this genus, unlike Streptococcus pyogenes which is bacitracin sensitive.
Resistance to vancomycin is a well known and clinically important trait of some Enterococcus strains, called VRE (vancomycin resistant enterococcus), and it is a recognised cause of hospital acquired peritonitis after abdominal surgery.
Streptococcus pneumoniae is ruled out because it does not tolerate 6.5% NaCl broth and is a common cause of pneumonia and meningitis, not typically a post laparotomy abdominal abscess.
Staphylococcus aureus is a catalase positive coccus, and while it can cause abscesses, it does not match the salt tolerant, bacitracin resistant profile described.
Peptostreptococcus is an anaerobic coccus and would not grow in the aerobic conditions implied by the biochemical work up, and it is not associated with vancomycin resistance in this way.

Step 4: Final Answer:
The vancomycin resistant, bacitracin resistant, salt tolerant profile fits Enterococcus faecalis, so that is the causative organism.

Quick Tip: Growth in 6.5% NaCl broth plus bacitracin resistance is the classic identification pattern for Enterococcus.

59. A patient has a brain abscess. The aspirated material is foul smelling and shows red fluorescence under ultraviolet light. Which organism could be implicated?

(A) Staphylococcus aureus

(B) Peptostreptococcus

(C) Bacteroides

(D) Acanthamoeba

Correct Answer: (C) Bacteroides

Solution:

Step 1: Understanding the Question:
The vignette gives two strong clues: a foul smelling abscess, which points to anaerobic infection, and red fluorescence of the pus under UV light, which is a specific laboratory sign.

Step 2: Key Concept:
Foul smelling pus is typical of mixed anaerobic infection, since anaerobes produce short chain fatty acids and volatile compounds that give the pus its bad odour. Among anaerobes, pigmented species of Bacteroides and Prevotella (older name Bacteroides melaninogenicus) produce a black pigment that glows brick red under ultraviolet light, which is used as a quick identifying test in the lab.

Step 3: Detailed Explanation:

Bacteroides fits both clues directly: it is an obligate anaerobe, so its infections are classically foul smelling, and the pigmented group shows the characteristic red fluorescence under UV light. Brain abscess is also a recognised site for anaerobic infection, often from spread of dental or sinus infection.
Staphylococcus aureus is a facultative anaerobe and its pus does not have a foul odour or red fluorescence, so it is inconsistent with both clues.
Peptostreptococcus is also anaerobic and can cause foul smelling pus, but it does not produce the pigment responsible for the red UV fluorescence, so it does not match the second clue.
Acanthamoeba is a free living amoeba that causes granulomatous encephalitis or keratitis, not a foul smelling pyogenic abscess, and it has no UV fluorescence property.

Step 4: Final Answer:
The combination of foul odour and red fluorescence under UV light points to pigmented Bacteroides.

Quick Tip: Red fluorescence under UV light is a classic clue for pigmented Bacteroides species.

60. Which of the following does NOT feature preformed toxins?

(A) Staphylococcus aureus

(B) ETEC (Enterotoxigenic E. coli)

(C) Bacillus cereus

(D) Clostridium botulinum

Correct Answer: (B) ETEC (Enterotoxigenic E. coli)

Solution:

Step 1: Understanding the Question:

Food poisoning organisms are grouped by whether the toxin is already made in the food before it is eaten (preformed toxin) or whether the organism grows inside the gut after ingestion and makes toxin there. The question asks which organism does NOT belong to the preformed toxin group.

Step 2: Key Concept:

Preformed toxin food poisoning has a short incubation period, usually 1 to 6 hours, because no bacterial growth is needed inside the body, the toxin acts as soon as it is absorbed. Toxin mediated infections, where the organism first colonises the gut and then produces toxin, take longer to cause symptoms, usually many hours to a few days.

Step 3: Detailed Explanation:

Staphylococcus aureus makes an enterotoxin in improperly stored food, such as cream pastries or salads, before it is eaten, so illness starts within 1 to 6 hours. This is preformed toxin.

Bacillus cereus has an emetic type caused by a preformed toxin in reheated rice, with onset in 1 to 6 hours, similar to Staph aureus.

Clostridium botulinum grows in improperly canned or anaerobically packed food and releases botulinum toxin into the food itself before ingestion, so the toxin is also preformed, though the onset of paralysis symptoms is delayed for other reasons related to nerve binding.

ETEC is different: the bacteria are swallowed alive, then colonise the small intestine and produce heat labile and heat stable enterotoxins inside the gut. The toxin is made in the body, not in the food, so ETEC toxin is not preformed.

Step 4: Final Answer:
Since ETEC produces its toxin only after colonising the gut, it is the one that does not feature a preformed toxin.

Quick Tip: Preformed toxins act fast, 1 to 6 hours, because the toxin is already in the food. ETEC needs to colonise the gut first.

61. Which of the following is a marker for recent (acute) hepatitis B infection?

(A) IgM anti-HBs

(B) IgM anti-HBc

(C) HBeAg

(D) HBsAg

Correct Answer: (B) IgM anti-HBc

Solution:

Step 1: Understanding the Question:
Hepatitis B has several serological markers that appear at different times during infection, and the question asks which one specifically tells us the infection is recent, meaning acute.

Step 2: Key Concept:
HBsAg and HBeAg can be present in both acute and chronic infection, so on their own they cannot separate a brand new infection from a long standing one. Anti-HBc, the antibody against the core antigen, appears early and its IgM form is short lived, which makes IgM anti-HBc a marker of a recent infection, not an old or chronic one.

Step 3: Detailed Explanation:
IgM anti-HBc rises within weeks of infection and typically stays positive for about six months, exactly the window we call acute hepatitis B. It is also the only positive marker during the so called window period, when HBsAg has already cleared and anti-HBs has not yet appeared, so it is the single most reliable marker of recent infection.
HBsAg shows that the virus is present, but it stays positive in chronic carriers for years, so a positive HBsAg alone does not tell us whether the infection is new.
HBeAg reflects active viral replication and high infectivity, and it can be positive in both acute and chronic active hepatitis B, so it does not specifically mark recency either.
IgM anti-HBs is not the marker used clinically, since anti-HBs (mostly IgG) appears late, after recovery, and signals immunity or past vaccination rather than a recent infection.

Step 4: Final Answer:
IgM anti-HBc is the marker that specifically identifies a recent, acute hepatitis B infection.

Quick Tip: IgM anti-HBc is positive early and is the only marker seen during the window period of acute hepatitis B.

62. What may be seen in a case of acute hepatitis B infection?

(A) Dane particle

(B) HBsAg

(C) IgM anti-HBc

(D) Anti-HBe

Correct Answer: (C) IgM anti-HBc

Solution:

Step 1: Understanding the Question:
This question again deals with hepatitis B markers, but this time it asks which finding is the one that specifically defines a case as acute, rather than simply being present at some point during infection.

Step 2: Key Concept:
Several hepatitis B markers, such as the Dane particle (the complete infectious virion), HBsAg, and anti-HBe, can appear at multiple stages of infection, acute or chronic, and so cannot by themselves diagnose an acute case. IgM anti-HBc, however, is only found in the first several months after infection, which is what makes it diagnostic of the acute phase specifically.

Step 3: Detailed Explanation:
The Dane particle is simply the complete, infectious hepatitis B virion, made of the outer envelope (HBsAg) and inner core (HBcAg with viral DNA and polymerase). It circulates whenever the virus is actively replicating, in both acute and chronic infection, so its presence does not confirm acuteness.
HBsAg appears in acute infection but also persists in chronic carriers for years, so on its own it cannot distinguish acute from chronic disease.

Anti-HBe usually appears later, as the disease starts to resolve or in the inactive carrier state, and its presence does not specifically define an acute infection.
IgM anti-HBc is different: it rises within weeks of exposure and disappears by about six months, so its presence is the specific laboratory sign that pins the infection down to the acute or recent window. It is also the key marker used to diagnose acute hepatitis B during the window period, when HBsAg may already be undetectable.

Step 4: Final Answer:
IgM anti-HBc is the finding that is specifically seen in, and diagnostic of, acute hepatitis B infection.

Quick Tip: Dane particle, HBsAg and anti-HBe can occur at multiple stages, but IgM anti-HBc is specific to the acute phase.

63. If a PCR reaction is working at 100% efficiency, how many copies of the target are produced after 3 cycles, relative to the initial amount?

(A) Double the initial amount

(B) Thrice the initial amount

(C) Four times the initial amount

(D) Eight times the initial amount

Correct Answer: (D) Eight times the initial amount

Solution:

Step 1: Understanding the Question:
PCR (polymerase chain reaction) copies a target DNA sequence again

and again using repeated heating and cooling cycles. At 100% efficiency, every single strand present at the start of a cycle is copied exactly once during that cycle. The question asks how many times the starting amount has multiplied after 3 such cycles.

Step 2: Key Formula or Approach:
At 100% efficiency, the amount of target DNA doubles with every cycle, because each strand present makes one new complementary copy. After n cycles, the amplification factor is

$$2^n$$

This is the standard formula used to describe exponential amplification in PCR.

Step 3: Detailed Explanation:
Start with 1 unit of target DNA before cycling begins.
After cycle 1, the amount doubles to

$$2^1 = 2$$

After cycle 2, the amount doubles again to

$$2^2 = 4$$

After cycle 3, the amount doubles a third time to

$$2^3 = 8$$

So after 3 cycles, the DNA amount is 8 times what it was at the start.

Step 4: Final Answer:
At 100% efficiency, 3 cycles give $2^3 = 8$ times the initial amount of target DNA.

Quick Tip: At 100% efficiency, PCR doubles the target every cycle, so after n cycles the amount is 2 to the power n.

64. For which of these is northern blotting with hybridization useful?

(A) Protein antigen

(B) RNA

(C) DNA

(D) Histone

Correct Answer: (B) RNA

Solution:

Step 1: Understanding the Question:
The question asks which type of molecule is picked up when northern blotting is combined with hybridization.

Step 2: Key Concept:
Blotting techniques in molecular biology are named for the molecule each one detects. Southern blotting, named after its inventor Edwin Southern, detects DNA. Northern blotting is the RNA version of the same idea, built on the same steps but applied to RNA. Western blotting detects protein, using labeled antibodies instead of a nucleic acid probe.

Step 3: Detailed Explanation:
In northern blotting, RNA is first extracted from the sample and separated by size on a gel using electrophoresis.
The separated RNA bands are then transferred, or blotted, onto a nylon or nitrocellulose membrane.
A labeled probe, either a DNA or RNA strand whose sequence is complementary to the RNA of interest, is added and allowed to hybridize, meaning it binds the matching RNA band by base pairing.
This tells the lab whether a specific messenger RNA is present in the sample, and roughly how much of it there is, which is why northern blotting is a standard tool to study gene expression.

Step 4: Final Answer:
Protein antigen is picked up by Western blotting using antibodies, since a protein has no base sequence to hybridize with. DNA is the target of Southern blotting, not northern blotting. Histone is a DNA binding protein, so like any protein it needs Western blotting or an immunological method, not hybridization. Northern blotting with hybridization is useful for detecting RNA.

Quick Tip: Remember the naming pattern: Southern blot detects DNA, northern blot detects RNA, and Western blot detects protein using antibodies.

65. Which of these may feature auto infection?

(A) Ancylostoma

(B) Enterobius

(C) Ascaris

(D) Paragonimus

Correct Answer: (B) Enterobius

Solution:

Step 1: Understanding the Question:
Auto infection means a person keeps reinfecting themselves with a parasite without the eggs or larvae having to leave the body and mature outside first. We need to pick the parasite among the options where this can happen.

Step 2: Key Concept:
Enterobius vermicularis, the pinworm, is the classic example of auto infection among the common intestinal worms. The gravid female migrates out through the anus at night to lay her eggs on the perianal skin, and this causes intense itching.

Step 3: Detailed Explanation:
When the child or adult scratches the itchy perianal area, the sticky eggs get caught under the fingernails.
If the hands then touch the mouth, or food is handled with unwashed hands, the eggs are swallowed again, completing a new cycle inside the same host, this is hand to mouth auto infection.
A second route also exists called retro infection, where eggs hatch right on the perianal skin and the newly hatched larvae crawl back up through the anus into the rectum to mature there, without ever leaving the host.

Because of this, Enterobius infection tends to persist and spread easily within a household even with treatment, unless hygiene measures are also followed.

Step 4: Why the other options are wrong:
Ancylostoma, the hookworm, has infective filariform larvae that must develop in warm moist soil for days before they can penetrate human skin, so there is no way for a freshly passed egg to reinfect the same person directly.
Ascaris eggs passed in stool need about two to three weeks of development in soil to become infective, so they cannot cause auto infection either.
Paragonimus, the lung fluke, has a complex life cycle needing a snail and then a crab or crayfish as intermediate hosts before humans can get infected by eating undercooked crab meat, so direct auto infection in the same host is not possible.

Step 4: Final Answer:
Enterobius vermicularis is the correct answer, since both hand to mouth spread and retro infection allow it to reinfect the same host repeatedly.

Quick Tip: Enterobius (pinworm) causes perianal itching, and scratching plus hand to mouth contact lets the same person reinfect themselves.

66. What medium is used for culture in a suspected case of legionnaires disease?

(A) Thayer Martin

(B) Buffered charcoal with yeast extract

(C) Chocolate agar

(D) Bordet Gengou

Correct Answer: (B) Buffered charcoal with yeast extract

Solution:

Step 1: Understanding the Question:
Legionnaires disease is caused by Legionella pneumophila, and the question asks which culture medium is used to grow this organism.

Step 2: Key Concept:
Legionella is a fastidious gram negative bacillus, meaning it will not grow on ordinary media because it has special nutrient needs, in particular it needs L cysteine and iron salts to grow.

Step 3: Detailed Explanation:
Buffered charcoal yeast extract agar, usually written as BCYE agar, is prepared with yeast extract as a nutrient source, activated charcoal to soak up toxic compounds that would otherwise stop the bacteria from growing, and it is buffered to keep the pH steady, plus it is supplemented with L cysteine and ferric salts that Legionella specifically needs.
Antibiotics can also be added to BCYE to make a selective version, which helps recover Legionella from samples like sputum or bronchoalveolar lavage that also contain other faster growing bacteria.
This is why BCYE is the standard recommended medium whenever legionnaires disease is suspected.

Step 4: Why the other options are wrong:
Thayer Martin medium is a selective chocolate agar based medium used to culture Neisseria gonorrhoeae from sites with mixed flora, and does not support Legionella growth.
Plain chocolate agar is used for fastidious organisms like Haemophilus influenzae and Neisseria species, but it lacks the cysteine and iron supplementation Legionella needs, so Legionella will not grow well on it.
Bordet Gengou medium is the classic medium for Bordetella pertussis, the whooping cough organism, and has nothing to do with Legionella.

Step 4: Final Answer:
Buffered charcoal yeast extract agar is the medium used to culture Legionella in a suspected case of legionnaires disease.

Quick Tip: Legionella needs cysteine and iron to grow, which is why buffered charcoal yeast extract (BCYE) agar is the standard culture medium for it.

67. Following a bee sting, the person develops periorbital edema, laryngospasm, breathing difficulty. These reactions are mediated through which of the following?

(A) Cytotoxic T cell

(B) IgE mediated

(C) IgA mediated

(D) Immune complex reactions

Correct Answer: (B) IgE mediated

Solution:

Step 1: Understanding the Question:

The person develops swelling around the eyes, spasm of the voice box and breathing difficulty right after a bee sting. This picture describes an anaphylactic reaction, and we are asked which immune mechanism causes it.

Step 2: Key Concept:

This is a Type I hypersensitivity reaction, which is mediated by IgE antibodies.

Step 3: Detailed Explanation:

On a first exposure to bee venom, the immune system makes IgE antibodies specific to venom proteins, and these IgE molecules attach to receptors on mast cells and basophils, this is called sensitization.
On a repeat sting, the venom antigen binds and cross links the IgE already sitting on the mast cell surface, and this cross linking triggers the mast cell to degranulate within minutes.
The released mediators, mainly histamine along with leukotrienes and prostaglandins, cause blood vessels to leak fluid into tissue, giving periorbital edema, and cause smooth muscle spasm in the airway, giving laryngospasm and breathing difficulty.
This fast, IgE driven, mast cell mediated response is exactly what is seen in anaphylaxis after an insect sting, a drug, or a food allergen.

Step 4: Why the other options are wrong:

Cytotoxic T cell reactions are Type IV, cell mediated, delayed hypersensitivity that takes one to several days to develop, such as in

contact dermatitis or graft rejection, which does not fit a reaction within minutes of a sting.
IgA is a mucosal antibody involved in local defense at gut and respiratory surfaces, it is not the antibody class that drives immediate hypersensitivity reactions.
Immune complex reactions are Type III hypersensitivity, where antigen antibody complexes deposit in tissues and activate complement over hours to days, as in serum sickness, not the immediate picture described here.

Step 4: Final Answer:
The reaction is IgE mediated, a Type I hypersensitivity response to bee venom.

Quick Tip: Immediate swelling and breathing trouble after a sting is anaphylaxis, a Type I hypersensitivity reaction driven by IgE on mast cells.

68. CLED is preferred to McConkeys medium because?

(A) It is a differential medium

(B) Prevents swarming of Proteus

(C) Supports the growth of Pseudomonas

(D) Supports growth of Candida and Staphylococcus

Correct Answer: (D) Supports growth of Candida and Staphylococcus

Solution:

Step 1: Understanding the Question:
CLED stands for cystine lactose electrolyte deficient medium, and it is

commonly used for culturing urine samples. The question asks for the specific reason it is preferred over MacConkey medium.

Step 2: Key Concept:
MacConkey medium contains bile salts and crystal violet, which are added on purpose to inhibit gram positive bacteria and fungi, since MacConkey is designed to be selective for gram negative organisms only. CLED medium does not contain these inhibitory substances.

Step 3: Detailed Explanation:
Because a urine sample can contain a mix of gram negative rods, gram positive cocci like Staphylococcus saprophyticus, and yeasts like Candida, a lab wants one plate that lets all of these grow so none are missed.
CLED is nutritionally rich with peptone, lactose, and cystine, and lacks bile salts, so gram positive organisms and Candida grow on it just as well as gram negative organisms do, letting the lab detect the true range of urinary pathogens.
MacConkey, in contrast, would suppress the growth of Staphylococcus and Candida because of its bile salts and crystal violet content, so a mixed infection could be missed if MacConkey were used alone.
CLED also happens to lack electrolytes, which as a side effect stops Proteus from swarming across the plate, but the main and most commonly quoted reason it is chosen over MacConkey specifically is its ability to support the wider range of urinary organisms, including gram positive cocci and Candida.

Step 4: Why the other options are wrong:
CLED is not really described as a differential medium in the way lactose fermenting media like MacConkey are, since CLED uses a

bromothymol blue lactose system mainly to show colony color, not primarily to separate organism groups.
Preventing Proteus swarming is a real property of CLED but it is a secondary benefit tied to the missing electrolytes, not the chief reason it is preferred over MacConkey in this comparison.
CLED does not specifically favor Pseudomonas growth over other organisms, Pseudomonas can grow on many non selective and selective media alike, so this is not the distinguishing feature being asked about.

Step 4: Final Answer:
CLED is preferred to MacConkey because it supports the growth of Candida and Staphylococcus, organisms that MacConkey's bile salts and crystal violet would otherwise inhibit.

Quick Tip: CLED lacks the bile salts and crystal violet found in MacConkey, so it does not inhibit gram positive cocci or Candida, unlike MacConkey.

69. Two farmers were brought dead, autopsy done revealed viscera that had the smell of bitter almonds. The most likely poisoning is due to that by?

(A) Organophosphorus

(B) Hydrocyanic acid

(C) Morphine

(D) Atropine

Correct Answer: (B) Hydrocyanic acid

Solution:

Step 1: Understanding the Question:

The key clue in this question is the smell of bitter almonds coming from the viscera at autopsy. We need to match this smell to a specific poison.

Step 2: Key Concept:

A smell of bitter almonds on the viscera or breath is the classic and most tested sign of cyanide poisoning, and hydrocyanic acid, also called prussic acid, is a cyanide compound.

Step 3: Detailed Explanation:

Cyanide works by binding tightly to cytochrome c oxidase inside mitochondria, which blocks the last enzyme of the electron transport chain, so cells cannot use oxygen even though it is present in the blood, this is called histotoxic or cytotoxic hypoxia.

Because of this, death from cyanide can happen very fast, within minutes to a couple of hours, which fits the scenario of farmers being found already dead.

Farmers often have access to cyanide containing pesticides and rodenticides, or to compounds like potassium cyanide used in some agricultural and industrial settings, making accidental or suicidal cyanide poisoning plausible in this context.

At autopsy, the smell of bitter almonds is a well known and specific finding for cyanide, though it should be noted not everyone can detect this smell due to a genetic difference in the ability to smell cyanide.

Step 4: Why the other options are wrong:

Organophosphorus poisoning gives a garlic like odor, along with pinpoint pupils, excessive salivation and sweating, and muscle

fasciculations, not a bitter almond smell.
Morphine poisoning causes pinpoint pupils and severe respiratory depression but has no characteristic odor on the viscera.
Atropine poisoning causes dilated pupils, dry flushed skin, and delirium, again with no bitter almond odor.

Step 4: Final Answer:
The bitter almond smell on the viscera points to hydrocyanic acid, meaning cyanide poisoning.

Quick Tip: A bitter almond smell on the viscera at autopsy is the classic sign of cyanide (hydrocyanic acid) poisoning.

70. A case of poisoning was brought to the casualty, a gastric lavage was done, and the lavage turned black when it was heated after being treated with silver nitrate. The poisoning is most likely to have been due to which of the following?

(A) Tik-20

(B) Celfos

(C) Malathion

(D) Parathion

Correct Answer: (B) Celfos

Solution:

Step 1: Understanding the Question:
The question describes a bedside test on gastric lavage fluid, silver nitrate is added and the mixture turns black on heating. We need to

identify which poison this test points to.

Step 2: Key Concept:
This blackening with silver nitrate on heating is a classic test for phosphine gas, which is released from metal phosphide compounds such as aluminium phosphide in the presence of moisture or stomach acid.

Step 3: Detailed Explanation:
Aluminium phosphide is a solid fumigant and rodenticide that, once it meets moisture or gastric hydrochloric acid, breaks down and releases phosphine gas.
Phosphine is a strong reducing agent, and when the lavage fluid containing it is treated with silver nitrate and heated, the phosphine reduces the silver ions to metallic silver, which deposits as a black precipitate, this is the basis of the silver nitrate paper or solution test used to detect phosphide poisoning.
Celfos is a well known trade name for aluminium phosphide used widely as a grain fumigant and rodenticide in India, and it is a common agent of both accidental and suicidal poisoning, which fits a case reaching the casualty as an emergency.

Step 4: Why the other options are wrong:
Tik-20 is also associated with phosphide type compounds in casual naming, but Celfos is the specific and most standard aluminium phosphide brand linked with this exact silver nitrate blackening test in toxicology teaching, so it is taken as the intended answer here.
Malathion is an organophosphate insecticide that works by inhibiting acetylcholinesterase, causing a cholinergic toxidrome with excess salivation, pinpoint pupils and muscle fasciculations, it does not release phosphine gas and will not blacken silver nitrate on heating.

Parathion is likewise an organophosphate compound with the same cholinergic mechanism as malathion, and it also does not produce phosphine or a positive silver nitrate blackening test.

Step 4: Final Answer:
The blackening of silver nitrate on heating after gastric lavage points to phosphine release, most likely due to Celfos, an aluminium phosphide compound.

Quick Tip: Silver nitrate turning black on heating with gastric lavage fluid is the classic test for phosphine from aluminium phosphide (Celfos) poisoning.

71. Hemodialysis is mandatory in which poisoning?

(A) Copper sulphate poisoning

(B) Ethanol poisoning

(C) Amphetamine poisoning

(D) Organophosphorus poisoning

Correct Answer: (B) Ethanol poisoning

Solution:

Step 1: Know when hemodialysis works for a poison.
Hemodialysis pulls a poison out of the blood across a membrane. It works best when the poison is small in size, is not bound tightly to plasma proteins, does not spread widely into body tissues, and dissolves well in water.

Step 2: Check ethanol against these rules.

Ethanol is a small molecule that barely binds to plasma proteins, stays mostly in body water, and mixes freely with water. This makes it easy to pull out with a dialysis machine. When a patient has a dangerously high blood alcohol level, is in coma, or has breathing that is shutting down, and supportive care alone is not enough, or another toxic alcohol was taken along with it, hemodialysis is used to bring the level down fast.

Step 3: Rule out copper sulphate poisoning.
Copper sulphate poisoning causes vomiting, hemolysis, and can injure the kidney and liver. Treatment relies on chelating agents such as D-penicillamine and supportive care for the hemolysis and organ damage. Dialysis is used only after the kidney has already failed, not as the main tool against the poison itself.

Step 4: Rule out amphetamine poisoning.
Amphetamine is handled with sedation, cooling for high body temperature, and control of blood pressure and heart rhythm. It binds strongly to tissue and does not stay mainly in the blood, so a dialysis machine cannot pull much of it out.

Step 5: Rule out organophosphorus poisoning.
Organophosphorus compounds are treated with atropine and pralidoxime, plus airway support. These poisons are fat soluble and stick to tissue, so little stays in the blood where a dialysis membrane could reach it.

Step 6: Final answer.
Among the choices, only ethanol has the size, water solubility, and low protein binding that make it truly dialyzable.

Ethanol poisoning

Quick Tip: Think about which poison is small and water soluble with low protein binding, since dialysis clears those best.

72. Fine leathery froth that emanates from the nostrils on chest compression is diagnostic of death due to?

(A) Drowning

(B) Hanging

(C) Morphine poisoning

(D) Strangulation

Correct Answer: (A) Drowning

Solution:

Step 1: Recall the classic sign of drowning.
In a drowning death, water mixes with air, mucus, and surfactant in the airway during the struggle to breathe. This mixture is churned into a fine, sticky froth that fills the trachea and bronchi.

Step 2: Understand why chest compression brings it out.
When the chest is pressed during autopsy, the froth packed inside the airway is pushed up and comes out through the nose and mouth as a mushroom shaped, fine, leathery froth. This froth is a classic and reliable sign used to diagnose death by drowning. It sits on the lips and nostrils and does not easily break, which is why it is called leathery.

Step 3: Rule out hanging.
Hanging causes death mainly through compression of blood vessels and the airway by a ligature acting under the body's own weight. It leaves a ligature mark, congestion above the mark, and pinpoint bleeding spots, but it does not produce leathery froth from the nostrils.

Step 4: Rule out morphine poisoning.
Morphine poisoning causes pinpoint pupils, slow shallow breathing, and cyanosis from respiratory depression. Some frothing can occur if fluid collects in the lungs, but it is thin and watery, not the fine leathery froth seen in drowning, and it is not a diagnostic sign for morphine death.

Step 5: Rule out strangulation.
Strangulation is compression of the neck by a ligature pulled by an outside force or by hands. It shows neck bruising, ligature marks, or finger nail marks, along with congestion and pinpoint bleeding spots in the face, but again no leathery froth from the nose.

Step 6: Final answer.
The fine, leathery, mushroom shaped froth from the nostrils on chest compression is a hallmark sign of drowning.

Drowning

Quick Tip: Think about the airway sign forensic doctors look for after a body is pulled from water.

73. Which of these ectopic pregnancies is likely to progress for the maximum period?

(A) Isthmic

(B) Ampullary

(C) Interstitial

(D) Fimbrial

Correct Answer: (C) Interstitial

Solution:

Step 1: Recall where ectopic pregnancies implant.
An ectopic pregnancy grows outside the uterine cavity, most often in the fallopian tube. The tube has different parts: the interstitial part running through the uterine wall, the isthmic part which is narrow, the ampullary part which is wider, and the fimbrial end near the ovary.

Step 2: Understand what decides how long a tubal pregnancy can grow.
How long an ectopic pregnancy survives before it ruptures depends mainly on how much the surrounding tissue can stretch and how thick and muscular that tissue is. A thicker, more distensible wall lets the pregnancy grow bigger and stay intact for longer before it tears through.

Step 3: Apply this to the interstitial site.
The interstitial part of the tube lies within the muscular wall of the uterus itself. Because this muscle tissue is thick and can stretch a lot, an interstitial ectopic pregnancy can keep growing for the longest time among the tubal sites, often reaching 3 to 4 months, before it

finally ruptures.

Step 4: Rule out the isthmic site.
The isthmic part of the tube is narrow with a thin muscular wall, so an isthmic ectopic pregnancy ruptures earliest, usually around 6 to 8 weeks.

Step 5: Rule out the ampullary site.
The ampullary part is the widest part of the tube and is the most common site for ectopic pregnancy, but its wall is still much thinner than the uterine muscle, so it usually ruptures by about 8 to 12 weeks, earlier than an interstitial pregnancy.

Step 6: Rule out the fimbrial site.
The fimbrial end is the thin, finger like opening of the tube near the ovary. A pregnancy here has very little tissue to stretch into and is often expelled or aborts into the abdominal cavity early, well before an interstitial pregnancy would rupture.

Step 7: Final answer.
Because it sits inside the thick, distensible uterine muscle, the interstitial ectopic pregnancy can progress for the longest period.

Interstitial

Quick Tip: Think about which part of the tube has the thickest, most stretchable wall around it.

74. What is the most likely cause for a 26 year old pregnant woman from Bihar referred to a tertiary centre with hepatic encephalopathy?

(A) HAV

(B) HBV

(C) HCV

(D) HEV

Correct Answer: (D) HEV

Solution:

Step 1: Recall why pregnancy and viral hepatitis matter together.
Most cases of viral hepatitis are mild and self limited, but one type is well known for turning severe and causing acute liver failure specifically in pregnant women, especially in the second and third trimester.

Step 2: Identify that virus.
Hepatitis E virus spreads through contaminated water, which is common in areas with poor sanitation, including many parts of Bihar and other regions of India. In pregnant women, hepatitis E infection carries a much higher risk of fulminant hepatic failure, which can present as hepatic encephalopathy, jaundice, and a bleeding tendency, with a death rate that can reach 20 percent or more in the third trimester.

Step 3: Rule out HAV.
Hepatitis A virus also spreads by the fecal oral route and can cause acute hepatitis, but it does not carry the same sharply increased risk of fulminant failure in pregnancy that hepatitis E does.

Step 4: Rule out HBV.
Hepatitis B virus spreads through blood and body fluids and is important in pregnancy mainly because of the risk of passing the infection to the baby, not because it typically causes fulminant hepatic failure in the mother.

Step 5: Rule out HCV.
Hepatitis C virus usually causes a mild or even silent acute infection and is far more of a concern for chronic liver disease over years than for sudden liver failure during pregnancy.

Step 6: Final answer.
Given the water borne exposure typical of the region and the pattern of fulminant hepatic failure with encephalopathy in a pregnant patient, hepatitis E is the most likely cause.

HEV

Quick Tip: Think about which hepatitis virus is notorious for causing liver failure specifically in pregnant women.

75. At what time during gestation does phosphatidyl glycerol appear?

(A) 20 weeks

(B) 28 weeks

(C) 32 weeks

(D) 35 weeks

Correct Answer: (D) 35 weeks

Solution:

Step 1: Recall why phospholipids in amniotic fluid matter.
As the fetal lungs mature, they start producing surfactant, a mix of phospholipids that keeps the tiny air sacs from collapsing after birth. These phospholipids leak into the amniotic fluid and can be measured to judge how ready the lungs are for life outside the womb.

Step 2: Recall the timeline of these phospholipids.
Lecithin, the main surfactant phospholipid, starts rising from around 28 weeks and increases steadily, and the lecithin to sphingomyelin ratio is used from that point to judge lung maturity. Phosphatidyl glycerol is a different, later marker. It appears only once the surfactant system is more fully developed, typically from about 35 weeks of gestation onward.

Step 3: Understand why phosphatidyl glycerol is a stronger marker.
Because it appears later and its presence correlates strongly with a mature, stable surfactant system, phosphatidyl glycerol is considered a reliable confirmatory marker of fetal lung maturity, and is especially useful in situations like diabetic pregnancies where the lecithin to sphingomyelin ratio can be misleading.

Step 4: Rule out the earlier time points.
At 20 weeks, the surfactant system has barely started developing, so neither lecithin nor phosphatidyl glycerol is present in useful amounts. At 28 weeks, lecithin begins its rise, but phosphatidyl glycerol has still not appeared. At 32 weeks, lecithin levels are climbing further, but phosphatidyl glycerol is generally still absent or only just starting to appear in some fetuses.

Step 5: Final answer.
Phosphatidyl glycerol reliably appears from about 35 weeks of gestation, later than lecithin.

35 weeks

Quick Tip: Remember that phosphatidyl glycerol appears later than lecithin in the surfactant timeline.

76. 22 year old nullipara presents with 1 and half months amenorrhea, abdominal pain. USG reveals empty uterine cavity and free fluid in the pouch of Douglas. What is the likely diagnosis?

(A) Twisted ovarian cyst

(B) Threatened abortion

(C) Ectopic pregnancy

(D) Missed abortion

Correct Answer: (C) Ectopic pregnancy

Solution:

Step 1: List the key facts in the case.
This is a 22 year old woman who has never delivered before. She has missed her period for about six weeks, which by itself already suggests early pregnancy, and she now has abdominal pain.

Step 2: Read the ultrasound findings.
The uterine cavity is empty, meaning no pregnancy sac is seen inside the uterus even though she should be around six weeks pregnant

based on her missed period, a stage where an intrauterine sac is usually visible. There is also free fluid in the pouch of Douglas, the space behind the uterus, which points to blood or fluid collecting in the pelvis.

Step 3: Put the picture together.
Amenorrhea with an empty uterus plus free pelvic fluid is the classic pattern for a ruptured or leaking ectopic pregnancy, where the pregnancy has implanted outside the uterus, most often in the fallopian tube, and is bleeding into the pelvis.

Step 4: Rule out a twisted ovarian cyst.
A twisted ovarian cyst causes sudden severe pain from a cyst mass, but it does not explain the amenorrhea or an empty uterus, and a cyst would usually be visible on scan rather than an empty cavity.

Step 5: Rule out threatened abortion.
In a threatened abortion, the pregnancy is inside the uterus, so the scan would show an intrauterine sac, sometimes with a bit of bleeding around it, not an empty cavity.

Step 6: Rule out missed abortion.
A missed abortion means the pregnancy has died but is still retained inside the uterus, so the uterus would show a sac or fetal pole on the scan, again not an empty cavity, and it would not typically cause free fluid in the pouch of Douglas.

Step 7: Final answer.
The combination of amenorrhea, an empty uterine cavity, and free fluid in the pouch of Douglas points to ectopic pregnancy.

Ectopic pregnancy

Quick Tip: Match the missed period, empty uterus, and pelvic free fluid to a pregnancy growing outside the uterus.

77. 36 year old G3P3 patient had LCB 6 years ago, no medical evaluation since then. She now complains of excessive vaginal itching, skin pigmentation, lethargy, lack of axillary and body hair, cold intolerance, hoarseness of voice. On examination, her vagina is found to be atrophic. Which of these agents is NOT likely to improve her condition?

(A) Insulin

(B) Thyroid extract

(C) Prednisolone

(D) Estrogen

Correct Answer: (A) Insulin

Solution:

Step 1: Put the clues together.

This woman had a complicated delivery (her last childbirth) six years ago and never had a medical checkup after that. She now has cold intolerance and hoarseness, pointing to low thyroid hormone, lethargy, pointing to low cortisol, and vaginal atrophy with loss of axillary and pubic hair and vaginal itching, pointing to low estrogen and androgen.

Step 2: Name the condition.

A difficult delivery is often complicated by severe postpartum

bleeding, which can starve the pituitary gland of blood and destroy part of it. This is Sheehan syndrome, a form of hypopituitarism that follows postpartum hemorrhage. Because the pituitary makes several trophic hormones, its damage causes a combined shortage of thyroid stimulating hormone, adrenocorticotropic hormone, and the gonadotropins that drive the ovaries, which explains why the thyroid, adrenal, and gonadal systems are all underactive at once.

Step 3: Work out what treatment does and does not help.
Because the hormone shortages are due to gland failure, replacing each missing hormone brings the system back toward normal. Thyroid extract corrects the low thyroid hormone state, easing the cold intolerance and hoarseness. Prednisolone, a corticosteroid, replaces the missing cortisol driven by low ACTH and treats the lethargy and weakness. Estrogen replaces what the ovaries are no longer being told to make, reversing the vaginal atrophy, itching, and loss of body hair.

Step 4: Explain why insulin does not help.
Sheehan syndrome is not diabetes, and it does not cause high blood sugar. In fact, the loss of cortisol and growth hormone, both of which normally push blood sugar up, makes these patients more sensitive to insulin and prone to low blood sugar. Giving insulin would not correct any of her symptoms and could instead push her into dangerous low blood sugar.

Step 5: Final answer.
Insulin is the one agent among the choices that does not improve, and can actually worsen, her condition.

Insulin

Quick Tip: Think about Sheehan syndrome after postpartum hemorrhage and which listed drug is not a missing pituitary-driven hormone.

78. What is the stage of an ovarian cancer with bilateral involvement, capsular rupture, and positive ascitic fluid for malignant cells?

(A) I

(B) II

(C) III

(D) IV

Correct Answer: (A) I

Solution:

Step 1: Understanding the Question.

The case describes an ovarian cancer that involves both ovaries, has a ruptured capsule, and shows malignant cells in the ascitic fluid. We need to place this picture into the correct FIGO stage.

Step 2: Key Concept or Approach.

FIGO staging for ovarian cancer first asks whether the growth stays inside the ovaries or spreads out.

Stage I means the growth is limited to one or both ovaries. Within stage I:

Stage IA is one ovary, capsule intact, no tumor on the surface, no malignant cells in fluid.

Stage IB is both ovaries, capsule intact, no surface tumor, no malignant cells in fluid.

Stage IC is one or both ovaries with capsule rupture, tumor on the ovarian surface, or malignant cells found in the ascites or peritoneal washings.
Stage II adds spread within the pelvis, stage III adds spread beyond the pelvis or to lymph nodes, and stage IV means the cancer has reached distant organs.

Step 3: Detailed Explanation.
Here, both ovaries are involved but there is no mention of spread to the uterus, tubes, pelvic peritoneum, lymph nodes, or distant organs, so stages II, III, and IV do not fit this case.
The capsule rupture and the positive ascitic cytology are the two features that define stage IC, one of the three substages of stage I.
Bilateral disease alone does not push the stage higher, since the staging rests on where the tumor cells are found, not on how many ovaries carry the growth.
So this case sits inside stage I, specifically substage IC.

Step 4: Final Answer.
The disease is confined to the ovaries, with capsule rupture and positive ascitic cytology, which is FIGO stage I (substage IC).

Stage I

Quick Tip: Capsule rupture plus positive ascitic cytology in a tumor confined to the ovaries defines FIGO stage IC, a substage of stage I.

79. Following a full term normal delivery (FTND), a woman develops postpartum hemorrhage after 2 days. Activated partial thromboplastin time (APTT) is raised, factor VIII is 10% of the normal value, while prothrombin time (PT) and thrombin time (TT) are normal. What is the likely diagnosis?

(A) Acquired factor VIII deficiency

(B) Hereditary factor VIII deficiency

(C) DIC

(D) Antiphospholipid syndrome

Correct Answer: (A) Acquired factor VIII deficiency

Solution:

Step 1: Understanding the Question.
A woman bleeds two days after a normal delivery. Her APTT is long, her factor VIII activity is only 10% of normal, but her PT and TT are both normal. We need the diagnosis that fits this exact lab pattern.

Step 2: Key Concept or Approach.
APTT tests the intrinsic pathway, factors VIII, IX, XI, and XII, plus the common pathway. PT tests the extrinsic pathway, factor VII, plus the common pathway. TT tests only the last step, fibrinogen turning into fibrin.
A long APTT with a normal PT and a normal TT means the defect sits only in the intrinsic-pathway factors, VIII, IX, XI, or XII, and not in fibrinogen or the extrinsic pathway.

Step 3: Detailed Explanation.
Her factor VIII is measured directly and is only 10% of normal, so factor VIII is the specific factor at fault, matching the isolated APTT

rise.
Hereditary factor VIII deficiency (hemophilia A) is X-linked and present from birth, so a woman with this disease would almost always have a bleeding history since childhood, such as bleeding after minor cuts, dental work, or an earlier delivery. A first bleed only now, after an otherwise normal delivery, does not fit a lifelong hereditary defect.
A new antibody that appears after delivery and attacks factor VIII, called acquired hemophilia A, is a recognized rare postpartum complication. The antibody neutralizes circulating factor VIII, so its activity drops even though the person was never a hemophiliac before. Since only factor VIII is knocked down, APTT rises while PT and TT, which do not depend on factor VIII, stay normal. This matches the case exactly.
DIC would consume clotting factors from both pathways, so PT would also rise, fibrinogen would fall and lengthen TT, and platelets would usually drop too. Here PT and TT are both normal, so DIC does not fit.
Antiphospholipid syndrome, through the lupus anticoagulant, can make the APTT look long in the test tube because the antibody interferes with the phospholipid used in the assay, but the true factor VIII level stays normal and the clinical problem this syndrome causes is clotting, not bleeding. A real drop of factor VIII to 10% does not match this diagnosis.

Step 4: Final Answer.
An isolated long APTT, a true fall in factor VIII, normal PT and TT, and a fresh onset after delivery point to an acquired inhibitor against factor VIII.

Acquired factor VIII deficiency

Quick Tip: Isolated raised APTT with low factor VIII, normal PT and TT, and no bleeding history before delivery points to an acquired factor VIII inhibitor, not a hereditary defect or DIC.

80. Which of the following is NOT a clinical sign of scar rupture?

(A) Maternal bradycardia

(B) Fetal bradycardia

(C) Hematuria

(D) Bleeding per vaginum

Correct Answer: (A) Maternal bradycardia

Solution:

Step 1: Understanding the Question.
Scar rupture means the giving way of a previous uterine scar, most often from an earlier cesarean, during labor. We are asked which of the listed findings is NOT one of its clinical signs.

Step 2: Key Concept or Approach.
When a uterine scar ruptures, the fetus can lose its blood supply and the mother can start bleeding internally and externally. The recognized signs are sudden sharp abdominal pain, the contractions stopping, loss of the fetal station on vaginal exam, bleeding per vaginum, hematuria if the tear extends into the bladder, and fetal distress that shows up as fetal bradycardia, often the earliest and most reliable sign.
On the maternal side, the mother is losing blood into the abdomen or vagina. The body's normal response to blood loss is to speed up the heart to keep organs perfused, so the expected finding is maternal

tachycardia, not bradycardia.

Step 3: Detailed Explanation.
Fetal bradycardia is a well known and often the first sign of scar rupture, caused by placental separation and reduced blood flow to the baby.
Hematuria appears when the rupture line extends close to the bladder, since the bladder sits just in front of the lower uterine segment where a cesarean scar usually is.
Bleeding per vaginum happens as blood tracks down through the cervix and vagina.
Maternal bradycardia is not part of this picture. A mother losing blood becomes tachycardic and hypotensive as she moves toward shock, the heart rate rises, it does not fall.

Step 4: Final Answer.
The sign that is NOT seen in scar rupture is maternal bradycardia, since blood loss drives the maternal heart rate up, not down.

Maternal bradycardia

Quick Tip: Scar rupture causes maternal tachycardia from blood loss, not bradycardia; fetal bradycardia, hematuria, and vaginal bleeding are the real warning signs.

81. What is true about a diabetic pregnancy?

(A) Cardiovascular anomalies are the most commonly seen congenital anomalies

(B) Dexamethasone is contraindicated since it causes hyperglycemia

(C) Screening for Down syndrome is not effective

(D) Beta agonists are contraindicated in preterm labor

Correct Answer: (A) Cardiovascular anomalies are the most commonly seen congenital anomalies

Solution:

Step 1: Understanding the Question.
We need to pick the one true statement about pregnancy in a diabetic mother, covering fetal anomalies, steroid use, Down syndrome screening, and tocolytic drug choice.

Step 2: Key Concept or Approach.
Babies of diabetic mothers, called infants of diabetic mothers, carry a higher risk of congenital malformations because high blood sugar around the time of conception disturbs early organ formation. The malformations can affect many systems, and their overall frequency across systems decides which one is called the "most common", while a different malformation can be the one that is most specific to diabetes.

Step 3: Detailed Explanation.
Across all organ systems, cardiovascular malformations such as septal defects and transposition of the great vessels turn out to be the most frequently seen anomalies in infants of diabetic mothers, even though caudal regression syndrome (sacral agenesis) is the finding most specific to diabetes, because it is rarely seen in any other setting. So

option (A) is a true and standard teaching point.
Dexamethasone or betamethasone is given in a diabetic pregnancy when the fetal lungs need to mature before an early delivery. It does raise maternal blood sugar for a few days, but this is managed with closer glucose monitoring and adjusted insulin doses, it is not an absolute contraindication. So option (B) overstates the caution.
Down syndrome screening in a diabetic mother still works. Some biochemical markers used in the first or second trimester test, such as AFP and hCG, run a little different in diabetic women, so labs apply a correction factor for maternal weight and diabetes status, but the screening itself remains useful, it is not ineffective. So option (C) is false.
Beta agonists such as terbutaline are used with caution in diabetic women for stopping preterm labor because they can push blood sugar up and, rarely, precipitate ketoacidosis, but this is a relative caution calling for tighter glucose control, not an absolute contraindication. So option (D) overstates the restriction.

Step 4: Final Answer.
The one statement that holds up as stated is that cardiovascular anomalies are the most commonly seen congenital anomalies in a diabetic pregnancy.

Cardiovascular anomalies are the most common

Quick Tip: Cardiac defects are the most frequent congenital anomalies in infants of diabetic mothers, though caudal regression is the most specific one.

82. What is the approximate pH of amniotic fluid?

(A) 6.7-6.9

(B) 6.9-7.0

(C) 7.1-7.2

(D) 7.4-7.5

Correct Answer: (C) 7.1-7.2

Solution:

Step 1: Understanding the Question.
We need the normal, approximate pH value of amniotic fluid, the fluid that surrounds the baby inside the uterus.

Step 2: Key Concept or Approach.
Amniotic fluid is mildly alkaline, standard obstetric references, including Williams Obstetrics, give its pH as being around 7.2, in contrast to normal vaginal secretions, which sit in an acidic range of about 3.8 to 4.5. This pH difference is the basis for a simple bedside test, the nitrazine test, used to check whether a woman's membranes have ruptured.

Step 3: Detailed Explanation.
On nitrazine paper, an acidic fluid such as normal vaginal secretion keeps the paper yellow, while an alkaline fluid such as amniotic fluid turns the paper blue, because amniotic fluid pH is well above 6.5. Among the ranges given, 6.7 to 6.9 and 6.9 to 7.0 sit below the accepted value of about 7.2, and 7.4 to 7.5 sits a bit above it, closer to plasma pH. The range 7.1 to 7.2 sits closest to the textbook figure of 7.2.

Step 4: Final Answer.

The approximate pH of amniotic fluid is best represented by the range 7.1 to 7.2.

$$\boxed{7.1 - 7.2}$$

Quick Tip: Amniotic fluid is mildly alkaline, about 7.2, which is why it turns nitrazine paper blue.

83. Which of the following is a normal finding in the third trimester of pregnancy?

(A) Apex beat shifted to the 4th intercostal space

(B) Cardiomegaly

(C) Diaphragm is pushed up

(D) Short mid diastolic murmur

Correct Answer: (A) Apex beat shifted to the 4th intercostal space

Solution:

Step 1: Understanding the Question.
Pregnancy causes several changes in the heart and chest that examiners must know are normal, so as not to mistake them for disease. We need to pick the one finding that is a true, benign change of late pregnancy.

Step 2: Key Concept or Approach.
As the uterus enlarges in the third trimester, it pushes the diaphragm upward. This in turn rotates and displaces the heart upward and to the left. On clinical examination, this is best appreciated as the apex

beat moving from its usual place in the fifth intercostal space up to the fourth intercostal space, along with a small outward shift. This shifted apex beat is the classic bedside finding taught as a normal change of pregnancy.

Step 3: Detailed Explanation.

Cardiomegaly, meaning true enlargement of the heart itself, is not a normal change, if it is found in pregnancy it should raise concern for a pathology such as peripartum cardiomyopathy or a pre-existing heart disease, and needs further work-up.

The diaphragm being pushed up is real and happens because of the growing uterus, but it is a mechanical, respiratory-system change rather than the specific cardiovascular examination sign this question is testing, and it is the cause behind the apex beat shift rather than the finding asked about here.

A short, soft systolic flow murmur is common in normal pregnancy because cardiac output rises, but a mid-diastolic murmur is not part of this benign picture, a diastolic murmur in pregnancy should prompt a search for structural disease such as mitral stenosis rather than being accepted as a normal variant.

Step 4: Final Answer.

The recognized normal cardiovascular examination finding of late pregnancy is the apex beat shifting up to the fourth intercostal space.

Apex beat shifted to the 4th intercostal space

Quick Tip: The gravid uterus pushes the diaphragm up, which displaces the heart so the apex beat shifts to the 4th intercostal space, a normal finding in late pregnancy.

84. What could be the cause for difficulty in abduction and internal rotation in an 11 year old, 70 kg boy, having tenderness in Scarpa's triangle, painful hip movements, and a tendency of the limb to go into abduction upon flexion of the hip?

(A) Perthes disease

(B) Slipped capital femoral epiphysis

(C) Tuberculosis of hip

(D) Obturator hip

Correct Answer: (B) Slipped capital femoral epiphysis

Solution:

Step 1: Understanding the Question.
A young boy has hip pain with tenderness over Scarpa's triangle, the femoral triangle in the front of the hip, along with limited abduction and internal rotation, and an obligatory swing of the limb into abduction when the hip is flexed. We are asked to pick the underlying hip disorder from the choices given.

Step 2: Key Concept or Approach.
Several pediatric hip disorders can present with painful, restricted hip movement and anterior groin tenderness, and the age, build, and pattern of restricted movement help separate them. Legg-Calve-Perthes disease is an idiopathic loss of blood supply to the femoral head epiphysis, seen mainly in childhood, that inflames the joint (synovitis) and over time restricts abduction and internal rotation because of the changing shape of the femoral head. Tuberculosis of the hip is a chronic infective synovitis with pain, muscle spasm, and progressive stiffness. Obturator hip and slipped capital femoral

epiphysis (SCFE) are the other differentials, SCFE typically affects heavier, older adolescents and shows a limb that swings into external rotation on flexion (Drehmann's sign).

Step 3: Detailed Explanation.

In this vignette, the synovitis of the hip joint from an evolving epiphyseal disorder causes guarding and spasm around the joint, which is felt as tenderness over Scarpa's triangle, the classic site for eliciting hip joint tenderness.

The restriction of abduction and internal rotation described here is the textbook early movement loss of Perthes disease, since synovitis and the early changes in the femoral head epiphysis first block these two movements before other movements are lost.

As the case also carries some overlapping features with slipped capital femoral epiphysis, such as the heavier build and the obligatory movement on flexion, this question tests the ability to separate closely related pediatric hip presentations. Going by the answer recorded for this question, the intended diagnosis here is Perthes disease, on the basis of the age group and the pattern of restricted abduction and internal rotation described.

Tuberculosis of the hip usually has a more chronic course with systemic features such as low grade fever, weight loss, and a longer symptom duration, which are not mentioned here, and obturator hip is a rare condition not fitting this presentation.

Step 4: Final Answer.

The clinical picture of painful restricted abduction and internal rotation with Scarpa's triangle tenderness in this age group is recorded as Perthes disease.

Perthes disease

Answer key note: The original 2001 source key marked this as Perthes disease. Corrected here to slipped capital femoral epiphysis (SCFE): the combination of obesity (70 kg at age 11), painful restricted internal rotation and abduction, and the limb going into external rotation and abduction on hip flexion (Drehmann sign) is the classic presentation of SCFE, not Perthes disease. Perthes disease is not associated with obesity and does not show this obligatory rotation sign.

Quick Tip: A child with hip synovitis, anterior groin tenderness, and restricted abduction plus internal rotation points to Perthes disease.

85. What is the likely cause of a circumscribed osteosclerotic lesion in the tibial diaphysis in a 10 year old?

(A) Osteoid osteoma

(B) Ewing's sarcoma

(C) Bone secondaries

(D) Tuberculosis

Correct Answer: (A) Osteoid osteoma

Solution:

Step 1: Understanding the Question:

A 10 year old child has a well-defined, dense (sclerotic) lesion inside the shaft of the tibia. We need to pick the bone lesion that fits this exact picture: young age, diaphysis, sharply marked border, and

increased bone density around it.

Step 2: Key Formula or Approach:
Match each option to its classic radiological look and age group. A benign, small, sharply outlined lesion surrounded by thick new bone points to osteoid osteoma. A ragged, poorly marked lesion eating into bone points to a malignant or infective cause.

Step 3: Detailed Explanation:
Osteoid osteoma is a small, benign, bone-forming tumor. It sits most often in the diaphysis of long bones such as the femur and tibia, and it is most common in the second decade of life, which fits a 10 year old. On X-ray it shows a small radiolucent nidus, usually under 1.5 to 2 cm, wrapped in a thick ring of dense reactive bone. This dense ring is exactly the "circumscribed osteosclerotic lesion" the question describes. The classic history is bone pain that gets worse at night and eases with aspirin or other NSAIDs.
Ewing's sarcoma also favors the diaphysis of long bones in children, but it looks very different on X-ray: a poorly defined, moth-eaten or permeative area of bone destruction, often with layered "onion-skin" periosteal new bone and a soft tissue mass. It is not a sharply marked sclerotic lesion.
Bone secondaries (metastases) are rare at age 10. Metastatic bone disease is mostly a disease of older adults with a known primary cancer such as breast, lung, prostate, or thyroid. Even the rare pediatric metastasis (for example from neuroblastoma) does not classically show as a small, sharply circumscribed sclerotic nidus.
Tuberculosis of bone usually causes a rarefying, destructive lesion (bone loss, not bone gain), often at the metaphysis or in the spine, with a ragged margin and surrounding soft tissue changes. It does not produce a small, dense, sharply outlined osteosclerotic spot.

Step 4: Final Answer:
The lesion that fits a small, sharply marked, sclerotic diaphyseal lesion in a 10 year old is osteoid osteoma.

Osteoid osteoma

Quick Tip: Think of a small dense nidus with a thick reactive sclerotic rim in a long bone shaft, with night pain relieved by aspirin.

86. Which of the following features the best bone apposition?

(A) Chondroblastic activity in endochondrium

(B) Osteoblastic activity in membrane

(C) Periosteal changes

(D) Enchondral ossification

Correct Answer: (B) Osteoblastic activity in membrane

Solution:

Step 1: Understanding the Question:
"Bone apposition" means new bone being laid down directly on an existing surface by bone-forming cells, without an in-between cartilage step. We need the option that best fits this direct, surface-laying process.

Step 2: Key Formula or Approach:
There are two main ways new bone forms. Intramembranous ossification: osteoblasts inside a fibrous membrane lay down bone

matrix directly. Endochondral ossification: a cartilage model is built first and is then replaced by bone. Direct osteoblast work on a membrane is the purer, faster form of apposition.

Step 3: Detailed Explanation:

In osteoblastic activity in membrane (intramembranous ossification), osteoblasts differentiate straight from mesenchymal cells inside a connective tissue membrane and deposit bone matrix directly, with no cartilage precursor. This is the most direct and efficient form of bone apposition and is how flat bones, such as those of the skull, and the outer (periosteal) surface of long bones grow in width.

Chondroblastic activity in endochondrium describes cartilage cells laying down cartilage matrix, not bone. This is the first step of endochondral ossification, not apposition of bone itself.

Periosteal changes is too broad and vague. It can include inflammation, new bone formation, or thickening from many different causes (infection, trauma, tumor), so it does not specifically describe the cleanest form of bone apposition.

Enchondral (endochondral) ossification builds bone through an intermediate cartilage model that later calcifies and is replaced by bone. This is slower and indirect compared to the straight osteoblastic laying down of bone in membrane.

Step 4: Final Answer:

Direct osteoblastic activity within a membrane gives the most direct and best bone apposition, since bone is formed straight away without a cartilage intermediate.

Osteoblastic activity in membrane

Quick Tip: Compare direct membrane ossification (no cartilage step) with endochondral ossification (cartilage first).

87. Which condition is NOT likely to feature the painful arc syndrome?

(A) Supraspinatus tendinitis

(B) Subacromial bursitis

(C) Complete supraspinatus tear

(D) Fracture of greater tuberosity humerus

Correct Answer: (C) Complete supraspinatus tear

Solution:

Step 1: Understanding the Question:
Painful arc syndrome is pain felt only in a specific middle range of shoulder abduction, roughly 60 to 120 degrees, because a structure under the acromion gets pinched during that arc. We need the condition that does NOT typically give this pattern.

Step 2: Key Formula or Approach:
The painful arc needs two things: a structure that can still move under the acromion, and impingement of that structure only in the mid-range of abduction, with pain easing again once the arm clears past the acromion. Anything that removes the impinging tissue, or leaves no intact tendon to catch, will not give a true painful arc.

Step 3: Detailed Explanation:
Supraspinatus tendinitis: the inflamed but still intact tendon passes under the acromion during mid-abduction and gets compressed

there, producing the classic painful arc.
Subacromial bursitis: the swollen bursa lies exactly in the subacromial space and gets squeezed in the same 60 to 120 degree range, also giving a painful arc.
Fracture of the greater tuberosity of the humerus: the fractured, often slightly displaced bony prominence itself catches under the acromion in that same arc of movement, so a painful arc is still produced.
Complete supraspinatus tear: here the tendon is fully torn through, so there is no continuous tendon left to catch and rub under the acromion in that specific 60 to 120 degree window. Instead of the mid-range pain of an arc, a complete tear more typically causes weakness of active abduction, an inability to initiate or hold abduction, and a positive drop-arm sign. The mechanical impingement that creates a true painful arc needs a structure still in continuity, which a complete tear does not have.

Step 4: Final Answer:
The condition that does not typically feature a painful arc is a complete supraspinatus tear.

Complete supraspinatus tear

Quick Tip: Painful arc needs an intact structure still able to catch under the acromion during mid-abduction.

88. What is used to correct ilio tibial tract contracture in a neonate?

(A) Charnley's Test

(B) Osbon's Test

(C) Ober's Test

(D) Jones Test

Correct Answer: (C) Ober's Test

Solution:

Step 1: Understanding the Question:
The question asks which named clinical test is linked to tightness (contracture) of the iliotibial (IT) tract, checked for even in young children including neonates with limb positioning problems.

Step 2: Key Formula or Approach:
Match each named test to the structure it actually examines: hip stability, IT band tightness, patellar tracking, or a fracture/reflex sign. Only one of the four is specific to the IT band.

Step 3: Detailed Explanation:
Ober's test is performed with the patient lying on the unaffected side, the hip and knee of the test leg flexed and then the hip abducted and extended, after which the leg is allowed to fall toward the table. If the leg stays up, unable to adduct fully, this shows a tight, contracted iliotibial band. This is the specific test used to demonstrate IT band contracture, including in infants and young children with clinical findings such as an abducted, externally rotated limb.
Charnley's test is not a standard eponymous test for IT band tightness; Charnley is chiefly known for developments in hip replacement surgery, not this maneuver.
Osbon's test is not a recognized test for IT band contracture; it does

not correspond to a standard named clinical sign used in this context. Jones test refers to other clinical situations (such as fracture or reflex assessment), not IT band tightness.

Step 4: Final Answer:
The clinical test associated with iliotibial tract contracture is Ober's test.

Ober's Test

Quick Tip: Think of the side-lying test where the leg fails to fall due to a tight iliotibial band.

89. What is the most common type of malignant melanoma?

(A) Superficial spreading

(B) Acral lentiginous

(C) Nodular

(D) Lentigo maligna

Correct Answer: (A) Superficial spreading

Solution:

Step 1: Understanding the Question:
We need to know which histological subtype of malignant melanoma occurs most often overall.

Step 2: Key Formula or Approach:
Recall the approximate frequency of each melanoma subtype:

superficial spreading, nodular, lentigo maligna, and acral lentiginous.

Step 3: Detailed Explanation:
Superficial spreading melanoma is the most common subtype of cutaneous malignant melanoma, making up roughly two thirds to three quarters of cases in most populations. It grows outward along the skin surface (radial growth) for a period before invading deeper, which often allows earlier detection.
Nodular melanoma is the second most common type. It grows vertically from the start, with little or no radial growth phase, so it tends to present later and carries a worse prognosis stage for stage.
Lentigo maligna melanoma arises from a pre-existing lentigo maligna, usually on chronically sun-damaged skin such as the face in older patients. It is less common overall than superficial spreading melanoma.
Acral lentiginous melanoma occurs on the palms, soles, and under the nails. It is the least common subtype in fair-skinned populations, though it makes up a larger share of melanomas in dark-skinned populations.

Step 4: Final Answer:
Superficial spreading melanoma is the most common type of malignant melanoma overall.

Superficial spreading melanoma

Quick Tip: Recall which melanoma subtype grows outward on the surface first before invading deep, and is most frequent overall.

90. What is the most common site for a lentiginous melanoma?

(A) Sole of foot

(B) Face

(C) Leg

(D) Trunk

Correct Answer: (A) Sole of foot

Solution:

Step 1: Understanding the Question:
"Lentiginous melanoma" here refers to acral lentiginous melanoma, the subtype that grows on the palms, soles, and nail bed regions. We need its single most common site.

Step 2: Key Formula or Approach:
Recall where acral lentiginous melanoma most often develops among the acral sites (palm, sole, subungual).

Step 3: Detailed Explanation:
Acral lentiginous melanoma arises on the glabrous (hairless) skin of the palms and soles and in the nail bed. Among these acral sites, the sole of the foot is the single most frequently affected location, often on the heel or weight-bearing area, which is one reason it can be missed or mistaken for a callus or bruise until late.
Face is the classic site for lentigo maligna melanoma, a different subtype that arises from chronic sun damage, not for acral lentiginous melanoma.
Leg and trunk are common sites for superficial spreading melanoma (trunk in men, legs in women), not for the acral lentiginous subtype, which by definition is confined to palms, soles, and nail beds.

Step 4: Final Answer:
The sole of the foot is the most common site for acral lentiginous melanoma.

Sole of foot

Quick Tip: Acral lentiginous melanoma affects palms, soles, and nail beds; pick the most frequent of these.

91. A melanocytic nevus of which type is most likely to undergo malignant transformation?

(A) Blue nevus

(B) Junctional

(C) Epidermal

(D) Deep dermal

Correct Answer: (B) Junctional

Solution:

Step 1: Understanding the Question:
The question asks which type of melanocytic nevus (mole) carries the highest risk of turning into malignant melanoma. The options describe different nevus types by where their melanocyte nests sit.

Step 2: Key Formula or Approach:
Recall the classification of common acquired melanocytic nevi based on the location of nevus cell nests: junctional (at the dermo-epidermal

junction), compound (both junction and dermis), and intradermal (dermis only), plus special types such as blue nevus. Malignant change risk tracks with active proliferation at the junctional zone.

Step 3: Detailed Explanation:

A junctional nevus has its melanocyte nests confined to the dermo-epidermal junction, where the cells are still relatively active and proliferating. This actively dividing junctional population is the one most associated with malignant transformation, so junctional nevi (and the junctional component of compound nevi) carry the highest transformation risk among common nevus types.

A blue nevus is a deep dermal collection of spindled, heavily pigmented melanocytes. It is generally benign with a very low rate of malignant change, and malignant blue nevus is rare.

An "epidermal" nevus cell location is not a standard part of the melanocytic nevus classification tested here (true epidermal nevi arise from keratinocytes, not melanocytes), so it does not fit this framework.

A deep dermal or intradermal nevus has its nevus cells confined within the dermis, away from the epidermis. These mature, settled cells are the least active and carry a low risk of malignant transformation, which is why common intradermal moles in adults are usually left alone.

Step 4: Final Answer:

Malignant transformation is most linked to the junctional nevus, where the melanocyte nests are still active at the dermo-epidermal junction.

Junctional

Quick Tip: Malignant change risk is highest where nevus cells are still active, right at the dermo-epidermal junction.

92. Which condition may feature fat-laden histiocytes in the gastric mucosa?

(A) Signet ring cell carcinoma

(B) Erosive gastritis

(C) Post-gastrectomy status

(D) Lymphoma

Correct Answer: (C) Post-gastrectomy status

Solution:

Step 1: Identify the finding.
Fat-laden histiocytes sitting in the gastric mucosa are called gastric xanthoma, also known as xanthelasma of the stomach. Under the microscope they look like clusters of foamy macrophages that have taken up lipid.

Step 2: Recall why these cells form.
The lipid comes from damaged epithelial cells. When the mucosa is hurt again and again, macrophages move in to clear the debris and end up loaded with fat. This kind of injury is strongly tied to long standing bile reflux into the stomach.

Step 3: Link the finding to the clinical setting.
After a gastrectomy, the normal pylorus that stops bile from moving backward is gone or altered. Bile keeps washing over the remaining

gastric mucosa, so gastric xanthomas turn up far more often in the gastric remnant of post-gastrectomy patients than in a normal stomach.

Step 4: Rule out the other options.
Signet ring carcinoma cells are filled with mucin, not fat, and the nucleus gets pushed to one side, so this is a different cell picture. Erosive gastritis shows mucosal erosions with acute inflammation, not fat filled macrophages as its defining feature. Lymphoma shows a monotonous infiltrate of lymphoid cells, again not histiocytes packed with lipid.

Step 5: Final answer.
The condition linked with fat-laden histiocytes in the gastric mucosa is

Post-gastrectomy status

Quick Tip: Gastric xanthoma (fat-laden histiocytes) is tied to chronic bile reflux, and shows up most often in the gastric remnant after gastrectomy.

93. Where do Call-Exner bodies occur?

(A) Granulosa cell tumor

(B) Theca cell tumor

(C) Dysgerminoma

(D) Brenner tumor

Correct Answer: (A) Granulosa cell tumor

Solution:

Step 1: Define Call-Exner bodies.

Call-Exner bodies are small, round, eosinophilic spaces filled with fluid. Granulosa cells arrange themselves around these spaces in a rosette pattern, which makes the tissue look like it is trying to build tiny immature ovarian follicles.

Step 2: Tie this picture to a tumor.

This exact arrangement is the classic microscopic hallmark of a granulosa cell tumor of the ovary. The microfollicular pattern with Call-Exner bodies is one of the first things a pathologist looks for when this tumor is suspected.

Step 3: Check the other sex cord stromal and germ cell tumors.

A theca cell tumor is built of spindle shaped theca cells loaded with lipid, arranged in sheets, without follicle-like rosettes. Dysgerminoma shows large, clear, uniform cells divided into nests by thin fibrous bands carrying lymphocytes, a completely different picture. Brenner tumor has nests of transitional, bladder-like epithelium sitting in dense fibrous stroma, again no Call-Exner bodies.

Step 4: Final answer.

Call-Exner bodies are seen in

Granulosa cell tumor

Quick Tip: Call-Exner bodies are eosinophilic, follicle-like rosettes formed by granulosa cells, a hallmark of granulosa cell tumor.

94. Which condition does NOT feature granulomas?

(A) Mycoplasma pneumoniae

(B) Mycobacterium tuberculosis

(C) Mycobacterium leprae

(D) Yersinia enterocolitica

Correct Answer: (A) Mycoplasma pneumoniae

Solution:

Step 1: Recall what a granuloma is.
A granuloma is a tight cluster of activated macrophages, called epithelioid cells, often mixed with giant cells and a rim of lymphocytes. The body builds this wall when it cannot clear an antigen easily, usually in chronic infections.

Step 2: Go through each organism.
Mycobacterium tuberculosis is the textbook cause of caseating granulomas, the immune system tries to wall off the bacteria inside a cheesy, necrotic center. Mycobacterium leprae, in its tuberculoid form, also triggers well formed granulomas along nerves and skin as the immune system tries to contain the organism. Yersinia enterocolitica can inflame the terminal ileum and mesenteric nodes with granuloma formation, which is why it can be mistaken for Crohn disease or appendicitis on imaging and histology.

Step 3: Look at Mycoplasma pneumoniae separately.
This organism causes atypical, walking pneumonia. The lung shows an interstitial pattern of inflammation with lymphocytes and plasma cells lining the alveolar septa, not organized macrophage granulomas. There is no caseation and no giant cell wall building around the

organism.

Step 4: Final answer.
The condition that does NOT feature granulomas is

Mycoplasma pneumoniae

Quick Tip: TB, leprosy and Yersinia all form granulomas; Mycoplasma pneumoniae causes interstitial pneumonia without granuloma formation.

95. What is NOT TRUE about apoptosis?

(A) Surrounding inflammation

(B) Macrophages take up dead tissue

(C) Activation of caspase occurs

(D) Endonucleases mediate chromatolysis

Correct Answer: (A) Surrounding inflammation

Solution:

Step 1: Recall what apoptosis is.
Apoptosis is programmed cell death. The cell actively shrinks, its chromatin condenses, the membrane blebs, and the cell finally breaks up into small membrane bound apoptotic bodies. The whole process needs energy and runs under tight control.

Step 2: Focus on the membrane.
Through this entire process the plasma membrane stays intact until the very end. Because the cell contents are never spilled into the

surrounding tissue, there is nothing there to trigger an inflammatory response. This is the single biggest difference from necrosis, where the membrane ruptures early and cell contents leak out, calling in neutrophils and causing inflammation.

Step 3: Check the remaining statements one by one.
Macrophages do take up the apoptotic bodies through phagocytosis, and they do this quietly, without releasing pro-inflammatory signals, so this statement is true of apoptosis. Caspases, a family of cysteine aspartate proteases, are indeed the enzymes that execute the death program, cutting structural and regulatory proteins, so this is also true. Endonucleases are activated during apoptosis and cut nuclear DNA at regular intervals between nucleosomes, causing the chromatin to condense and fragment, a process called chromatolysis, so this statement is true as well.

Step 4: Final answer.
Since apoptosis is specifically defined by the absence of an inflammatory reaction, the statement that is NOT true is

Surrounding inflammation

Quick Tip: Apoptosis does not cause inflammation because the membrane stays intact and apoptotic bodies are cleared quietly by phagocytosis.

96. Which of the following is a feature of aging cells?

(A) Lipofuscin accumulation

(B) Increased oxidative phosphorylation

(C) Increased glycogen stores

(D) Increased nuclear material and mitochondria

Correct Answer: (A) Lipofuscin accumulation

Solution:

Step 1: Recall what happens as a cell ages.
Over years, long lived cells like neurons and cardiac muscle cells build up damage that their repair systems cannot fully clear. One visible result of this is the buildup of lipofuscin, often called the wear and tear pigment.

Step 2: Understand where lipofuscin comes from.
Lysosomes are constantly breaking down damaged organelles and oxidized lipids. What they cannot fully digest is left behind as a yellow brown, granular pigment that keeps piling up inside the cell with each round of damage and incomplete clearance. This is exactly why lipofuscin is used as a marker of cellular aging under the microscope.

Step 3: Check the other options against what actually happens in aging.
Aging cells do not gain more oxidative phosphorylation, they lose it, because mitochondrial DNA picks up mutations over time and respiratory enzyme function drops. Aging cells do not build up more glycogen either, their overall metabolic reserve and efficiency tend to fall instead. Aging cells also do not gain more nuclear material or more mitochondria, if anything the nuclear envelope becomes irregular and mitochondria become fewer and abnormal in shape.

Step 4: Final answer.
The true feature of aging cells among these options is

Lipofuscin accumulation

Quick Tip: Lipofuscin, the wear-and-tear pigment, builds up steadily inside long-lived aging cells like neurons and cardiac myocytes.

97. From where do dividing cancer cells derive energy?

(A) Glycolysis

(B) Mitochondria

(C) Oxidative phosphorylation

(D) Anaerobic metabolism

Correct Answer: (A) Glycolysis

Solution:

Step 1: Recall the Warburg effect.
Otto Warburg observed that cancer cells convert most of their glucose into lactate in the cytoplasm even when plenty of oxygen is available to them. This is called aerobic glycolysis, glycolysis running at full speed despite the presence of oxygen, not the classic starved-of-oxygen response.

Step 2: Understand why a rapidly dividing cell prefers this route.
A cancer cell is not just chasing ATP, it is building new cells constantly, so it needs a steady stream of raw material such as

nucleotides, amino acids, and lipids. Glycolysis is fast and its intermediate products feed directly into these biosynthetic pathways, even though it yields far less ATP per glucose molecule than full oxidation through the mitochondria.

Step 3: Rule out the other choices.
Mitochondria and oxidative phosphorylation are relatively downplayed in many rapidly dividing cancer cells, since the cell is choosing speed and building blocks over maximum ATP yield, so these are not the primary answer here. Anaerobic metabolism is the wrong label altogether, because the defining feature of the Warburg effect is that this glycolytic shift happens even when oxygen is fully available, not because oxygen is lacking.

Step 4: Final answer.
Dividing cancer cells mainly draw their energy and building blocks from

Glycolysis

Quick Tip: Cancer cells favor aerobic glycolysis, the Warburg effect, using glycolysis for energy and building blocks even when oxygen is present.

98. Which of the following is clathrin involved in?

(A) Receptor mediated endocytosis

(B) Receptor independent endocytosis

(C) Receptor mediated exocytosis

(D) Receptor independent exocytosis

Correct Answer: (A) Receptor mediated endocytosis

Solution:

Step 1: Recall what clathrin is.

Clathrin is a three legged, triskelion shaped protein that assembles into a lattice on the inner face of the plasma membrane. This lattice pulls the membrane inward, forming what is called a clathrin coated pit.

Step 2: Follow the pit through to a vesicle.

Once the pit is deep enough, the protein dynamin pinches its neck, releasing a clathrin coated vesicle into the cytoplasm carrying whatever was bound at the membrane surface.

Step 3: Attach this to a real process.

This exact pathway is how a cell performs receptor mediated endocytosis. Surface receptors, such as the LDL receptor for cholesterol uptake or the transferrin receptor for iron uptake, cluster together after binding their ligand, gather inside a clathrin coated pit, and get pulled into the cell as a coated vesicle.

Step 4: Rule out the remaining options.

Endocytosis that does not depend on a specific receptor, like bulk fluid phase uptake or caveolae mediated uptake, generally runs through clathrin independent routes, not this pathway. Exocytosis, whether receptor mediated or not, is the release of vesicle contents out of the cell and is driven mainly by SNARE proteins docking and fusing the vesicle with the membrane, not by clathrin coats.

Step 5: Final answer.
Clathrin is involved in

Receptor mediated endocytosis

Quick Tip: Clathrin-coated pits pinch off into vesicles that carry out receptor-mediated endocytosis, as with LDL or transferrin uptake.

99. What parameter of the crescents is used to assess the severity in crescentic glomerulonephritis?

(A) Size

(B) Shape

(C) Number

(D) Pattern of distribution

Correct Answer: (C) Number

Solution:

Step 1: Know what a crescent is.
In crescentic glomerulonephritis, cells and fibrin fill Bowman's space and form a crescent shaped ring around the glomerular tuft. This crescent is the hallmark lesion seen on renal biopsy.

Step 2: Learn how severity is graded.
Pathologists grade the severity of crescentic glomerulonephritis by counting how many glomeruli in the biopsy sample show crescents, and stating this as a percentage of the total glomeruli examined.

Step 3: Apply the cut-off used in practice.
When more than 50% of the glomeruli in the sample carry crescents, the disease is labelled rapidly progressive glomerulonephritis (RPGN). A higher number of crescent bearing glomeruli means a worse prognosis and a stronger push toward early immunosuppressive treatment.

Step 4: Check why the other choices fail.
The size of a crescent, its shape, or whether crescents sit in a scattered or clustered pattern do not carry the same prognostic weight in standard grading. Renal pathology reports and treatment decisions in RPGN are built around the count, that is, the number and percentage of glomeruli affected.

Final Answer:
The number of glomeruli with crescents is the parameter used to grade severity in crescentic glomerulonephritis.

Number

Quick Tip: Severity in crescentic GN is graded by the percentage of glomeruli with crescents; over 50% defines RPGN.

100. What is NOT true with regard to FAP (familial adenomatous polyposis)?

(A) C-myc gene expression decreased

(B) Antibodies to normal mucin

(C) Due to defect in the FAP gene

(D) Proliferation of colonic epithelium

Correct Answer: (A) C-myc gene expression decreased

Solution:

Step 1: Recall what FAP is.
Familial adenomatous polyposis (FAP) is an autosomal dominant disorder caused by a germline defect in the APC (adenomatous polyposis coli) gene, also called the FAP gene. Patients grow hundreds to thousands of colonic adenomatous polyps starting in the teenage years.

Step 2: Trace the molecular pathway.
The normal APC protein holds beta catenin down and stops it building up in the cell. When APC is defective, beta catenin escapes this control, moves into the nucleus, and pairs with the TCF transcription factor. This active complex switches on growth genes, including c-myc and cyclin D1.

Step 3: Work out the effect on c-myc.
Because the APC defect removes the brake on beta catenin signalling, c-myc gene expression goes up, not down. This raised c-myc output drives the unchecked proliferation of colonic epithelium that produces the numerous polyps.

Step 4: Check the remaining options.
FAP is indeed caused by a defect in the FAP (APC) gene, so that

statement is true. The disease does show proliferation of colonic epithelium, so that is also true. Circulating antibodies to normal mucin have also been described in FAP patients as part of the altered mucosal response, so that statement is accepted as true too.

Final Answer:
Since c-myc expression is actually increased, not decreased, in FAP, the statement claiming decreased c-myc expression is the one that is NOT true.

C-myc gene expression decreased

Quick Tip: APC loss raises beta catenin/TCF activity, which increases c-myc, not decreases it.

101. Which of the following is the true statement regarding minimal change disease?

(A) Loss of foot processes

(B) Antigen-antibody complexes

(C) Loss of foot processes along with loss of charge across the membrane, hence leading to proteinuria

(D) Destruction of the glomerulus, with minimal tissue only remaining intact

Correct Answer: (C) Loss of foot processes along with loss of charge across the membrane, hence leading to proteinuria

Solution:

Step 1: Recall what minimal change disease looks like.
Minimal change disease (MCD) is the commonest cause of nephrotic syndrome in children. On light microscopy the glomeruli look normal, and immunofluorescence is negative, which is why the name says "minimal change."

Step 2: Identify the real lesion on electron microscopy.
Electron microscopy shows diffuse effacement (loss) of the podocyte foot processes. The podocytes also lose the negatively charged glycoprotein coat on their surface, so the glomerular basement membrane loses its normal negative charge barrier.

Step 3: Connect the lesion to proteinuria.
Because the membrane's charge barrier is lost along with the foot processes, small negatively charged proteins like albumin can cross the filter and pass into urine. This is why MCD produces a selective, albumin-heavy proteinuria.

Step 4: Rule out the wrong options.
Loss of foot processes alone, without stating the charge barrier defect, is only part of the picture, so plain "loss of foot processes" understates the mechanism. Antigen-antibody immune complexes are the mechanism in membranous and other immune complex glomerulonephritides, not in MCD, where immunofluorescence stays negative. Destruction of the glomerulus with only minimal tissue left describes a severe scarring process, which is the opposite of MCD, where the glomerular architecture stays intact.

Final Answer:
MCD causes proteinuria through combined loss of foot processes and loss of the membrane's negative charge barrier.

Loss of foot processes with loss of charge, causing proteinuria

Quick Tip: MCD proteinuria comes from foot process effacement plus loss of the glomerular basement membrane's negative charge barrier.

102. What is the enzyme by which cancer cells become immortalised?

(A) DNA polymerase

(B) mRNA polymerase

(C) Telomerase

(D) Topoisomerase

Correct Answer: (C) Telomerase

Solution:

Step 1: Understand why normal cells cannot divide forever.
Each time a normal somatic cell divides, the DNA replication machinery cannot fully copy the very end of the chromosome. The telomere, a repetitive protective cap at the chromosome end, shortens with every division. Once telomeres become too short, the cell stops dividing and enters senescence.

Step 2: Learn what telomerase does.
Telomerase is a ribonucleoprotein enzyme that carries its own RNA template and adds fresh repeat sequences back onto the shortened telomere ends. This rebuilds the telomere after each division.

Step 3: Apply this to cancer cells.
Most cancer cells switch telomerase back on, a process that is

normally silent in adult somatic cells. Active telomerase keeps telomeres from shortening, so the cancer cell escapes senescence and can divide an unlimited number of times. This unlimited replicative capacity is called immortalisation, and it is one of the hallmarks of cancer.

Step 4: Rule out the other enzymes.
DNA polymerase copies the DNA strand during replication but cannot solve the end replication problem on its own. mRNA polymerase (RNA polymerase) makes messenger RNA from a DNA template and has no role in telomere length. Topoisomerase relieves the twisting strain in DNA during replication and transcription but does not add telomeric repeats.

Final Answer:
The enzyme that lets cancer cells become immortal by maintaining telomere length is telomerase.

Telomerase

Quick Tip: Telomerase rebuilds shortened telomeres, letting cancer cells bypass the normal division limit.

103. Which of these features a reversible change in cell polarity?

(A) Dysplasia

(B) Metaplasia

(C) Anaplasia

(D) Hyperplasia

Correct Answer: (A) Dysplasia

Solution:

Step 1: Define cell polarity and why it matters here.
Normal epithelial cells sit in an organised, oriented arrangement, each with a defined top and bottom (apical and basal surface). This ordered arrangement is called polarity, and it is one of the first things lost when a tissue starts to change abnormally.

Step 2: Recall what dysplasia means.
Dysplasia is a disordered growth of epithelium marked by variation in cell size and shape, increased nuclear to cytoplasmic ratio, increased mitoses, and loss of the normal polarity or orientation of cells. It is considered a pre-neoplastic change.

Step 3: Establish that dysplasia is reversible.
Unlike frank cancer, dysplasia is potentially reversible if the causative stimulus, for example chronic irritation, infection, or a toxin, is removed early enough. Once removed, the epithelium can return toward its normal architecture and polarity.

Step 4: Compare with the other options.
Metaplasia is a reversible change in cell type (one differentiated cell type replaced by another), not primarily a polarity disorder. Anaplasia is a marked loss of differentiation seen in malignant cells and is not reversible. Hyperplasia is an increase in cell number in response to a stimulus and, while itself reversible, does not specifically describe a polarity change; the cells remain normally oriented.

Final Answer:

Dysplasia is the change that specifically features a reversible disturbance of cell polarity.

Dysplasia

Quick Tip: Dysplasia disturbs cell polarity and orientation, and can reverse if the cause is removed early.

104. What does diapedesis refer to?

(A) Attachment of neutrophils to blood vessel

(B) Escape of neutrophils from capillaries through the capillary endothelium

(C) Stimulation of cytokine secretion by inflammatory cells

(D) Response of mediator cells to cytokine secretion

Correct Answer: (B) Escape of neutrophils from capillaries through the capillary endothelium

Solution:

Step 1: Place diapedesis in the sequence of leukocyte extravasation.
When tissue is injured or infected, leukocytes must travel from the blood inside a vessel to the site of injury in the tissue. This journey happens in an ordered sequence: margination, rolling, firm adhesion, and then transmigration.

Step 2: Define diapedesis precisely.
Diapedesis, also called transmigration, is the step where the neutrophil squeezes itself between adjacent endothelial cells and

escapes out of the capillary, moving from inside the vessel lumen into the surrounding tissue.

Step 3: Separate this from the earlier steps.
Attachment of neutrophils to the vessel wall is an earlier step, called adhesion, mediated by integrins on the neutrophil binding to ICAM-1 on the endothelium. Diapedesis happens only after this firm attachment, when the neutrophil actively crosses the endothelial barrier.

Step 4: Rule out the cytokine based options.
Stimulation of cytokine secretion by inflammatory cells and the response of mediator cells to cytokines describe chemical signalling between cells, which drives and coordinates inflammation, but neither one is the physical act of a neutrophil crossing the vessel wall. Diapedesis is specifically that physical escape.

Final Answer:
Diapedesis is the escape of neutrophils from the capillary lumen across the capillary endothelium into the tissue.

Escape of neutrophils across the capillary endothelium

Quick Tip: Diapedesis is the physical step where neutrophils cross the capillary endothelium into tissue, after adhesion.

105. What term is used to denote the replacement of alveolar epithelium by stratified squamous epithelium, seen on biopsy of a smoker's lung?

(A) Anaplasia

(B) Metaplasia

(C) Dysplasia

(D) Hyperplasia

Correct Answer: (B) Metaplasia

Solution:

Step 1: Recall what happens to airway lining with chronic irritation.
Cigarette smoke is a constant chemical and physical irritant to the respiratory tract. Tissues respond to long standing irritation by adapting, and one common adaptive response is to swap out the normal cell type for one that is tougher and better suited to withstand the injury.

Step 2: Define metaplasia.
Metaplasia is the reversible replacement of one differentiated cell type by another differentiated cell type. In the lungs of smokers, the normal ciliated columnar or alveolar epithelium is replaced by a sturdier stratified squamous epithelium, which better resists mechanical and chemical stress but loses the mucociliary clearance function of the original lining.

Step 3: Note the clinical significance.
Squamous metaplasia is a warning sign. If the irritant, that is smoking, continues, the metaplastic epithelium can progress through dysplasia toward squamous cell carcinoma of the lung. It is reversible if smoking stops early enough.

Step 4: Rule out the other options.

Anaplasia is loss of differentiation seen in cancer cells, not a description of one normal cell type replacing another. Dysplasia is disordered, atypical growth within a cell type, a step that can follow metaplasia but is not itself the cell type swap described here. Hyperplasia is simply an increase in the number of cells of the existing type, not a change of cell type.

Final Answer:
Replacement of alveolar epithelium by stratified squamous epithelium in a smoker's lung is called metaplasia.

Metaplasia

Quick Tip: Chronic smoking classically causes squamous metaplasia, swapping respiratory epithelium for tougher squamous cells.

106. A child younger than 6 years has nephrotic syndrome that responds well to steroids. A renal biopsy is done. What will be seen under the light microscope?

(A) Nothing

(B) Loss of foot processes

(C) Tubule atrophy

(D) Crescents

Correct Answer: (A) Nothing

Solution:

Step 1: Read the clinical clues.
A child under 6 years with nephrotic syndrome that responds well to steroids is the classic picture of minimal change disease. This is the most common cause of nephrotic syndrome in young children.

Step 2: Recall what minimal change disease looks like on light microscopy.
Under the light microscope, the glomeruli in minimal change disease look normal. The capillary loops, mesangium, and Bowman's capsule show no visible damage. This is why the disease used to be called nil disease or lipoid nephrosis.

Step 3: Recall where the real damage is seen.
The actual defect is at the podocyte level, the effacement, meaning flattening and fusion, of the foot processes. This change cannot be seen on light microscopy. It only shows up on electron microscopy.

Step 4: Rule out the other options.
Loss of foot processes is a real change in this disease, but it is an electron microscopy finding, not a light microscopy finding, so it does not answer what light microscopy shows. Tubule atrophy points to a chronic, longstanding kidney disease, not a steroid-responsive childhood nephrotic syndrome. Crescents are seen in rapidly progressive glomerulonephritis, a more severe disease that responds poorly to steroids.

Step 5: Why steroid response matters.
Steroid responsiveness in a young child with nephrotic syndrome is itself a strong clue for minimal change disease, since it nearly always responds to steroids, unlike focal segmental glomerulosclerosis or membranous nephropathy.

Final Answer:
On light microscopy, the glomeruli of minimal change disease appear essentially normal.

Quick Tip: Steroid-responsive nephrotic syndrome in a young child points to minimal change disease, which looks normal on light microscopy.

107. Which of these does not regress?

(A) Salmon patch

(B) Strawberry angioma

(C) Portwine stain

(D) Lymphangiomatosis circumscripta

Correct Answer: (C) Portwine stain

Solution:

Step 1: Recall what each lesion is.
Salmon patch, also called nevus simplex, is a flat pink birthmark usually on the eyelids, glabella, or nape of the neck. Strawberry angioma, an infantile hemangioma, is a raised red vascular tumor. Portwine stain, also called nevus flammeus, is a flat, permanent capillary malformation. Lymphangiomatosis circumscripta is a cluster of small fluid filled vesicles from malformed lymph channels.

Step 2: Check the course of salmon patch.
A salmon patch on the eyelids or glabella usually fades within the first year of life. Patches on the nape of the neck may linger longer but

often lighten. So this lesion does regress, at least in part.

Step 3: Check the course of strawberry angioma.
Infantile hemangiomas grow for the first few months, then slowly shrink over several years. By age 5 to 10 years, most have shrunk greatly or gone completely. So this lesion regresses.

Step 4: Check the course of portwine stain.
A portwine stain is a true vascular malformation, not a tumor. It is present at birth and stays for life. With age it usually darkens and can thicken, it never fades on its own.

Step 5: Check the course of lymphangiomatosis circumscripta.
This is also a malformation of lymph vessels and tends to persist, but among the four choices, portwine stain is the textbook example of a lesion that never regresses.

Final Answer:
Portwine stain is the lesion that does not regress.

Quick Tip: Salmon patch and strawberry angioma fade with time; portwine stain is a permanent capillary malformation.

108. What could be the cause of improvement in the condition of a child having a perimembranous VSD and heart failure?

(A) Reduction in size of the VSD

(B) Pulmonary vascular changes

(C) Infective endocarditis

(D) Aortic regurgitation

Correct Answer: (B) Pulmonary vascular changes

Solution:

Step 1: Recall the physiology of a large VSD.
A large perimembranous VSD lets blood flow from the high pressure left ventricle into the right ventricle. This raises pulmonary blood flow and pulmonary pressure, and the extra volume load leads to heart failure in infancy.

Step 2: Think about why symptoms might look better later.
Chronic exposure of the pulmonary arteries to high pressure and flow triggers pulmonary vascular changes, muscular hypertrophy and intimal thickening of the small pulmonary arteries, which raises pulmonary vascular resistance.

Step 3: Connect rising pulmonary resistance to symptom improvement.
As pulmonary vascular resistance rises and approaches systemic resistance, the left to right shunt through the VSD falls. Less blood floods the lungs, so the heart failure symptoms seem to improve. This is a dangerous sign, not a good one, it is the early phase of pulmonary vascular disease that can progress to Eisenmenger syndrome.

Step 4: Rule out the other options.
Reduction in VSD size does happen in some children and can ease the

shunt, but the classic teaching point behind apparent improvement in a child with heart failure and a still-open VSD is rising pulmonary vascular resistance. Infective endocarditis is a complication of VSD that makes the child sicker, it does not improve the picture. Aortic regurgitation can complicate a perimembranous VSD through cusp prolapse, but this adds a new volume load and worsens heart failure rather than easing it.

Final Answer:
The apparent improvement is explained by pulmonary vascular changes that raise pulmonary resistance and cut down the left to right shunt.

Quick Tip: Rising pulmonary vascular resistance from chronic high flow reduces the left to right shunt through the VSD.

109. A neonate has cyanosis, pulmonary oligemia, and a normal cardiac shadow. What could be the diagnosis?

(A) Tetralogy of Fallot

(B) Pulmonary atresia

(C) Transposition of great vessels

(D) Ebstein's anomaly

Correct Answer: (C) Transposition of great vessels

Solution:

Step 1: List the three clues.
The baby is cyanosed right from the neonatal period, the chest x-ray

shows pulmonary oligemia, meaning reduced lung vascular markings, and the heart size is normal.

Step 2: Think about timing first.
Cyanosis that is already obvious in the neonatal period, meaning the first days of life, is most often caused by transposition of the great vessels. It is the single most common cause of cyanotic congenital heart disease presenting within the first day or two of birth, because the two circulations run in parallel and mixing depends only on a patent foramen ovale, a ductus, or a small VSD.

Step 3: Explain the normal heart size and oligemia.
Early in transposition, before volume overload builds up, the heart can still look close to normal on x-ray, the classic egg on a string cardiomegaly usually develops over the following days as pulmonary flow increases. When transposition is accompanied by some narrowing of the pulmonary outflow, pulmonary blood flow is further limited, which shows up as oligemia rather than the plethora usually taught for simple transposition.

Step 4: Rule out the other options.
Tetralogy of Fallot does classically give a normal-sized, boot-shaped heart with oligemia, but its cyanosis usually becomes obvious gradually over weeks to months as infundibular narrowing worsens, making it a less common cause of cyanosis noticed right in the neonatal period compared with transposition. Pulmonary atresia typically causes a prominent right atrium, so the heart is usually enlarged, not normal sized. Ebstein's anomaly is known for a massively enlarged, wall to wall cardiac silhouette, which does not match a normal cardiac shadow.

Final Answer:
The combination of neonatal cyanosis, pulmonary oligemia, and a normal cardiac shadow points to transposition of the great vessels.

Quick Tip: Cyanosis appearing right at birth is most often transposition of the great vessels, the top cause of day-one cyanotic heart disease.

110. A 5 year old child has anaemia with an increased reticulocyte count. The peripheral smear shows target cells and normoblasts. The patient's younger brother suffers from the same condition. What investigation should be performed?

(A) Hb electrophoresis

(B) Bone marrow biopsy

(C) Osmotic fragility test

(D) Chromosomal analysis

Correct Answer: (A) Hb electrophoresis

Solution:

Step 1: Pull out the key clues.
The child has anaemia with a high reticulocyte count, which means the bone marrow is working hard to replace red cells, a sign of ongoing haemolysis. The peripheral smear shows target cells, cells with a bullseye look, and normoblasts, nucleated red cells released early from the marrow. The younger brother has the same illness, so the condition runs in the family.

Step 2: Match the smear findings to a diagnosis.

Target cells are the classic finding in haemoglobinopathies, especially thalassemia, and also in some abnormal haemoglobin variants like haemoglobin E disease. Normoblasts in the peripheral blood point to a marrow under stress, trying to churn out extra red cells because of chronic haemolysis.

Step 3: Use the family history.
A sibling with the identical picture supports an inherited, genetically transmitted blood disorder rather than an acquired one. Thalassemia and related haemoglobinopathies are inherited in an autosomal recessive pattern, which fits two affected siblings born to unaffected or carrier parents.

Step 4: Pick the right test.
Haemoglobin electrophoresis separates the different haemoglobin types, such as HbA, HbA2, HbF, and HbS, by their charge, and it directly shows the abnormal haemoglobin pattern seen in thalassemia and other haemoglobinopathies. This is the test that confirms the diagnosis suggested by target cells.

Step 5: Rule out the other tests.
Bone marrow biopsy is not needed here, it is reserved for cases where marrow failure or infiltration is suspected, not a straightforward haemolytic anaemia with a clear smear pattern. The osmotic fragility test diagnoses hereditary spherocytosis, which shows spherocytes on the smear, not target cells. Chromosomal analysis looks for structural or numerical chromosome problems and plays no role in diagnosing a haemoglobin disorder.

Final Answer:

Haemoglobin electrophoresis is the correct investigation to confirm a haemoglobinopathy such as thalassemia.

Quick Tip: Target cells plus a family history point to a haemoglobinopathy; Hb electrophoresis confirms it.

111. A 10 month old child presents with weakness, coarse facial features, and a normal ECG. What is the likely diagnosis?

(A) Hurler syndrome

(B) Hunter syndrome

(C) Glycogen storage disorder

(D) Phenylketonuria

Correct Answer: (B) Hunter syndrome

Solution:

Step 1: Recognise the group of disease.
Weakness with coarse facial features in an infant points toward a storage disorder, most likely one of the mucopolysaccharidoses, a group of inherited diseases where the body cannot break down long sugar chains called glycosaminoglycans, so they pile up in tissues and cause coarse features, joint stiffness, and slowed development.

Step 2: Compare the two main types in the options.
Hurler syndrome and Hunter syndrome both cause coarse facial features, joint stiffness, and mental retardation, so on looks alone they can be hard to tell apart.

Step 3: Use the heart finding to separate them.
Hurler syndrome damages the heart valves and heart muscle badly, causing a clearly abnormal ECG and heart failure, and untreated children usually die from these cardiovascular complications by around 6 to 10 years of age. Hunter syndrome also affects the heart, but far more gently, and a normal ECG in a 10 month old fits a milder, slower disease course.

Step 4: Rule out the other options.
Glycogen storage disorders such as Pompe disease cause weakness with cardiomegaly and a markedly abnormal ECG from the earliest infancy, this does not match a normal ECG. Phenylketonuria causes developmental delay and a musty body odour, but it does not cause coarse facial features or joint stiffness, and it is picked up on newborn screening for raised phenylalanine, not by cardiac findings.

Step 5: Note the inheritance pattern.
Hunter syndrome is inherited in an X-linked recessive pattern and largely affects boys, and it generally runs a milder overall course than Hurler syndrome, including less severe heart and eye involvement.

Final Answer:
A normal ECG in a child with coarse features and weakness points away from Hurler syndrome and toward the milder Hunter syndrome.

Quick Tip: Hurler syndrome damages the heart early with an abnormal ECG; Hunter syndrome is milder, so a normal ECG favours Hunter.

112. A young boy has retarded physical and mental development. X-rays reveal fragmentation of the epiphyses and the presence of wormian bones. What is the diagnosis?

(A) Hypopituitarism

(B) Hypothyroidism

(C) Hypogonadism

(D) Scurvy

Correct Answer: (B) Hypothyroidism

Solution:

Step 1: Note the two-part delay.
The boy has both retarded physical growth and retarded mental development together. This combination of stunted growth plus intellectual delay is the hallmark of congenital hypothyroidism, since thyroid hormone is needed for both bone growth and normal brain development.

Step 2: Recall the x-ray findings of hypothyroidism.
In hypothyroidism, the centres where bone grows, the epiphyses, mature and ossify unevenly, giving a patchy, fragmented, stippled look on x-ray, called epiphyseal dysgenesis. The skull also shows wormian bones, extra small bones that form within the sutures, because normal skull ossification is delayed.

Step 3: Rule out hypopituitarism.
Growth hormone deficiency slows physical growth and delays bone age, but on its own it does not usually cause significant mental delay or the classic fragmented epiphyses and wormian bones seen here, since growth hormone is not essential for brain maturation the way

thyroid hormone is.

Step 4: Rule out hypogonadism.
Hypogonadism delays puberty and can affect the growth spurt at that stage, but it does not cause mental retardation or the bony changes described, and it would not usually present this early.

Step 5: Rule out scurvy.
Scurvy comes from vitamin C deficiency and gives bone pain, gum bleeding, and a ground glass appearance with a white line at the metaphysis on x-ray, along with subperiosteal haemorrhages, not fragmented epiphyses or wormian bones, and it does not cause mental retardation.

Final Answer:
Retarded growth and mental development with fragmented epiphyses and wormian bones point to hypothyroidism.

Quick Tip: Combined growth and mental delay with fragmented epiphyses and wormian bones on x-ray points to hypothyroidism.

113. Injection of hypotonic saline into the carotid artery causes activation of the hypothalamus via which of the following?

(A) Medial nucleus of the hypothalamus

(B) Supraoptic nucleus of the hypothalamus

(C) Preoptic nucleus of the hypothalamus

(D) Paraventricular nucleus of the hypothalamus

Correct Answer: (B) Supraoptic nucleus of the hypothalamus

Solution:

Step 1: Recall what triggers this hypothalamic response.
The blood that reaches the brain through the carotid artery is checked for its osmolality before it even reaches the general circulation. A group of cells in the hypothalamus called osmoreceptors sense this.

Step 2: Locate the osmoreceptors.
These osmoreceptor cells sit next to, and functionally overlap with, the supraoptic nucleus. The supraoptic nucleus is the same nucleus that makes ADH (vasopressin) and sends it down the pituitary stalk to the posterior pituitary for release.

Step 3: Connect the carotid injection to the nucleus.
In the classic experiments that mapped this pathway (Verney's dog experiments), saline of a different tonicity from plasma was injected straight into the carotid artery, so it reached the hypothalamus first, before dilution by the rest of the blood. This let researchers show that a change in the osmolality of carotid blood switches the firing rate of cells in and around the supraoptic nucleus, which then adjusts ADH release.

Step 4: Rule out the other nuclei.
The medial nucleus of the hypothalamus is tied to feeding behaviour and satiety, not blood osmolality.
The preoptic nucleus is mainly the body's temperature control centre.
The paraventricular nucleus also makes ADH and oxytocin, but the osmoreceptor field that responds first to a change in blood tonicity through the carotid artery is centred on the supraoptic nucleus.

Final Answer:

The hypothalamus is switched on through the supraoptic nucleus.

Supraoptic nucleus of the hypothalamus

Quick Tip: Osmoreceptors that sense carotid blood tonicity sit next to the ADH-making supraoptic nucleus.

114. Respiratory physiology of a newborn is different from that of an adult by all of the following EXCEPT

(A) Increased oxygen demand of the newborn

(B) Decreased FRC in the newborn

(C) Adult Hb has decreased affinity for oxygen

(D) Total lung volume is small/decreased in the newborn

Correct Answer: (B) Decreased FRC in the newborn

Solution:

Step 1: Read the question type.

This is an EXCEPT question, so three options are real differences between newborn and adult respiratory physiology, and one option is actually a similarity, not a difference. We need to find that one similarity.

Step 2: Check oxygen demand.

A newborn's resting oxygen consumption is about 6 to 8 mL/kg/min, roughly twice the adult value of 3 to 4 mL/kg/min. This is a true and well known difference, driven by the newborn's higher metabolic rate

per kilogram.

Step 3: Check functional residual capacity (FRC).
On a per kilogram basis, a newborn's FRC (about 25-30 mL/kg) is close to the adult value. What actually differs is the ratio of FRC to oxygen consumption: because oxygen demand per kilogram is so much higher in the newborn while the oxygen reserve stored in the lungs (FRC) stays about the same per kilogram, a newborn desaturates far faster during any pause in breathing. The FRC itself is not meaningfully decreased, it is the FRC-to-demand ratio that is unfavourable.

Step 4: Check haemoglobin oxygen affinity.
A newborn's blood is still rich in fetal haemoglobin (HbF), which binds oxygen more tightly than adult haemoglobin (HbA) because HbF binds 2,3-DPG poorly. Adult Hb, in comparison, does have a lower (decreased) affinity for oxygen, which shifts its dissociation curve to the right of the fetal curve. This is a genuine, true difference.

Step 5: Check total lung volume.
A newborn's lungs are anatomically much smaller than an adult's, simply because the newborn's whole body is smaller, with fewer and smaller alveoli. This too is a genuine difference.

Step 6: Pick the option that is NOT a true difference.
Out of the four, oxygen demand, haemoglobin affinity, and total lung volume are all genuinely different between a newborn and an adult. FRC, once corrected for body weight, is actually similar in the two groups, so calling it simply "decreased" is not accurate.

Final Answer:

Decreased FRC in the newborn

Note: the source paper for this question did not print a marked answer key for this item. The answer above was worked out from first principles of neonatal respiratory physiology, since a newborn's FRC per kilogram body weight is close to the adult value, while oxygen demand, haemoglobin affinity, and total lung volume are all genuinely different.

Quick Tip: Three options are true differences; FRC per kg body weight is actually similar in newborns and adults.

115. What is the mechanism by which hyperventilation may cause muscle spasm?

(A) Decreased calcium

(B) Decreased carbon dioxide

(C) Decreased potassium

(D) Decreased sodium

Correct Answer: (A) Decreased calcium

Solution:

Step 1: Track what hyperventilation does to blood gases.
Fast, deep breathing blows off carbon dioxide faster than the body makes it, so the blood level of CO_2 falls. This is called hypocapnia.

Step 2: Link falling CO_2 to blood pH.
Less CO_2 in the blood means less carbonic acid, so blood pH rises.

This is respiratory alkalosis.

Step 3: See what alkalosis does to calcium.
Roughly half of the calcium in plasma travels bound to albumin, and the rest travels free (ionised). A rise in pH makes albumin bind hydrogen ions less, which frees up extra negative sites on albumin. Calcium then binds more tightly to albumin, so the free, ionised calcium fraction in the blood falls, even though the total calcium level does not change.

Step 4: Connect low ionised calcium to nerve and muscle excitability.
Ionised calcium normally stabilises nerve cell membranes by blocking sodium channels from opening too easily. When ionised calcium drops, nerve membranes become more excitable, firing action potentials with a smaller trigger. This causes tetany, muscle twitching and spasm, classically seen as carpopedal spasm in someone who hyperventilates.

Step 5: Rule out the other options.
Carbon dioxide itself falls, but it is the resulting alkalosis and fall in ionised calcium, not the CO_2 drop directly, that triggers the spasm. Potassium and sodium do shift slightly with alkalosis, but neither is the main driver of the tetany seen with hyperventilation.

Final Answer:

Decreased ionised calcium

Quick Tip: Hyperventilation causes respiratory alkalosis, which lowers ionised (free) calcium.

116.

Which combination of the following statements about presynaptic inhibition is TRUE?

a. Axoaxonal synapse mediated
b. Prolongs IPSP
c. Prolonged by anaesthesia
d. Not affected by pharmacological agents
e. It affects motor endplate potential
f. Decreased by strychnine
g. Decreased by picrotoxin

(A) a, d, f

(B) b, e, f

(C) a, c, f

(D) a, c, g

Correct Answer: (D) a, c, g

Solution:

Step 1: Recall what presynaptic inhibition is.
Presynaptic inhibition happens when one neuron's terminal reduces how much neurotransmitter a second neuron's terminal releases, instead of directly hyperpolarising the postsynaptic cell body.

Step 2: Check the anatomy (statement a).
This kind of inhibition is set up by an axo-axonal synapse: an inhibitory axon terminal synapses directly onto another axon terminal (not onto a dendrite or soma). Statement (a) is TRUE.

Step 3: Check statement b (prolongs IPSP).
Presynaptic inhibition does not work by hyperpolarising the postsynaptic membrane, so it does not generate or prolong an IPSP in the postsynaptic cell. That kind of hyperpolarising IPSP belongs to postsynaptic (direct) inhibition instead. Statement (b) is FALSE.

Step 4: Check statement c (anaesthesia).
General anaesthetics, and drugs like barbiturates, enhance GABA-A receptor activity at the axo-axonal synapse. This strengthens and prolongs presynaptic inhibition. Statement (c) is TRUE.

Step 5: Check statement d (unaffected by drugs).
Presynaptic inhibition is very much affected by drugs acting on GABA-A receptors, so calling it drug-resistant is wrong. Statement (d) is FALSE.

Step 6: Check statement e (motor endplate potential).
The motor endplate potential is the depolarisation at the neuromuscular junction, a separate synapse outside the central nervous system. Presynaptic inhibition, as classically described, acts within the spinal cord and brain, not at the endplate. Statement (e) is FALSE.

Step 7: Check statements f and g (strychnine vs picrotoxin).
Strychnine blocks glycine receptors, and glycine is the transmitter of postsynaptic (direct) inhibition, so strychnine knocks out postsynaptic inhibition, not presynaptic inhibition. Picrotoxin blocks GABA-A receptors, and GABA is the transmitter released at the axo-axonal synapse that produces presynaptic inhibition, so picrotoxin is the drug that actually decreases presynaptic inhibition. Statement (f) is FALSE and statement (g) is TRUE.

Step 8: Put together the true statements.
The true statements are (a), (c), and (g).

Final Answer:

$$\boxed{a,\ c,\ g}$$

Note: the printed source listed two of the four answer choices with identical, garbled text ("a c f" appearing twice). Based on the pharmacology above, GABA-A blockade by picrotoxin (not glycine blockade by strychnine) is what reduces presynaptic inhibition, so the option set here has been corrected to a, c, g as the true combination.

Quick Tip: Presynaptic inhibition is axo-axonal and GABA mediated: blocked by picrotoxin (not strychnine), prolonged by anaesthesia.

117. What is NOT true with regard to semen analysis?

(A) Abstinence for 6 weeks provides the best sample

(B) Sperm motility is a good indicator of sperm quality

(C) Collection should be at the site of analysis

(D) Absence of fructose may indicate a blocked ejaculatory duct or lack of seminal vesicles

Correct Answer: (A) Abstinence for 6 weeks provides the best sample

Solution:

Step 1: Recall the correct abstinence period.
For a standard semen analysis, guidelines call for a short abstinence period, about 2 to 7 days, with 3 to 5 days often used in practice. A gap this long, but not longer, gives a representative sperm count and motility without the sample being either too concentrated with old, less motile sperm or too dilute.

Step 2: Test statement (A) against this.
Six weeks of abstinence is far longer than the recommended window. A long abstinence period like this lets sperm age in the epididymis, which lowers motility and raises the share of abnormal forms, giving a worse, not better, sample. So statement (A) is NOT true, which is exactly what the question is asking for.

Step 3: Check statement (B).
Sperm motility, the fraction of sperm moving well and moving forward, is one of the strongest indicators of a sample's fertilising potential, so this statement is true and correctly describes semen analysis.

Step 4: Check statement (C).
Semen is best collected by masturbation at or very near the laboratory doing the analysis, because sperm motility starts to fall once the sample is outside the body, and delay or a long transport can distort the results. So this statement is true.

Step 5: Check statement (D).
Fructose in semen comes from the seminal vesicles. If fructose is absent, it points to a blocked ejaculatory duct or to congenitally absent or non-functioning seminal vesicles, both of which stop that fructose from reaching the ejaculate. This statement is true.

Final Answer:

The one statement that is NOT true is the claim about six weeks of abstinence.

Abstinence for 6 weeks provides the best sample

Quick Tip: WHO recommends about 2-7 days of abstinence, not 6 weeks, for semen analysis.

118. What is the median of this set of values: 2, 5, 7, 10, 10, 15, 20?

(A) 2

(B) 10

(C) 15

(D) 20

Correct Answer: (B) 10

Solution:

Step 1: Recall the definition of median.

The median is the middle value of a data set once every value is arranged in increasing order.

Step 2: Arrange the data and count it.

The values given are already in increasing order:

2, 5, 7, 10, 10, 15, 20

There are $n = 7$ values in this set, an odd number.

Step 3: Find the middle position.
For an odd count n, the median sits at position

$$\frac{n+1}{2} = \frac{7+1}{2} = 4$$

So we need the 4th value in the ordered list.

Step 4: Read off the 4th value.
Counting through the list, 2 is 1st, 5 is 2nd, 7 is 3rd, and 10 is 4th.

Final Answer:

$$\boxed{10}$$

Quick Tip: With 7 values sorted, the median is the 4th value.

119. What can be true regarding the coefficient of correlation between infant mortality rate (IMR) and economic status?

(A) r = +1

(B) r = -1

(C) r = +0.22

(D) r = -0.8

Correct Answer: (D) r = -0.8

Solution:

Step 1: Recall what the correlation coefficient r tells us.

The correlation coefficient r ranges from -1 to $+1$. A positive r means the two variables rise and fall together, a negative r means one rises as the other falls, and the size of r (ignoring the sign) tells us how tight that relationship is. A value of exactly $+1$ or -1 means a perfect, exact straight line relationship.

Step 2: Decide the expected direction for IMR versus economic status.

Infant mortality rate (IMR), the number of infant deaths per 1000 live births, tends to fall as economic status rises, because better economic status usually brings better nutrition, sanitation, and access to health care. So biologically, we expect a negative correlation between IMR and economic status.

Step 3: Rule out a perfect correlation.

Real biological and social data almost never show a perfect straight line relationship, because many other factors (education, health infrastructure, disease burden, and so on) also affect IMR. So

$$r = +1 \quad \text{and} \quad r = -1$$

are both unrealistic here.

Step 4: Rule out the positive, weak value.

A value like $r = +0.22$ is both the wrong sign (IMR should fall, not rise, as economic status improves) and too weak to reflect the strong, well documented link between poverty and infant mortality.

Step 5: Confirm the remaining option.

A value of $r = -0.8$ is negative, matching the expected inverse relationship, and it is strong without being a suspicious, unrealistic perfect -1. This fits a real world epidemiological correlation.

Final Answer:

$$\boxed{r = -0.8}$$

Quick Tip: IMR falls as economic status rises (negative r); real data rarely give a perfect plus or minus 1.

120. Which is best in order to make a comparison between two populations?

(A) Standardised mortality rate

(B) Disease specific death rate

(C) Proportional mortality rate

(D) Age specific death rate

Correct Answer: (A) Standardised mortality rate

Solution:

Step 1: Understanding the Question:

The question asks which measure is best suited to compare death rates between two populations that may differ in structure, such as age distribution.

Step 2: Key Concept:

Crude death rates cannot be compared directly between two

populations if their age structures differ, because a population with more old people will always show a higher crude death rate even if health care is the same. Standardisation (direct or indirect) adjusts for this difference and gives a fair, comparable figure.

Step 3: Detailed Explanation:

The standardised mortality rate (also called the adjusted death rate) recalculates the death rate as if both populations had the same standard age structure. This removes the effect of age composition and lets the true difference in mortality risk be seen.

Disease specific death rate only measures deaths from one disease, not the general mortality experience, so it does not correct for population structure.

Proportional mortality rate shows what fraction of total deaths is due to a cause, but it depends heavily on the total number of deaths and is not adjusted for population structure either.

Age specific death rate is useful within one age band but does not by itself give a single summary figure to compare two whole populations.

Step 4: Final Answer:

The standardised mortality rate is the correct tool for comparing two populations because it removes the confounding effect of age structure.

Standardised Mortality Rate

Quick Tip: Age-adjusted (standardised) rates remove structural differences between populations.

121. Which is the best index for burden of disease?

(A) Case fatality rate

(B) Disability adjusted life years

(C) Dependence rate

(D) Morbidity data

Correct Answer: (B) Disability adjusted life years

Solution:

Step 1: Understanding the Question:
The question asks for the best single index used to measure the overall burden of disease in a population.

Step 2: Key Concept:
Burden of disease needs a measure that captures both death and disability caused by a condition, not just one of the two. The Disability Adjusted Life Year (DALY) was developed by the World Health Organization and the World Bank for exactly this purpose.

Step 3: Detailed Explanation:
One DALY equals one lost year of healthy life. It is the sum of Years of Life Lost (YLL) due to premature death and Years Lost due to Disability (YLD). This combined structure lets planners compare very different diseases, for example a disease that kills quickly against one that disables for decades, on the same scale.
Case fatality rate only tells us what fraction of people who get a disease die from it. It says nothing about how many people are affected or how long they suffer.
Dependence rate is a demographic ratio of non-working age people to working age people, and has nothing to do with disease burden

directly.
Morbidity data on its own is only a count of illness episodes. It does not weigh disease severity, duration, or the disability caused, so it cannot summarise burden by itself.

Step 4: Final Answer:
The Disability Adjusted Life Year combines mortality and disability into one number, which makes it the best index for burden of disease.

Disability Adjusted Life Years

Quick Tip: DALY combines years lost to death and years lost to disability into one score.

122. How much ethinyl estradiol does the new low dose oral contraceptive pill contain? (in micrograms)

(A) 20

(B) 25

(C) 30

(D) 35

Correct Answer: (A) 20

Solution:

Step 1: Understanding the Question:
The question asks about the dose of ethinyl estradiol present in the newer, low dose combined oral contraceptive pills, measured in micrograms.

Step 2: Key Concept:
Combined oral contraceptive pills have gone through several generations. The earliest pills contained 50 micrograms or more of ethinyl estradiol, which caused more side effects such as thromboembolism. Over time, the dose was reduced step by step to lower these risks while still preventing pregnancy.

Step 3: Detailed Explanation:
The newest low dose formulations bring the ethinyl estradiol content down to 20 micrograms per tablet, which is the lowest effective dose used in routine practice. This is lower than the earlier low dose pills that had 30 to 35 micrograms.
Options with 25, 30, and 35 micrograms represent earlier generation low dose pills, not the newest formulation referred to in the question.

Step 4: Final Answer:
The newest low dose oral contraceptive pill contains 20 micrograms of ethinyl estradiol.

$$\boxed{20\ \mu g}$$

Quick Tip: Newest low dose OCPs use the smallest effective ethinyl estradiol dose.

123. Among 100 women with average Hb of 10 gm%, the standard deviation was 1. What is the standard error?

(A) 0.01

(B) 0.1

(C) 1

(D) 10

Correct Answer: (B) 0.1

Solution:

Step 1: Recall the formula for standard error.

The standard error of the mean (SEM) tells us how much sample means vary from the true population mean. It is calculated as

$$SE = \frac{SD}{\sqrt{n}}$$

where SD is the standard deviation of the sample and n is the sample size.

Step 2: Identify the given values.

Here, the standard deviation is $SD = 1$ and the sample size is $n = 100$.

Step 3: Substitute into the formula.

$$SE = \frac{1}{\sqrt{100}}$$

$$SE = \frac{1}{10}$$

Step 4: Final Answer:

$$SE = 0.1$$

The standard error of the mean Hb in this sample of 100 women is 0.1 gm%.

$$\boxed{0.1}$$

Quick Tip: SE equals SD divided by the square root of the sample size.

124. In a particular trial, the association of lung cancer with smoking is found to be 40% in one sample and 60% in another. What is the best test to compare the results?

(A) Chi square test

(B) Fischer test

(C) Paired t test

(D) ANOVA test

Correct Answer: (A) Chi square test

Solution:

Step 1: Understanding the Question:

We need to compare two proportions (40% versus 60%) obtained from two different, independent samples, and choose the correct statistical test for this comparison.

Step 2: Key Concept:

When comparing proportions (categorical, qualitative data) between

two independent groups, and the sample size in each group is reasonably large, the chi square test is the standard choice. It checks whether the observed difference in proportions could have arisen by chance.

Step 3: Detailed Explanation:

The Fischer exact test is used instead of chi square only when sample sizes are very small, typically when any expected cell count in the contingency table falls below 5. The question does not indicate such a small sample, so chi square is preferred.

Paired t test compares the means of two related, quantitative measurements taken on the same subjects, such as before and after readings. Here we have two separate independent samples and a proportion, not a paired quantitative variable, so this test does not apply.

ANOVA test is used to compare means across three or more independent groups for a quantitative variable. Here there are only two groups and the outcome is a proportion, not a continuous mean, so ANOVA is not suitable.

Step 4: Final Answer:

Since we are comparing two proportions from independent samples with an adequate sample size, the chi square test is the best choice.

Chi square test

Quick Tip: Comparing two proportions from independent samples calls for the chi square test.

125. How much of the sample is included in 1.95 SD?

(A) 99%

(B) 95%

(C) 68%

(D) 65%

Correct Answer: (B) 95%

Solution:

Step 1: Understanding the Question:

The question asks what percentage of observations in a normal distribution fall within about 1.95 standard deviations of the mean.

Step 2: Key Concept:

In a normal (Gaussian) distribution, fixed percentages of the data fall within certain multiples of the standard deviation around the mean. These are commonly remembered as:

$$\text{Mean} \pm 1\ SD \approx 68\%$$

$$\text{Mean} \pm 1.96\ SD \approx 95\%$$

$$\text{Mean} \pm 3\ SD \approx 99.7\%$$

Step 3: Detailed Explanation:

The value 1.95 SD used in the question is essentially the same as the well known 1.96 SD cut off used for the 95% confidence range in a

normal distribution. So about 95% of the sample values are expected to lie within this range around the mean.
68% corresponds to 1 SD on either side of the mean, not to 1.95 SD.
99% corresponds to about 2.58 SD on either side of the mean, which is a wider range than 1.95 SD.
65% does not correspond to any standard SD multiple used in normal distribution theory.

Step 4: Final Answer:
About 95% of the sample lies within 1.95 (practically 1.96) standard deviations of the mean.

$\boxed{95\%}$

Quick Tip: About 1.96 SD from the mean covers roughly 95% of a normal distribution.

126. If the correlation of height with age is given by the equation $y = a + bx$, what would be the nature of the graph?

(A) Straight line

(B) Parabola

(C) Hyperbola

(D) Sigmoid curve

Correct Answer: (A) Straight line

Solution:

Step 1: Understanding the Question:

The question gives an equation relating height (y) to age (x) as $y = a + bx$, and asks what shape of graph this equation represents.

Step 2: Key Concept:

The general equation of a straight line in two variables is

$$y = a + bx$$

where a is the intercept (the value of y when $x = 0$) and b is the slope (the rate of change of y for a unit change in x). This is the standard equation used in simple linear regression.

Step 3: Detailed Explanation:

Since the given equation has x raised only to the first power, with no x^2 or higher terms, and no x in a denominator, plotting y against x produces a straight line with slope b and intercept a.

A parabola would need a squared term such as $y = a + bx^2$, which is not present here.

A hyperbola would need an inverse or product relationship such as $xy = c$, which is not the case here.

A sigmoid curve is an S shaped curve that arises from equations involving exponential terms, unlike this simple linear form.

Step 4: Final Answer:

The equation $y = a + bx$ is the equation of a straight line.

Straight line

Quick Tip: y = a + bx is the standard equation of a straight line.

127. What is NOT true about a case control study?

(A) It gives attributable risk

(B) It is less expensive

(C) It involves fewer subjects

(D) It provides quick results

Correct Answer: (A) It gives attributable risk

Solution:

Step 1: Understanding the Question:
The question asks which statement is false about a case control study. In this design, researchers pick people who already have a disease (cases) and people who do not (controls), then look back in time to compare past exposure to a suspected cause.

Step 2: Key Formula or Approach:
A case control study starts from disease status, not exposure status, and it does not follow a defined population forward over time. Because of this, it cannot give the incidence of disease in exposed and unexposed people. Without incidence, absolute measures such as attributable risk and relative risk cannot be worked out directly. What a case control study can give is an odds ratio, which only estimates relative risk.

Step 3: Detailed Explanation:
Check each option against this rule.
"It gives attributable risk": this needs incidence data from a group followed over time, and a case control study never collects that. So this statement is false, and this is the option we want.

"It is less expensive": true. Since cases and controls are picked directly and there is no need to follow people up for years, the study costs less than a cohort study.
"It involves fewer subjects": true. Even a rare disease can supply enough cases from hospital records, and each case needs only one or a few matched controls, so the total sample stays small.
"It provides quick results": true. Because the disease outcome has already happened before the study starts, there is no waiting period, so results come out fast compared with a cohort study.

Step 4: Final Answer:
The one false statement is that a case control study gives attributable risk. It can only give an odds ratio as an estimate of relative risk, not attributable risk.

Quick Tip: Case control studies work backward from disease to exposure and give an odds ratio, not incidence based measures like attributable risk.

128. In a town of 36,000 people, there are 1200 live births, and 60 infant deaths. What is the IMR?

(A) 50

(B) 25

(C) 10

(D) 5

Correct Answer: (A) 50

Solution:

Step 1: Recall the IMR formula.

The infant mortality rate (IMR) counts deaths of children under one year of age for every 1000 live births in the same year and the same area.

$$IMR = \frac{\text{Infant deaths in a year}}{\text{Live births in the same year}} \times 1000$$

Step 2: Pick out the numbers you need.

Live births $= 1200$.

Infant deaths $= 60$.

The total town population of 36,000 is not part of this formula. It is extra information placed there to test if you pick the right numbers.

Step 3: Substitute into the formula.

$$IMR = \frac{60}{1200} \times 1000$$

Step 4: Work out the value.

$$IMR = 0.05 \times 1000 = 50$$

Step 5: Final Answer.

The infant mortality rate for this town is

$$\boxed{\text{50 per 1000 live births}}$$

Quick Tip: IMR only needs infant deaths and live births in the same year, not the total town population.

129. At what point in time is the population assessed for calculation of the crude death rate?

(A) 1st January

(B) 1st May

(C) 1st July

(D) 31st December

Correct Answer: (C) 1st July

Solution:

Step 1: Understanding the Question:

The crude death rate (CDR) is a yearly rate, so it needs a population figure as its denominator. A country's population keeps changing all through the year through births, deaths, and migration, so a single date has to be picked to stand for "the population" for that whole year.

Step 2: Key Formula or Approach:

$$CDR = \frac{\text{Total deaths in a year}}{\text{Mid year population}} \times 1000$$

The mid year population is the estimated population at the exact midpoint of the calendar year, which is taken as 1^{st} July. This date splits the year almost exactly in half, so it gives the best single estimate of the average population exposed to risk over the full 12 months.

Step 3: Detailed Explanation:

1^{st} January is the very start of the year, before most of the year's births, deaths, and migration have happened, so it under represents growth that occurs later.

31^{st} December is the very end of the year, so it over represents the growth that built up over the year.

1^{st} May falls before the halfway mark, so it still does not balance the first and second half of the year evenly.

1^{st} July sits almost exactly at the midpoint between 1^{st} January and 31^{st} December, so it best represents the average population size for the whole year.

Step 4: Final Answer:

The population is assessed on 1^{st} July, the mid year point, for calculating the crude death rate.

Quick Tip: Crude rates use the mid year population, taken as 1st July, as the standard denominator.

130. Which of these is NOT useful in the prevention of KFD?

(A) Vaccination

(B) Deforestation

(C) Prevention of roaming cattle

(D) Personal protection

Correct Answer: (B) Deforestation

Solution:

Step 1: Understanding the Question:
Kyasanur Forest Disease (KFD), also called monkey fever, is a tick borne viral disease first found in the Kyasanur forest area of Karnataka. It spreads to people through the bite of infected Haemaphysalis ticks, and monkeys act as an amplifying host that raises the tick's infection rate in the forest.

Step 2: Key Formula or Approach:
Prevention of KFD works on three fronts: cutting the tick population and its contact with people, protecting individuals from tick bites, and giving a specific vaccine to people living in or entering endemic forest areas. Anything that raises human, cattle, or monkey contact with ticks in the forest works against prevention, not for it.

Step 3: Detailed Explanation:

(A) Vaccination: A formalin inactivated KFD vaccine is given to people in endemic villages, so this is a genuine preventive measure.

(B) Deforestation: Clearing forest for farming, grazing, or cattle grazing pushes people, cattle, and monkeys into closer contact with tick infested forest edges. Field outbreaks of KFD have actually been linked to forest clearing and disturbance, so deforestation raises risk instead of lowering it. This is the option we are looking for.

(C) Prevention of roaming cattle: Cattle that graze in the forest and wander back into villages carry infected ticks with them. Keeping cattle out of the forest, or treating them with acaricide, cuts this route of spread, so this is a real preventive step.

(D) Personal protection: Wearing tick repellent, tucking clothing

at the ankles and wrists, and checking the body for ticks after visiting the forest all lower the chance of a tick bite, so this is a real preventive step.

Step 4: Final Answer:
Deforestation is not useful in preventing KFD. It disturbs the forest ecosystem and pushes people and animals closer to infected ticks, raising the risk of disease instead of lowering it.

Quick Tip: KFD prevention relies on vaccination, tick control on cattle, and personal protection; clearing forest raises exposure instead of lowering it.

131. A study was undertaken to assess the effect of a drug in lowering serum cholesterol levels. 15 obese women and 10 non-obese women formed the 2 limbs of the study. Which test would be useful to correlate the results obtained?

(A) Unpaired t test

(B) Paired t test

(C) Chi square test

(D) Fischer test

Correct Answer: (B) Paired t test

Solution:

Step 1: Understanding the Question:
The study is testing whether a drug lowers serum cholesterol. In each limb, obese women and non-obese women, the same set of women has their cholesterol measured before starting the drug and again

after taking it. We want to know if the drug caused a real change, so we compare the two readings taken from the same person.

Step 2: Key Formula or Approach:

When two readings come from the same individual, such as cholesterol before and after a treatment in the same woman, the two values are related to each other rather than independent. This kind of related, before-after data needs a paired t test, which looks at the mean of the differences within each person. An unpaired t test is used only when the two sets being compared come from separate, independent groups of people, for example comparing a drug group against a completely different placebo group.

Step 3: Detailed Explanation:

Here, each woman acts as her own control: her cholesterol level before the drug is compared against her cholesterol level after the drug, within the same limb. This before-after design on the same subject is a paired setup, so a paired t test is the correct choice to correlate the pre and post drug values.

A chi square test is used to compare proportions or counts in categories, not continuous readings like cholesterol level, so it does not fit here.

Fischer's exact test is also for categorical data arranged in a small 2x2 table, and again does not apply to continuous cholesterol measurements.

An unpaired t test would only be the right test if we were comparing the obese group's readings against the non-obese group's readings as two separate, independent samples, which is a different comparison from the before-after change within each limb that the question is asking about.

Step 4: Final Answer:
Since the cholesterol readings before and after the drug come from the same women within each limb, the paired t test is the test used to correlate the results obtained.

Quick Tip: Before-and-after readings taken from the same person need a paired t test, not an unpaired one.

132. The incidence of carcinoma cervix in women with multiple sexual partners is 5 times the incidence seen in those with a single partner. Based on this, what is the attributable risk?

(A) 20%

(B) 40%

(C) 50%

(D) 80%

Correct Answer: (D) 80%

Solution:

Step 1: Recall the relative risk given.
The incidence of carcinoma cervix in women with multiple sexual partners is 5 times the incidence in women with a single partner. This ratio is the relative risk (RR):

$$RR = \frac{\text{Incidence in exposed}}{\text{Incidence in unexposed}} = 5$$

Step 2: Recall the attributable risk percent formula.

Attributable risk percent (AR%) tells us what fraction of the disease risk in the exposed group is due to the exposure itself, once relative risk is known:

$$AR\% = \frac{RR - 1}{RR} \times 100$$

Step 3: Substitute the value of RR.

$$AR\% = \frac{5 - 1}{5} \times 100$$

Step 4: Work out the value.

$$AR\% = \frac{4}{5} \times 100 = 80\%$$

Step 5: Final Answer.

So, among women with multiple sexual partners, 80% of the risk of carcinoma cervix can be attributed to having multiple partners, while the rest would have occurred anyway from other causes.

$$\boxed{80\%}$$

Quick Tip: Attributable risk percent equals (RR minus 1) divided by RR, then multiplied by 100.

133. What is the best determinant of the health status of a country?

(A) Couple protection rate

(B) IMR

(C) MMR

(D) Crude birth rate

Correct Answer: (B) IMR

Solution:

Step 1: Understanding the Question:
We need the single indicator that best reflects the overall health status of a country, meaning it should mirror both the reach of health services and the general standard of living, nutrition, and sanitation.

Step 2: Key Formula or Approach:
Among simple mortality based indicators, the infant mortality rate (IMR) is regarded as the most sensitive index of a community's health, because infants are the most vulnerable group to poor maternal health, poor nutrition, unsafe water, and weak health care access. So a fall or rise in IMR closely tracks the overall level of health development in a country.

Step 3: Detailed Explanation:

(A) Couple protection rate: This only measures how many eligible couples are using a family planning method. It reflects family planning coverage, not the general health status of the population.

(B) IMR: Because infants react quickly and strongly to poor sanitation, undernutrition, poor antenatal care, and weak health infrastructure, IMR is treated as the most sensitive single marker of a

country's overall health status.

(C) MMR: Maternal mortality rate reflects the safety of pregnancy and childbirth care specifically. It is an important indicator, but it looks at a much narrower slice of health, pregnancy related deaths, rather than the health of the whole population.

(D) Crude birth rate: This measures fertility, that is, how many births occur per 1000 population. It says little about how healthy people are, since a country can have a high birth rate along with poor health services.

Step 4: Final Answer:
The best determinant of a country's health status is the infant mortality rate (IMR).

Quick Tip: IMR reacts strongly to nutrition, sanitation, and health care access, making it the most sensitive health status indicator.

134. Which of these is NOT a component of the Human Development Index (HDI)?

(A) Life expectancy at age one

(B) Educational level

(C) Per capita income

(D) Infant Mortality Rate (IMR)

Correct Answer: (D) Infant Mortality Rate (IMR)

Solution:

Step 1: Understanding the Question:
The Human Development Index (HDI) is a composite score put out each year by the United Nations Development Programme (UNDP) to rank countries on human development. We need to find the option that is not one of its building blocks.

Step 2: Key Concept:
HDI rests on three dimensions only: a long and healthy life, knowledge, and a decent standard of living. Anything outside these three has no place in the index.

Step 3: Detailed Explanation:
The health dimension is read from life expectancy at birth, so a life-expectancy figure belongs here.
The knowledge dimension is read from years of schooling, which is what educational level refers to.
The standard-of-living dimension is read from gross national income (GNI) per capita, which is close to per capita income.
The Infant Mortality Rate (IMR) does not sit under any of these three headings. IMR is used in an older composite score, the Physical Quality of Life Index (PQLI), not the HDI.

Step 4: Final Answer:
Since IMR plays no part in the HDI formula, option (D), Infant Mortality Rate, is the one that is NOT a component of HDI.

Quick Tip: HDI stands on three legs only: health, education, and income. IMR belongs to the older PQLI, not the HDI.

135. Which of these is NOT a component of the Physical Quality of Life Index (PQLI)?

(A) Infant Mortality Rate (IMR)

(B) Life expectancy at one year

(C) Maternal Mortality Rate (MMR)

(D) Literacy rate

Correct Answer: (C) Maternal Mortality Rate (MMR)

Solution:

Step 1: Understanding the Question:
The Physical Quality of Life Index (PQLI) was put forward by Morris D. Morris as a simple way to compare how well people live across countries, without relying on income alone. We need to find the option that does not belong to it.

Step 2: Key Concept:
PQLI is built from exactly three indicators, each scored on a scale of 1 to 100 and then averaged.

Step 3: Detailed Explanation:
The first indicator is the Infant Mortality Rate (IMR), which reflects how well a society protects its newborns.
The second indicator is life expectancy at age one, not at birth, because starting the clock at age one removes the heavy pull of infant deaths on the number.
The third indicator is the basic literacy rate, which stands in for education.
Maternal Mortality Rate (MMR) is never part of this formula. MMR is tracked separately as a measure of maternal health, not as part of

PQLI.

Step 4: Final Answer:
Since MMR is not one of the three PQLI indicators, option (C), Maternal Mortality Rate, is correct.

Quick Tip: PQLI = IMR + life expectancy at age one + literacy rate. MMR is not one of its three legs.

136. A study of blood pressure (BP) is done on 100 healthy individuals aged 25-27 years. The result is a normal distribution with median BP of 120 mm Hg. What percentage of the subjects will have a BP reading higher than 120?

(A) 25

(B) 50

(C) 75

(D) 100

Correct Answer: (B) 50

Solution:

Step 1: Understanding the Question:
We are told the BP readings of 100 healthy people follow a normal distribution, and the median value is 120 mm Hg. We need to find what share of readings sit above 120.

Step 2: Key Concept:
A normal distribution is a symmetric, bell-shaped curve. In a perfectly

symmetric curve, the mean, median, and mode all sit at the same point, right at the centre of the bell.

Step 3: Detailed Explanation:
Because the curve is symmetric about its centre, exactly half the area under the curve lies to the left of that centre point and exactly half lies to the right.
Here the centre point is the median, 120 mm Hg.
So half of the 100 subjects, meaning 50 subjects, will have a BP reading below 120, and the other half will have a reading above 120.
The reading of exactly 120 mm Hg sits at the centre itself and is not counted as higher than 120.

Step 4: Final Answer:
50 percent of the subjects will have a BP reading higher than 120 mm Hg, so option (B) is correct.

Quick Tip: In a normal distribution, mean = median = mode, and the curve splits exactly 50-50 about that centre value.

137. In which of these conditions is post-exposure prophylaxis NOT useful?

(A) Measles

(B) Rabies

(C) Pertussis

(D) Hepatitis B

Correct Answer: (C) Pertussis

Solution:

Step 1: Understanding the Question:

Post-exposure prophylaxis (PEP) means giving a vaccine, immunoglobulin, or drug right after someone has been exposed to an infection, so as to stop or soften the disease before it sets in. We need the disease where this approach does not work.

Step 2: Key Concept:

PEP works best when the incubation period of the disease is long enough to let the body build protection, or when a ready-made antibody (immunoglobulin) can be given straight away.

Step 3: Detailed Explanation:

In measles, a contact can be given the measles vaccine within 72 hours of exposure, or immunoglobulin within 6 days, since measles has an incubation period of about 10 to 14 days, long enough for protection to build.

In rabies, PEP is the standard of care: wound care plus rabies vaccine and, where indicated, rabies immunoglobulin, since the incubation period is usually weeks to months.

In hepatitis B, a non-immune contact (for example a needle-stick exposure) is given hepatitis B immunoglobulin (HBIG) together with the vaccine, and this combination works well because the incubation period runs into weeks to months.

Pertussis (whooping cough) has a short incubation period of about 7 to 10 days, and there is no vaccine-based post-exposure prophylaxis that reliably prevents disease in an exposed contact. Instead, close contacts of a pertussis case are given a short course of a macrolide antibiotic (chemoprophylaxis), which is a different strategy from vaccine or immunoglobulin based PEP.

Step 4: Final Answer:
Because pertussis is not prevented by a vaccine or immunoglobulin given after exposure, option (C), Pertussis, is the correct answer.

Quick Tip: Measles, rabies, and hepatitis B all have effective vaccine or immunoglobulin based PEP; pertussis instead relies on antibiotic chemoprophylaxis.

138. Hb of less than what value is the cut-off used by WHO guidelines to label an infant under 6 months of age as being anemic?

(A) 100 g/L

(B) 105 g/L

(C) 110 g/L

(D) 115 g/L

Correct Answer: (D) 115 g/L

Solution:

Step 1: Understanding the Question:
WHO defines anemia using different haemoglobin (Hb) cut-offs for different age and sex groups, since normal Hb naturally varies with age. We need the cut-off used specifically for infants under 6 months of age.

Step 2: Key Concept:
The commonly quoted WHO Hb cut-off for children 6 to 59 months of age is below 110 g/L (11 g/dl). Infants under 6 months are treated as a separate, slightly different group in some WHO reference tables

because Hb naturally dips in the first weeks of life as fetal haemoglobin is replaced by adult haemoglobin.

Step 3: Detailed Explanation:
Per the reference used for this exam key, the cut-off for infants below 6 months is set a little higher, at less than 115 g/L (11.5 g/dl), compared to the 110 g/L cut-off used from 6 months to 6 years.

Note: WHO's published Hb thresholds for anemia are revised across different technical documents over time, and different textbooks are not fully uniform on the exact figure used for infants under 6 months specifically (some sources quote 110 g/L for this group too). This exam's official answer key uses 115 g/L for infants under 6 months, and that is the answer kept here since it is not certain to be wrong under current guidance.

Step 4: Final Answer:
The cut-off used for infants under 6 months of age is Hb less than 115 g/L, so option (D) is correct.

Quick Tip: WHO uses 110 g/L for children 6 to 59 months; the cut-off quoted for infants under 6 months in this exam key is 115 g/L.

139. What are the amounts of calories and protein received by a pregnant woman from the anganwadi worker?

(A) 300 calories, 15 gm protein

(B) 500 calories, 15 gm protein

(C) 300 calories, 25 gm protein

(D) 500 calories, 25 gm protein

Correct Answer: (D) 500 calories, 25 gm protein

Solution:

Step 1: Understanding the Question:
The anganwadi worker, under the Integrated Child Development Services (ICDS) scheme, gives supplementary nutrition to pregnant women, lactating mothers, and young children through the anganwadi centre. We need the calorie and protein amount fixed for a pregnant woman under the norms used for this exam.

Step 2: Key Concept:
ICDS lays down fixed daily supplementation targets for each beneficiary group (children 6 months to 3 years, children 3 to 6 years, severely malnourished children, and pregnant and lactating women), and the amount is higher for pregnant and lactating women than for young children because of the extra demand of pregnancy.

Step 3: Detailed Explanation:
Under the norms this question is based on, a pregnant woman is given supplementary nutrition worth 500 calories and 25 gm of protein per day through the anganwadi worker.
This is higher than the amount given to a normal preschool child (500 calories, 12 to 15 gm protein) because pregnancy raises the protein need much more than it raises the plain calorie need.

Note: ICDS supplementation norms have been revised more than once since this exam was set in 2001 (later revisions set different exact figures for pregnant and lactating women), so a current-day figure quoted in a newer textbook may differ slightly from the value used in this answer key. The answer kept here matches the reference this paper's key was built on.

Step 4: Final Answer:
The amount given to a pregnant woman is 500 calories and 25 gm protein, so option (D) is correct.

Quick Tip: Under ICDS, a pregnant woman gets more protein per day than a young child gets, because pregnancy raises protein need sharply.

140. The incidence of malaria in an area is 20, 20, 50, 56, 60, 5000, 678, 898, 345, 456. Which of these methods is the best to calculate the average incidence?

(A) Arithmetic mean

(B) Geometric mean

(C) Median

(D) Mode

Correct Answer: (C) Median

Solution:

Step 1: Understanding the Question:
We are given ten yearly (or area-wise) malaria counts, and one of these values, 5000, is far larger than all the rest. We need to pick the

measure of central tendency that best represents the typical or average incidence here.

Step 2: Key Concept:
The arithmetic mean is pulled strongly toward extreme values (outliers), so a single very large or very small number can drag the mean away from where most of the data actually sits. The median, on the other hand, is simply the middle value once the data is sorted, and it is not affected by how large or small the extreme values are.

Step 3: Detailed Explanation:
Sort the data: 20, 20, 50, 56, 60, 345, 456, 678, 898, 5000.
If we compute the arithmetic mean, the value 5000 alone would inflate the average far above where most of the other nine readings actually sit, giving a misleading picture of typical incidence.
The geometric mean handles skew a little better than the arithmetic mean, but it is mainly used for rates of growth or ratios, and can still be distorted by one extreme value, plus it is awkward to interpret directly as an incidence figure.
The mode is not useful here either, since none of the values repeat, so there is no single most frequent value to report.
The median is simply the average of the 5th and 6th values in the sorted list, (60 + 345) / 2, and this middle value is completely unaffected by how extreme the number 5000 is.
Because of this resistance to outliers, the median is the standard choice whenever a data set is highly skewed by one or a few extreme readings, exactly the situation here.

Step 4: Final Answer:
Since the data is skewed by the outlier 5000, the median gives the best representative average incidence, so option (C) is correct.

Quick Tip: One extreme value (5000) skews this data badly; the median resists outliers far better than the arithmetic mean does.

141. A 6 year old child has a history of birth asphyxia. The child does not communicate well, shows slow mental and physical growth, does not mix with other people, has limited interests, and gets wildly agitated if disturbed. What is the diagnosis?

(A) Autistic disorder

(B) Histrionic personality

(C) ADHD (Attention Deficit Disorder)

(D) Schizophrenia

Correct Answer: (A) Autistic disorder

Solution:

Step 1: Understanding the Question:

A 6 year old boy has a history of birth asphyxia. He does not communicate well, shows slow mental and physical growth, avoids mixing with other people, has a narrow set of interests, and becomes very agitated if his routine is disturbed. We need to name this pattern.

Step 2: Key Concept:

Autism spectrum disorder is marked by three core features that begin early in childhood: poor social interaction, delayed or poor communication, and repetitive, restricted behavior with a strong need for sameness. Birth asphyxia is a known risk factor for neurodevelopmental disorders, including autism.

Step 3: Detailed Explanation:

The child's poor communication and reluctance to mix with people point to a social and language deficit. His limited interests and extreme agitation when his routine is disturbed show the repetitive behavior and resistance to change seen in autism. Slow mental and physical growth fits the developmental delay often seen alongside autism.

Histrionic personality shows up as excessive emotion seeking and attention seeking behavior in older children or adults, not as a failure to communicate or mix with people, so it does not fit here.

ADHD centers on inattention, overactivity, and impulsiveness, not on a core failure of social bonding and communication, so this child's picture does not match ADHD either.

Schizophrenia needs psychotic features such as hallucinations or delusions, and it is rare to diagnose it in a 6 year old; nothing here points to psychosis.

Step 4: Final Answer:

The pattern of poor communication, social withdrawal, restricted interests, and agitation on disruption of routine, starting in early childhood, points to autistic disorder.

Quick Tip: Look for the three core features of autism: poor social interaction, poor communication, and repetitive or restricted behavior with resistance to change.

142. A man feels that his nose is too long, though his friends feel otherwise. He has consulted three plastic surgeons, but all of them have refused to treat him. What condition does he suffer from?

(A) Hypochondriasis

(B) Somatisation

(C) Munchausen syndrome

(D) Delusional disorder

Correct Answer: (D) Delusional disorder

Solution:

Step 1: Understanding the Question:
A man is convinced that his nose is too long, even though his friends and, by implication, three separate plastic surgeons do not find anything wrong with it. He has gone from surgeon to surgeon looking for correction, and each one has turned him down. We need the condition that fits this fixed, false belief about his own body.

Step 2: Key Concept:
Hypochondriasis is fear of having a serious underlying disease based on misreading normal body sensations, not a fixed belief about one's physical appearance.
Somatisation is repeated physical complaints in many body systems, without an organic cause, used to seek medical attention.
Munchausen syndrome is fabricating or inducing symptoms on purpose to play the sick role and gain attention.
Delusional disorder, somatic type, is a fixed, false belief about the body that will not budge even with clear evidence against it. This form has also been called monosymptomatic hypochondriacal psychosis in older texts.

Step 3: Detailed Explanation:

This man's belief about his nose is not backed by any objective defect, since his friends see nothing wrong and three independent plastic surgeons, who would have every professional reason to operate if a real deformity existed, all refused. Despite this consistent, repeated reassurance, he keeps chasing surgery. That refusal to accept solid outside evidence is what makes the belief delusional rather than an ordinary worry.

It is not hypochondriasis, because he is not worried about a hidden disease, he is fixed on how his body looks.

It is not somatisation, because there are no multiple unexplained physical symptoms across body systems here, only one fixed idea about the nose.

It is not Munchausen syndrome, because he is not faking a disease to get attention or play a sick role, he genuinely believes his nose has a physical flaw.

Note: in current classification, a milder version of this pattern with some retained insight is called body dysmorphic disorder, but when the belief is completely fixed and does not bend to repeated, consistent contrary evidence from multiple experts, as written here, it carries delusional intensity and is placed under delusional disorder, somatic type.

Step 4: Final Answer:

A fixed, unshakeable false belief about a body part, undisturbed by consistent reassurance from multiple specialists, is delusional disorder.

Quick Tip: A fixed false belief about the body that does not change even after repeated expert reassurance points to a delusion, not simple health anxiety.

143. In a patient with no significant previous history, no history of any drug intake, and a normal ECG, what is the likely cause of palpitations, sweating, and a feeling of impending doom, with each episode lasting about 10 minutes?

(A) Hysteria

(B) Panic attack

(C) Agoraphobia

(D) Generalised anxiety disorder

Correct Answer: (B) Panic attack

Solution:

Step 1: Understanding the Question:
A patient with no past illness, no drugs on board, and a normal ECG has short episodes, about 10 minutes each, of palpitations, sweating, and a feeling that something terrible is about to happen. We need the diagnosis that best explains discrete episodes like this.

Step 2: Key Concept:
A panic attack is a sudden burst of intense fear that peaks within minutes and comes with physical symptoms such as a racing heart, sweating, shortness of breath, chest discomfort, and a sense of impending doom or loss of control. It is discrete, meaning it has a clear start and end, unlike a constant background state.

Step 3: Detailed Explanation:

The normal ECG and the absence of drug intake rule out a cardiac arrhythmia or a stimulant-induced cause for the palpitations, which pushes the diagnosis toward a primary anxiety disorder.

The short, self-limited, episodic nature of the symptoms, about 10 minutes and then resolving, matches the defined course of a panic attack far better than a constant condition.

Hysteria, better called conversion disorder today, usually shows up as a loss of motor or sensory function, such as a limb that will not move or sudden blindness, not as episodes of palpitations and sweating, so it is a poor fit.

Agoraphobia is fear of situations where escape might be hard or help unavailable, such as crowds or open spaces; it is often a complication that develops after repeated panic attacks, not the direct cause of a single episode of palpitations and doom.

Generalised anxiety disorder is excess worry about everyday things spread across most days for six months or more, a steady low grade state, not a sharp 10 minute spike of fear and physical symptoms.

Step 4: Final Answer:

The sudden, short, intense episode of palpitations, sweating, and impending doom, with no cardiac or drug cause found, is a panic attack.

Quick Tip: A discrete, minutes-long episode of palpitations, sweating, and doom, with a normal heart workup, is the hallmark of a panic attack.

144. A person on treatment with lithium for a mood disorder presents with seizures, increased reflexes, and epileptic fits. The patient also has a history of severe gastroenteritis. On investigation, the serum lithium level was found to be 1.95 mEq/L. This patient is most likely suffering from

(A) Severe dehydration

(B) Lithium toxicity

(C) Epilepsy

(D) Manic episode

Correct Answer: (B) Lithium toxicity

Solution:

Step 1: Understanding the Question:
A patient on lithium develops seizures, brisk reflexes, and fits after an episode of severe gastroenteritis, and the serum lithium level comes back at 1.95 mEq per litre. We need the condition that best explains this combination.

Step 2: Key Concept:
The safe treatment range for lithium is about 0.6 to 1.2 mEq per litre. Levels above roughly 1.5 mEq per litre are considered toxic, and levels above 2.5 mEq per litre are a severe, life threatening emergency. Signs of lithium toxicity include coarse tremor, ataxia, confusion, increased deep tendon reflexes, and seizures. Gastroenteritis causes vomiting and diarrhoea, which leads to dehydration and salt loss, and this reduces the kidney's ability to clear lithium, letting the drug build up in the blood.

Step 3: Detailed Explanation:
Here the serum lithium of 1.95 mEq per litre sits well above the toxic

threshold, so the number itself already points to toxicity rather than a therapeutic level.
The gastroenteritis is not a coincidence, it is the trigger: fluid loss shrank the volume the kidneys had to work with and cut down lithium clearance, so the drug that was previously at a safe dose became toxic.
Increased reflexes and seizures are classic neurological signs of lithium toxicity, since lithium is neurotoxic at high concentration and irritates the nervous system once it builds up.
Severe dehydration alone can cause weakness and low blood pressure, but it does not typically cause seizures and hyperreflexia by itself, and it does not explain why the specific measured lithium level is so high; here dehydration is the cause of the toxicity, not the full diagnosis on its own.
Epilepsy is a primary seizure disorder unrelated to a drug level, and nothing in this history points to an independent seizure condition; the seizure here has a clear drug related cause.
A manic episode would show mood and behavior change, not seizures or a raised drug level, so it does not fit the findings described.

Step 4: Final Answer:
A lithium level of 1.95 mEq per litre with seizures and hyperreflexia, in a patient who became dehydrated from gastroenteritis, is lithium toxicity.

Quick Tip: Lithium level above about 1.5 mEq/L, plus dehydration from gastroenteritis reducing renal clearance, plus seizures and hyperreflexia, means toxicity.

145. A 25 year old female was brought to the casualty after she allegedly attempted suicide, with her wrists slashed. She has a past history of difficulty in maintaining interpersonal relationships and also recurrent mood fluctuation episodes. What is the most likely diagnosis?

(A) Depression

(B) Borderline personality disorder

(C) Histrionic personality

(D) Schizophrenia

Correct Answer: (B) Borderline personality disorder

Solution:

Step 1: Understanding the Question:
A 25 year old woman has slashed her wrists in a suicide attempt, and her history shows long standing trouble keeping relationships stable along with repeated swings in mood. We need the diagnosis that best fits this long term pattern plus the self harm act.

Step 2: Key Concept:
Borderline personality disorder is a long standing pattern, present since early adulthood, of unstable relationships, an unstable self image, marked impulsivity, recurrent self harm or suicidal behavior such as wrist slashing, and sharp mood swings that can shift within hours to days. It is a personality disorder, meaning it is a fixed way of relating to the world, not a single mood episode.

Step 3: Detailed Explanation:
The wrist slashing is a classic self harm act seen often in borderline personality disorder, used at times to cope with intense emotional pain.

Her past history of difficulty keeping relationships stable is a core marker of borderline personality disorder, where relationships often swing between idealising a person and devaluing them.
The recurrent mood fluctuation episodes fit the affective instability of this disorder, where mood can shift quickly in reaction to stress, rather than staying low for weeks as in a depressive episode.
Depression is a mood episode with sustained low mood, loss of interest, and low energy over at least two weeks; it does not by itself explain a long standing pattern of unstable relationships going back further than the current crisis, so it is a less complete fit than a personality disorder.
Histrionic personality centers on excessive emotionality and attention seeking behavior, often through dramatic or seductive presentation, but it does not carry the same weight of recurrent self harm and identity instability that borderline personality disorder does, so it fits less well here.
Schizophrenia needs psychotic features such as hallucinations, delusions, or disorganised thinking, none of which are mentioned in this history.

Step 4: Final Answer:
A pattern of unstable relationships, recurrent mood swings, and impulsive self harm such as wrist slashing points to borderline personality disorder.

Quick Tip: Long standing unstable relationships plus mood swings plus impulsive self harm such as wrist slashing point to borderline personality disorder, not a single depressive episode.

146. A 70 year old man presents with a history of prosopagnosia, loss of memory, and third person hallucinations since 1 month. On examination, deep tendon reflexes are increased, and the mini mental state examination score is 20/30. What is the most likely diagnosis?

(A) Dissociated dementia

(B) Schizophrenia

(C) Multi infarct dementia

(D) Alzheimers disease

Correct Answer: (C) Multi infarct dementia

Solution:

Step 1: Understanding the Question:
A 70 year old man has trouble recognising familiar faces, called prosopagnosia, along with memory loss and hallucinations of a voice commenting on him in the third person, all starting within the last month. His reflexes are increased on exam, and his mini mental state score is 20 out of 30, showing mild to moderate cognitive impairment. We need the best diagnosis for this picture.

Step 2: Key Concept:
Multi infarct, or vascular, dementia comes from a series of small strokes in the brain. It often shows a relatively sudden or step wise onset, focal neurological signs such as increased or asymmetric reflexes, and cognitive loss that can include specific deficits like face recognition trouble depending on which brain areas were hit. Alzheimer's disease, in contrast, usually creeps in slowly over one to several years, without early focal neurological signs like brisk reflexes.

Step 3: Detailed Explanation:

The short one month time course here is far too fast for typical Alzheimer's disease, which develops gradually and is usually noticed only after a year or more of slow decline.
The increased deep tendon reflexes are a focal neurological sign, pointing to actual damage in specific motor pathways in the brain, which is a hallmark of vascular injury rather than the diffuse, gradual degeneration seen in Alzheimer's disease.
Prosopagnosia points to damage in a specific brain region that handles face recognition, fitting with patchy, localized infarcts rather than the more even, widespread cortical loss typical of Alzheimer's disease.
Hallucinations and cognitive decline can both be explained as complications of the vascular brain injury affecting multiple areas.
Schizophrenia is very unlikely to begin for the first time at age 70, and it would not explain the memory loss, the prosopagnosia, or the increased reflexes, since schizophrenia is a primary psychotic illness without this kind of focal neurological sign.
Dissociated dementia is not a standard diagnostic entity and does not fit this organic, focal neurological picture at all.

Step 4: Final Answer:
The fast onset, focal neurological sign of increased reflexes, and patchy cognitive deficits such as prosopagnosia together point to multi infarct dementia.

Quick Tip: A fast, about 1 month, onset plus a focal neurological sign such as increased reflexes plus patchy deficits like prosopagnosia points to vascular, multi infarct dementia rather than the slow course of Alzheimer's.

147. What is the investigation of choice for a parameningeal rhabdomyosarcoma?

(A) CECT

(B) CSF cytology

(C) MRI

(D) SPECT

Correct Answer: (C) MRI

Solution:

Step 1: Understanding the Question:
Parameningeal rhabdomyosarcoma is a soft tissue cancer that grows near the meninges, in sites such as the middle ear, nasal cavity and sinuses, or the area near the base of the skull. Because of its location, it can spread into the skull base and even reach the meninges and brain, so choosing the right imaging matters for staging and planning treatment. We need the investigation of choice.

Step 2: Key Concept:
MRI gives far better soft tissue contrast than CT, which makes it the best tool for showing the exact extent of a soft tissue tumor, especially when it may be tracking into the skull base, along cranial nerves, or into the meninges. This detail changes how radiation and surgery are planned.

Step 3: Detailed Explanation:
CECT, contrast enhanced CT, is useful for looking at bone destruction, but it is weaker than MRI at separating tumor from normal soft tissue and does not show meningeal or intracranial extension as clearly, so it falls short as the primary staging tool for a

parameningeal site.
CSF cytology can pick up cancer cells if the tumor has already spread into the cerebrospinal fluid, but it is a test for confirming spread once it is suspected, not the main imaging test used to define how far the primary tumor itself extends.
SPECT is a functional nuclear medicine scan more suited to looking at blood flow or metabolic activity in certain conditions, and it does not give the detailed anatomical picture of local tumor extension that is needed here.
MRI, with its excellent soft tissue detail, is the standard way to map how far a parameningeal rhabdomyosarcoma has grown, including any extension toward the meninges or skull base, which is exactly the information needed for staging and treatment planning.

Step 4: Final Answer:
Because of its superior soft tissue and skull base detail, MRI is the investigation of choice for parameningeal rhabdomyosarcoma.

Quick Tip: Parameningeal sites, such as the ear, nose, sinuses, and skull base, need the best soft tissue and skull base detail to check meningeal spread, and that means MRI.

148. What is the investigation of choice for neuroendocrine tumors?

(A) Endoscopic USG

(B) SPECT

(C) Radionuclide study

(D) MRI

Correct Answer: (C) Radionuclide study

Solution:

Step 1: Know what neuroendocrine tumors are.
Neuroendocrine tumors (NETs) are growths that come from cells of the diffuse neuroendocrine system, seen in the gut, pancreas, lungs, and other sites. Common examples are carcinoid tumors and pancreatic NETs.

Step 2: Recall the key feature these tumors share.
Most neuroendocrine tumor cells carry a large number of somatostatin receptors on their surface. This receptor is the target used for imaging.

Step 3: Match the receptor to the test.
A radionuclide study called somatostatin receptor scintigraphy (the octreotide scan, done with indium-111 labeled octreotide) binds to these receptors and lights up the tumor and any spread on a whole-body scan. This makes it the investigation of choice for finding the primary tumor and looking for metastases in one sitting.

Step 4: Rule out the other options.
Endoscopic ultrasound is useful for small pancreatic lesions but cannot survey the whole body for spread. SPECT is only a type of image acquisition, not a receptor-specific test by itself. MRI shows anatomy well but does not pick out neuroendocrine tissue the way a receptor-based scan does.

Step 5: Note the modern update.
Today Ga-68 DOTATATE PET/CT, a newer form of the same receptor-based radionuclide imaging, has mostly replaced the older octreotide scan because it is more sensitive. This does not change the

answer here, since both fall under the same principle of a radionuclide study.

Final Answer:
The investigation of choice for neuroendocrine tumors is

Radionuclide study (somatostatin receptor scintigraphy)

Quick Tip: NET cells carry somatostatin receptors; a receptor-targeted radionuclide scan (octreotide/DOTATATE) finds the tumor and any spread in one test.

149. What is the investigation of choice for screening renovascular hypertension with bilateral renal artery stenosis?

(A) Duplex Doppler study

(B) Captopril enhanced radionuclide scan

(C) USG

(D) MR angiography

Correct Answer: (A) Duplex Doppler study

Solution:

Step 1: Understand what is being screened for.
Renovascular hypertension is high blood pressure caused by narrowing of one or both renal arteries, which drops blood flow to the kidney and turns on the renin-angiotensin system.

Step 2: Recall why the captopril scan is usually a good screening test.

The captopril enhanced radionuclide renal scan compares how each kidney handles a tracer before and after giving captopril, and a stenotic kidney shows a delayed or reduced uptake after the drug.

Step 3: See why this test fails when both sides are narrowed.
The captopril scan relies on comparing one kidney against the other. If both renal arteries are narrowed to a similar degree, both kidneys respond the same way to captopril, so the test cannot show a clear side-to-side difference. This makes it unreliable in bilateral disease.

Step 4: Identify the better test for bilateral stenosis.
Duplex Doppler ultrasound directly measures blood flow velocity in each renal artery. It does not depend on comparing kidney function between the two sides, so it still works even when both arteries are affected. It is also non-invasive, free of radiation, and can be repeated easily.

Step 5: Rule out the remaining options.
Plain USG shows kidney size and structure but cannot directly assess arterial flow the way Doppler does. MR angiography is an accurate anatomical test but is used more to confirm stenosis after screening, not as the first-line screening test, and is costlier.

Final Answer:
For screening renovascular hypertension when both renal arteries may be stenosed, the preferred test is

Duplex Doppler study

Quick Tip: Captopril renogram needs a normal kidney to compare against; in bilateral stenosis, use Duplex Doppler instead.

150. What is the next investigation to be done in a case with recurrent hemoptysis, normal chest X-ray, and production of purulent sputum?

(A) Spiral CT

(B) HRCT

(C) MRI

(D) Bronchoscopy

Correct Answer: (B) HRCT

Solution:

Step 1: Set up the clinical picture.
The patient has recurrent hemoptysis (coughing blood), a normal chest X-ray, and purulent sputum. Purulent sputum with recurrent bleeding on a normal X-ray points toward a chronic airway disease that a plain film can miss, most often bronchiectasis.

Step 2: Recall the best test for suspected bronchiectasis.
High resolution CT (HRCT) of the chest gives thin, detailed slices of the lung that can show the dilated, thick-walled airways typical of bronchiectasis even when the chest X-ray looks normal. It is the most sensitive test for this diagnosis.

Step 3: Compare with spiral CT.
Spiral (helical) CT scans the chest continuously and is better suited to picking up pulmonary embolism or a vascular cause of bleeding, such

as an active bleeding vessel before embolization. It is not the first test chosen when the picture points to a chronic airway problem rather than a vascular one.

Step 4: Compare with MRI.
MRI is not good at imaging air-filled lung tissue because it has poor spatial resolution for lung parenchyma, so it is not used to work up bronchiectasis or hemoptysis.

Step 5: Compare with bronchoscopy.
Bronchoscopy is very useful for finding the exact bleeding site, taking biopsies, or ruling out a tumor, but it is usually done after imaging has localized the disease, or when imaging is inconclusive, not as the very next step when the picture already suggests bronchiectasis.

Final Answer:
The next investigation of choice here is

HRCT chest

Quick Tip: Normal CXR + purulent sputum + recurrent hemoptysis suggests bronchiectasis; HRCT is the most sensitive test for it.

151. What is the diagnosis in a woman who has scarring alopecia, thinning of nails, and hyperpigmented patches over the leg?

(A) Lichen planus

(B) Psoriasis

(C) Secondary syphilis

(D) Dermatophytosis

Correct Answer: (A) Lichen planus

Solution:

Step 1: List the three clinical clues given.
The woman has three findings together: scarring hair loss, thin nails, and dark patches on the legs. Diagnosing the disease means matching one condition that explains hair, nail, and skin changes together.

Step 2: Match hair loss to the diagnosis.
Lichen planopilaris is the form of lichen planus that attacks hair follicles. It destroys the follicle permanently, so the hair loss it causes is scarring, meaning the scalp skin looks smooth and shiny where the follicles used to be, unlike patchy but reversible hair loss in other diseases.

Step 3: Match the nail change.
Lichen planus of the nail causes thinning of the nail plate, longitudinal ridging, and in bad cases a permanent scar called pterygium, where a skin fold grows over the nail bed. Thin nails fit this pattern.

Step 4: Match the skin change on the leg.
A variant called lichen planus pigmentosus produces flat, dark brown to slate colored patches, often on sun exposed or flexural skin including the legs. This explains the hyperpigmented patches.

Step 5: Rule out the other options.
Psoriasis causes nail pitting and thickening, not thinning, and its hair loss is non-scarring. Secondary syphilis gives a diffuse rash with moth-eaten, non-scarring hair loss, not the pattern described here. Dermatophytosis is a fungal skin infection that does not cause scarring alopecia or nail thinning in this pattern.

Final Answer:
The combination of scarring alopecia, nail thinning, and hyperpigmented leg patches points to

Lichen planus

Quick Tip: Lichen planus can affect the scalp (scarring alopecia), nails (thinning/pterygium), and skin (pigmented patches) together.

152. Acne vulgaris involves which one of the following?

(A) Pilosebaceous glands

(B) Eccrine glands

(C) Apocrine glands

(D) Sebaceous glands

Correct Answer: (A) Pilosebaceous glands

Solution:

Step 1: Recall the site of acne vulgaris.
Acne vulgaris is a common skin disorder that develops in the

pilosebaceous unit, the small structure made of a hair follicle joined to its sebaceous gland and a fine hair.

Step 2: Walk through how acne actually forms.
The process starts with the sebaceous gland making extra sebum under hormone drive, but the disease is not caused by the gland alone. Dead skin cells build up and block the follicle opening, trapping sebum inside. This blockage forms a comedone, the first visible lesion of acne.

Step 3: Add the bacterial step.
Once the follicle is blocked, the bacterium Cutibacterium acnes multiplies in the trapped sebum, and its byproducts trigger inflammation, giving the red papules and pustules seen in acne.

Step 4: See why pilosebaceous is the accurate answer.
Since both the follicle (the pilo part) and the gland (the sebaceous part) are needed for a comedone and later inflammation to form, the disease is best described as one of the pilosebaceous unit, not the sebaceous gland alone.

Step 5: Rule out the other glands.
Eccrine glands produce watery sweat for temperature control and play no role in acne. Apocrine glands, found in the armpit and groin, are linked to body odor and conditions like hidradenitis suppurativa, not acne. Sebaceous glands alone are only part of the structure involved; the follicle itself is equally essential to the disease process.

Final Answer:
Acne vulgaris is a disease of the

Pilosebaceous glands (pilosebaceous unit)

Quick Tip: Acne forms from BOTH the hair follicle and sebaceous gland together, the pilosebaceous unit, not the gland alone.

153. A patient diagnosed with psoriasis was put on treatment with high dose dexamethasone for 2 weeks. The patient stopped treatment after which he develops high-grade fever and generalized pustular lesions all over his body. What is the most likely diagnosis?

(A) Septicaemia

(B) Drug reaction

(C) Pustular psoriasis

(D) Secondary bacterial infection

Correct Answer: (C) Pustular psoriasis

Solution:

Step 1: Note the key trigger in the history.
The patient already has psoriasis and was given a high dose systemic steroid, then stopped it abruptly after 2 weeks. Sudden withdrawal of systemic steroids in a psoriasis patient is a well known trigger for a flare.

Step 2: Recall what steroid withdrawal does in psoriasis.
Steroids suppress the immune activity driving psoriasis while they are given. Stopping them abruptly removes that suppression fast, and the disease rebounds, often in a more severe form than before treatment started.

Step 3: Match the rebound to its severe form.
In some patients this rebound takes the form of generalized pustular psoriasis (the von Zumbusch pattern), with widespread sheets of small sterile pustules, high-grade fever, and a feeling of being very unwell. This is exactly the picture described.

Step 4: Rule out the other options.
Septicaemia would show positive blood cultures and a source of infection, not a clean trigger of steroid withdrawal in a known psoriasis patient. A drug reaction usually appears after starting a new drug, not after stopping one, and does not typically produce this pustular pattern. Secondary bacterial infection would show pus that grows organisms on culture and usually starts at a local site, not as a sudden generalized eruption right after steroid withdrawal.

Final Answer:
The most likely diagnosis is

Pustular psoriasis (generalized, von Zumbusch type)

Quick Tip: Abrupt steroid withdrawal in a psoriasis patient can trigger generalized pustular psoriasis (von Zumbusch type).

154. What is the treatment of choice for hyperparathyroidism?

(A) Removal of the hyperplastic gland

(B) Removal of all 4 glands

(C) Radical parathyroidectomy

(D) 3 and 1/2 parathyroidectomy

Correct Answer: (B) Removal of all 4 glands

Solution:

Step 1: Split the causes of hyperparathyroidism.
Primary hyperparathyroidism is caused by an overactive parathyroid gland or glands. About 85% of cases come from a single adenoma in one gland, but a meaningful minority come from hyperplasia, where all four glands are enlarged and overactive together, as seen in some familial syndromes and in secondary or tertiary hyperparathyroidism from long-standing kidney disease.

Step 2: Focus on the four-gland hyperplasia scenario.
When all four glands are diseased, removing just one gland leaves three overactive glands behind, so the disease keeps going. The surgery has to deal with all four glands, not just one.

Step 3: Explain what removal of all 4 glands means in practice.
The standard operation for four-gland hyperplasia is a total parathyroidectomy, where all four enlarged glands are taken out of the neck, and then a small piece of parathyroid tissue is immediately reimplanted, usually into a forearm muscle. This autotransplant keeps a source of parathyroid hormone that can be adjusted or removed later if needed, while clearing the neck completely of diseased tissue.

Step 4: Compare with the other options.
Removing only the hyperplastic gland would miss the other enlarged glands and fail to cure the patient when hyperplasia is bilateral and multi-gland. Radical parathyroidectomy is not a standard recognized operation name for this condition. A subtotal, three and a half gland parathyroidectomy (leaving a remnant in the neck) is an alternative approach some surgeons use, but it carries a higher chance the disease recurs in the remaining neck tissue, and reoperating in a scarred neck is riskier than trimming a small forearm graft if hyperplasia comes back after a full four-gland clearance with autotransplant.

Step 5: Final conclusion.
For parathyroid hyperplasia, the preferred approach is to clear the neck completely.

Removal of all 4 glands (with autotransplantation)

Quick Tip: In four-gland parathyroid hyperplasia, all 4 glands are removed and a small piece is autotransplanted (often to the forearm).

155. Which gastric surgery results in the least degree of bilious vomiting, dumping, and diarrhea?

(A) Truncal vagotomy and pyloroplasty

(B) Truncal vagotomy and antrectomy

(C) Highly selective vagotomy (HSV)

(D) Gastrojejunostomy

Correct Answer: (C) Highly selective vagotomy (HSV)

Solution:

Step 1: Know what each surgery does to the pylorus.
Truncal vagotomy cuts both vagal trunks near the esophagus. This denervates the whole stomach, so the pylorus stops relaxing on its own and a drainage step (pyloroplasty) has to be added to let food leave the stomach.
Truncal vagotomy with antrectomy removes the acid producing vagal supply and the gastrin producing antrum together, giving the lowest ulcer recurrence but destroying the pylorus completely.
Gastrojejunostomy joins the stomach straight to the jejunum and bypasses the pylorus without removing it, so food and bile move through with no valve to slow them down.

Step 2: Connect pyloric loss to the symptoms in the question.
Bilious vomiting happens when bile refluxes back into the stomach because the pylorus no longer acts as a one way valve.
Dumping happens when a hyperosmolar meal empties too fast into the small bowel, since there is no pylorus left to hold it back and release it slowly.
Diarrhea follows the same rapid transit, plus loss of vagal control over small bowel motility.
All three problems are worst when the pylorus is bypassed or removed, as in pyloroplasty, antrectomy, or gastrojejunostomy.

Step 3: See why highly selective vagotomy avoids this.
HSV only cuts the vagal branches that go to the acid making parietal cells in the upper stomach (the fundus and body). It leaves the nerve of Latarjet, the antrum, and the pylorus fully intact and working.

Because the pylorus still opens and closes normally, gastric emptying stays close to normal, and no drainage procedure is needed.

Step 4: Rule out the other options.
Truncal vagotomy with pyloroplasty forces the pylorus open, so bile reflux and rapid emptying are common.
Truncal vagotomy with antrectomy removes the pylorus outright, giving the highest rates of dumping and diarrhea among the standard operations.
Gastrojejunostomy alone leaves a wide open channel next to a bypassed pylorus, so bile still washes into the stomach freely.

Step 5: Final answer.
Since HSV is the only option that keeps the pylorus working, it causes the least bilious vomiting, dumping, and diarrhea.

Highly selective vagotomy

Quick Tip: Look for the operation that leaves the pylorus untouched and working.

156. A 20-year-old man presents with massive hematemesis. He gives a history of taking some drugs for fever over the past 2 weeks. What is the likely diagnosis?

(A) Acute peptic ulceration due to NSAIDs

(B) Erosive gastritis

(C) Esophageal erosion

(D) Esophageal varices

Correct Answer: (A) Acute peptic ulceration due to NSAIDs

Solution:

Step 1: Pull out the key clue in the history.
A young man with no mention of liver disease, alcohol use, jaundice, or an enlarged spleen presents with massive hematemesis. The one detail the question gives on purpose is that he took drugs for fever over the last 2 weeks. Drugs taken for fever are almost always NSAIDs (aspirin, ibuprofen, diclofenac, and similar painkillers).

Step 2: Know how NSAIDs damage the stomach and duodenum.
NSAIDs block the COX-1 enzyme, which normally makes protective prostaglandins in the gastric lining. Losing this protection lets stomach acid injure the mucosa directly. Within days to a couple of weeks this can progress from small erosions to a frank peptic ulcer that erodes into a blood vessel, causing sudden, heavy hematemesis.

Step 3: Compare with the other options.
Erosive gastritis from NSAIDs is real but tends to cause diffuse, oozing bleeding from many shallow erosions rather than one massive bleed; a deep NSAID ulcer eroding a vessel better explains a large single bleed.
Esophageal erosion is usually linked to reflux disease or corrosive ingestion, neither of which is mentioned here.
Esophageal varices need portal hypertension, which usually comes with chronic liver disease, alcohol use, jaundice, or cirrhosis. None of that is given for this patient, so varices do not fit the history well.

Step 4: Match the vignette to the diagnosis.
The combination of a young patient, no liver disease clues, and a clear

2 week history of fever medication points to a drug induced peptic ulcer bleed rather than a chronic liver problem.

Note on the source answer key: the original paper's key marks esophageal varices as correct. Varices usually need an underlying cause such as cirrhosis or portal hypertension, and nothing in this vignette supports that, while the NSAID history is a specific, deliberate clue in the question. This answer has been corrected to acute peptic ulceration due to NSAIDs based on the clinical history given.

Step 5: Final answer.

Acute peptic ulceration due to NSAIDs

Quick Tip: Drugs taken for fever are usually NSAIDs; think about what they do to the stomach lining over 2 weeks.

157. What is NOT true in a case of urethral injury?

(A) Rare in women

(B) Immediate catheterisation is indicated

(C) Posterior urethral injury occurs in fracture of the pelvis

(D) Blood at the urinary meatus is diagnostic

Correct Answer: (B) Immediate catheterisation is indicated

Solution:

Step 1: Understand what the question wants.
We need the FALSE statement about urethral injury, not the true one. Three of the four statements are accepted facts, and one goes against standard teaching.

Step 2: Check "rare in women".
The female urethra is short and mobile, and it is well protected within the pelvis, so it is injured far less often than the longer, more fixed male urethra. This statement is true.

Step 3: Check "posterior urethral injury occurs in fracture pelvis".
The posterior (membranous) urethra is fixed at the urogenital diaphragm. A pelvic fracture, especially one with a shift at the pubic symphysis, can shear this fixed segment and tear it. This is the classic mechanism taught for posterior urethral injury, so this statement is true.

Step 4: Check "blood at the urinary meatus is diagnostic".
Blood dripping from the external meatus after trauma is the single most reliable clinical sign of a urethral injury and should always trigger further evaluation before any tube is passed. This statement is true.

Step 5: Check "immediate catheterisation is indicated".
This is the false one. When a urethral injury is suspected, passing a catheter blindly can convert a partial tear into a complete transection, push in infection, or create a false passage. The correct first step is a retrograde urethrogram to check whether the urethra is intact before any catheter is passed. So immediate catheterisation is not indicated, it is actually contraindicated until imaging is done.

Step 6: Final answer.
The statement that is NOT true is that immediate catheterisation is indicated.

Immediate catheterisation is indicated

Quick Tip: Think about what should be done first when blood is seen at the meatus after trauma.

158. What is the likely source of hematuria that has been persisting for the past 3 days, with red cell casts evident on urinalysis?

(A) Bladder

(B) Urethra

(C) Kidney

(D) Ureter

Correct Answer: (C) Kidney

Solution:

Step 1: Recall what a red cell cast actually is.
A cast is a cylindrical plug that forms inside the kidney's tubules, moulded by the tubule's own shape. Red cell casts form only when red blood cells get trapped inside this tubular mould before being washed out into the urine.

Step 2: Work out where that mould can happen.
Tubules exist only inside the kidney (nephron). The bladder, ureter, and urethra are just hollow tubes lined by transitional or squamous

cells; they have no tubular structure that could shape a cast.

Step 3: Apply this to the case.
Since red cell casts can only be made inside kidney tubules, their presence tells us the red blood cells entered the urine at the level of the kidney (glomerulus or tubule), not lower down in the urinary tract.

Step 4: Rule out the other options.
Bladder bleeding (from a stone, infection, or tumour) gives free red cells or clots, never true casts.
Ureteric bleeding also gives plain blood, since urine simply flows through the ureter without a tubular mould.
Urethral bleeding shows as blood at the start of urination or at the meatus, again with no casts.

Step 5: Final answer.
Red cell casts point specifically to a renal (glomerular) source of bleeding.

Kidney

Quick Tip: Only kidney tubules can mould red blood cells into a cast.

159. What is the treatment of choice for pleomorphic adenoma?

(A) Superficial parotidectomy

(B) Enucleation

(C) Deep parotidectomy

(D) Radical parotidectomy

Correct Answer: (A) Superficial parotidectomy

Solution:

Step 1: Recall what pleomorphic adenoma is.
Pleomorphic adenoma is the most common benign salivary gland tumour, usually found in the superficial lobe of the parotid gland. It has a thin, often incomplete capsule with small finger-like projections (pseudopodia) pushing into the surrounding gland tissue.

Step 2: Understand why simple removal fails.
Because the capsule is incomplete and pseudopodia extend beyond it, shelling the tumour out along its capsule (enucleation) leaves tumour cells behind. This is why enucleation carries a high rate of local recurrence and is no longer recommended.

Step 3: Understand why superficial parotidectomy works.
Superficial parotidectomy removes the whole superficial lobe of the parotid gland, along with the tumour and a margin of healthy tissue, while carefully identifying and preserving the facial nerve. Removing a wide margin of normal gland around the tumour takes out any pseudopodia along with it, giving a much lower recurrence rate.

Step 4: Rule out the other options.
Deep parotidectomy removes the deep lobe, which lies below the facial nerve plane; it is reserved for tumours that arise in or extend

into the deep lobe, not typical superficial pleomorphic adenoma. Radical parotidectomy removes the whole gland along with the facial nerve, done only for malignant tumours invading the nerve, which is far more aggressive than what a benign adenoma needs.

Step 5: Final answer.
For a typical pleomorphic adenoma of the superficial lobe, the standard treatment is

Superficial parotidectomy

Quick Tip: Think about why simply shelling out the tumour along its capsule is not enough.

160. What is the most effective treatment for a Warthin's tumor?

(A) Superficial parotidectomy

(B) Deep parotidectomy

(C) Enucleation

(D) Radiotherapy

Correct Answer: (A) Superficial parotidectomy

Solution:

Step 1: Recall what a Warthin's tumor is.
Warthin's tumor (papillary cystadenoma lymphomatosum) is the second most common benign salivary gland tumour, almost always found in the tail of the superficial lobe of the parotid gland. It can be bilateral and is strongly linked to smoking.

Step 2: Think about the surgical goal.
Like pleomorphic adenoma, this tumour sits in the superficial lobe, so it needs an operation that removes the tumour with a margin of normal gland while protecting the facial nerve that runs through this area.

Step 3: Match this to the surgery.
Superficial parotidectomy removes the superficial lobe along with the tumour and a safe margin, and lets the surgeon identify and spare the facial nerve. This gives complete removal with a low recurrence rate and is the standard operation.

Step 4: Rule out the other options.
Deep parotidectomy is for tumours in the deep lobe below the facial nerve, which is not where Warthin's tumor typically arises.
Enucleation just shells out the mass without a safety margin; it risks leaving tumour tissue behind and is not the preferred approach for parotid tumours.
Radiotherapy has no established role as primary treatment for this benign tumour; surgery is curative and radiotherapy is unnecessary here.

Step 5: Final answer.
The most effective treatment for Warthin's tumor is

Superficial parotidectomy

Quick Tip: Warthin's tumor arises in the tail of the superficial parotid lobe.

161. What is the treatment for a stage I testicular tumor?

(A) High orchidectomy

(B) High orchidectomy plus radiotherapy

(C) Scrotal orchidectomy

(D) Bilateral orchidectomy

Correct Answer: (B) High orchidectomy plus radiotherapy

Solution:

Step 1: Recall the standard first step for any testicular tumor.
Every suspected testicular tumor is removed through the groin, called a radical inguinal (high) orchidectomy, along with the spermatic cord up to the internal ring. This gives the diagnosis, the tumour type, and local control together.

Step 2: Understand what stage I means.
Stage I disease means the tumour is confined to the testis, with no spread to lymph nodes or distant organs on imaging. Testicular tumours, most often seminomas, spread first through the lymphatics to the para-aortic (retroperitoneal) lymph nodes.

Step 3: Understand why radiotherapy is added.
Seminoma is a very radiosensitive tumour. Even in stage I disease, a small number of patients can have microscopic spread that imaging misses. Adjuvant radiotherapy to the para-aortic nodal region after orchidectomy has classically been used to treat this hidden disease and cut down relapse.

Step 4: Rule out the other options.

High orchidectomy alone, without any further treatment, leaves microscopic nodal disease untreated in a proportion of patients, so relapse rates are higher when nothing more is done.
Scrotal orchidectomy is avoided on purpose. Cutting through the scrotum instead of the groin risks seeding tumour cells into the scrotal lymphatics and skin, so it is not the correct approach.
Bilateral orchidectomy removes both testes, which is not needed since disease is confined to one testis; this would cause needless loss of the healthy testis and hormone function.

Step 5: Note on modern practice.
Today, active surveillance after orchidectomy alone is also an accepted option for many stage I seminoma patients, since it avoids radiotherapy side effects, but high orchidectomy plus radiotherapy remains a standard, well established answer for stage I seminoma in exam settings.

Step 6: Final answer.

High orchidectomy plus radiotherapy

Quick Tip: Seminoma spreads early to para-aortic nodes and responds well to radiation.

162. Ameloblastoma of the mandible is most likely to involve which of the following locations?

(A) At symphysis menti

(B) Molar region of mandible

(C) In relation to upper 2nd molar

(D) In relation to incisors

Correct Answer: (B) Molar region of mandible

Solution:

Step 1: Recall what ameloblastoma is.
Ameloblastoma is a benign but locally invasive tumor that comes from the cells that would normally form tooth enamel (odontogenic epithelium). It grows slowly inside the jaw bone and can push through the cortex into soft tissue if left alone.

Step 2: Know where it likes to grow.
About 80 percent of ameloblastomas form in the mandible, and within the mandible the tumor favors the molar to ramus to angle region, most often starting near the third molar area and spreading into the ramus, angle, and body of the bone. The front of the jaw, the symphysis and incisor region, is affected far less often.

Step 3: Recall the typical clinical picture.
It usually shows up in the 4th or 5th decade of life. It grows slowly and is painless unless it gets infected. On imaging it often has a multilocular soap bubble look. Because it invades bone and soft tissue locally, wide excision with at least a 1 cm margin of normal bone is needed, since simple curettage has a high recurrence rate. It almost never spreads to lymph nodes, but late, incompletely removed tumors can seed the lungs. It does not respond well to radiotherapy.

Step 4: Rule out the other sites.
The symphysis menti (option A) and the incisor region (option D) are uncommon sites for ameloblastoma, these areas are more typical for lesions like odontomas or dentigerous cysts around impacted teeth. The area near the upper second molar (option C) is not even in the mandible, it is maxillary, and the maxilla is a far less common site for this tumor overall.

Step 5: Final answer.
The molar region of the mandible, extending toward the ramus and angle, is the classic and most common site.

Molar region of mandible

Quick Tip: Ameloblastoma is an odontogenic tumor that mostly affects the molar to ramus to angle region of the mandible.

163. What is the most common tumor involving the mandible?

(A) Osteosarcoma

(B) Ameloblastoma

(C) Lymphoma

(D) Squamous cell carcinoma

Correct Answer: (B) Ameloblastoma

Solution:

Step 1: Split tumor of the mandible into types.
Tumors that involve the mandible fall into a few groups: odontogenic

tumors that arise from tooth forming tissue, primary bone tumors like osteosarcoma, tumors of blood or lymphatic origin like lymphoma, and cancers that spread into the jaw from nearby oral structures, like squamous cell carcinoma of the gum or floor of the mouth invading bone.

Step 2: Identify the most common odontogenic tumor.
Among all odontogenic tumors, ameloblastoma is the most common clinically significant one, and the jaws, mainly the mandible, are by far its favorite site. As a standalone primary tumor of the mandible bone, it is encountered more often than primary bone sarcomas in standard surgical teaching.

Step 3: Rule out the other options.
Osteosarcoma of the jaw is real but far less common than ameloblastoma, and it usually behaves more aggressively with pain and rapid growth. Lymphoma can involve the jaw bones but is an uncommon primary presentation there. Squamous cell carcinoma more often arises from the oral mucosa and secondarily invades the mandible, rather than being a primary tumor of the mandible bone itself.

Step 4: Final answer.
Taking ameloblastoma as the most frequently seen primary mandibular tumor, as cited from Bailey and Love's Short Practice of Surgery, the answer is

Ameloblastoma

Quick Tip: Ameloblastoma, the most common odontogenic tumor, is the classic answer here.

164. What is the treatment for a cancer of the lateral border of the tongue with lower neck lymph node secondaries?

(A) Radical neck dissection

(B) Supraomohyoid dissection

(C) Suprahyoid neck dissection

(D) Teletherapy

Correct Answer: (A) Radical neck dissection

Solution:

Step 1: Understand the clinical situation.
The patient has a squamous cell carcinoma of the lateral border of the tongue that has already spread to lymph nodes in the lower neck. The disease is no longer confined to the primary site, the neck is clinically node positive, and the involvement is in the lower neck specifically.

Step 2: Recall the types of neck dissection.
A radical neck dissection removes all lymph node levels I to V on the involved side, along with the internal jugular vein, sternocleidomastoid muscle, and spinal accessory nerve. A supraomohyoid dissection is a selective dissection removing only levels I to III, used mainly for a clinically node negative neck as an elective, staging procedure. A suprahyoid dissection removes only the submental and submandibular nodes, level I, an even smaller field.

Step 3: Match the extent of disease to the extent of surgery.
Because this patient already has clinically proven secondaries in the lower neck, levels III to V, a limited selective dissection like supraomohyoid or suprahyoid would leave disease behind. The standard of care for a clinically node positive neck with lower neck involvement is a comprehensive dissection that clears all the at risk levels, which is the radical neck dissection, or a modified version that spares an uninvolved structure such as the accessory nerve.

Step 4: Explain why teletherapy alone is wrong.
Teletherapy, external beam radiotherapy, is used as an adjuvant after surgery, or as a primary option in patients unfit for surgery, but it is not the first line answer when surgical clearance of bulky, lower neck nodal disease is feasible.

Step 5: Final answer.

Radical neck dissection

Quick Tip: Node positive lower neck disease from tongue cancer needs a comprehensive radical neck dissection, not a selective one.

165. What is the treatment of choice for an old man who has reflux of foul smelling food?

(A) Cricopharyngeal myotomy

(B) Sac removal

(C) Laser evaporation

(D) Myotomy with sac excision

Correct Answer: (D) Myotomy with sac excision

Solution:

Step 1: Recognise the clinical picture.
An elderly man with regurgitation of undigested, foul smelling food, sometimes hours after eating, along with a gurgling noise in the neck, bad breath, and occasional aspiration, is a classic description of a pharyngeal pouch, also called Zenker's diverticulum.

Step 2: Recall why the pouch forms.
The pouch is a pulsion diverticulum that bulges out through a weak spot in the pharyngeal wall, called Killian's dehiscence, between the oblique fibres of the inferior constrictor and the transverse fibres of the cricopharyngeus. The underlying problem is poor relaxation or spasm of the cricopharyngeus muscle, which raises pressure in the pharynx during swallowing and pushes mucosa out through the weak area.

Step 3: Understand why myotomy alone is not enough.
A cricopharyngeal myotomy alone, option A, treats the underlying high pressure problem and can help small pouches, but once a pouch has already formed and become symptomatic, as here with foul smelling reflux, the sac itself keeps trapping food and causing symptoms unless it is removed or fixed.

Step 4: Understand why sac removal alone is not enough.
Removing the sac alone, option B, without dividing the tight cricopharyngeus muscle leaves the high pressure problem in place, raising the risk of recurrence or of a leak at the repair site.

Step 5: Consider laser evaporation.
Endoscopic laser or stapling techniques, option C, are used in some centers, mainly for smaller pouches or frail patients where open surgery carries more risk, but they are not the standard treatment of choice answer in this classic teaching format.

Step 6: State the correct combined approach.
The accepted treatment of choice combines both steps, a cricopharyngeal myotomy to release the abnormal pressure that caused the pouch, plus excision of the sac to remove the diverticulum, done together in the same operation.

Step 7: Final answer.

Myotomy with sac excision

Quick Tip: Foul smelling food regurgitation in an elderly man points to a pharyngeal pouch, treated by myotomy with sac excision.

166. What is the treatment for a 4 x 6 mm dysgerminoma in a 12 year old girl?

(A) Right cystectomy

(B) Right oophorectomy

(C) TAH with BSO

(D) Bilateral oophorectomy

Correct Answer: (B) Right oophorectomy

Solution:

Step 1: Identify the tumor type.
Dysgerminoma is the most common malignant germ cell tumor of the ovary. It occurs mostly in children, teenagers, and young women, and it is highly sensitive to chemotherapy and radiotherapy if it does spread, but the first step in management is always surgical.

Step 2: Recall the surgical principle for germ cell tumors in young patients.
Because dysgerminoma affects young girls who have not completed their family, and because it is usually confined to one ovary at diagnosis, the standard fertility sparing operation taught for this tumor is removal of the involved ovary and tube, a unilateral salpingo-oophorectomy, together with surgical staging such as peritoneal washings and inspection of the other ovary, omentum, and nodes. This preserves the uterus and the opposite ovary for future fertility and hormone production.

Step 3: Explain why the more radical options are wrong here.
Total abdominal hysterectomy with bilateral salpingo-oophorectomy, option C, and bilateral oophorectomy, option D, remove far more than needed in a child with disease apparently confined to one small area, and would take away all future fertility and put the girl into surgical menopause. These are reserved for advanced or bilateral disease, not a small unilateral lesion.

Step 4: A note on the size given in this question.
The lesion described here is very small, only a few millimeters, which is why the option listed as correct in the original answer key is right sided cystectomy, meaning the lesion alone is shelled out while the rest of the ovary is conserved. It is worth flagging that in current gynae oncology teaching, once a germ cell malignancy like

dysgerminoma is confirmed, a right salpingo-oophorectomy is generally preferred over a simple cystectomy, because enucleation of a known or suspected malignant tumor risks leaving tumor cells behind and does not allow proper assessment of the ovarian capsule for staging. The answer given here is kept as per the original source key, but it should be treated with caution and cross checked against current departmental protocol.

Step 5: Final answer as per the given key.

Right cystectomy

Answer key note: The original 2001 source key marked this as right cystectomy. Corrected here to right oophorectomy (unilateral salpingo-oophorectomy): standard gynecologic oncology management for a confirmed or suspected dysgerminoma, a malignant germ cell tumor, is removal of the entire ovary and tube on the affected side. Cystectomy alone risks incomplete tumor removal, capsule rupture, and inadequate staging, so it is not accepted management even for a small lesion.

Quick Tip: Dysgerminoma in a young girl is usually managed with fertility sparing ovarian surgery on the involved side.

167. What is NOT true about a varicocele?

(A) More common on the right side

(B) Associated with infertility

(C) Left varicocele can be a late sign of a tumor in an elderly man

(D) 10% cases are bilateral

Correct Answer: (A) More common on the right side

Solution:

Step 1: Understand what the question is asking.
This is a NOT true question, so we need to find the one statement among the four that is false, while the other three are correct facts about varicocele.

Step 2: Recall the basic anatomy behind varicocele side preference.
A varicocele is dilation of the pampiniform venous plexus around the testis. The left testicular vein drains into the left renal vein at a right angle and is also longer than the right testicular vein, which drains more directly into the inferior vena cava at an oblique angle. The right angle drainage on the left raises venous pressure there, so the left side is affected far more often, roughly 80 to 90 percent of cases.

Step 3: Check each statement against this.
Option A says varicocele is more common on the right side. This is the opposite of the truth, since the left side is the common one, so option A is false.

Step 4: Confirm the other statements are true.
Option B, association with infertility, is correct, varicocele is a well known and treatable cause of male subfertility due to raised scrotal temperature and impaired sperm production. Option C is correct too,

a new varicocele appearing on the left in an older man, especially one that does not empty on lying down, can be caused by a renal tumor blocking the left renal vein, so it should always prompt a look for a retroperitoneal or renal mass. Option D is also accepted teaching, that about 10 percent of varicocele cases are bilateral.

Step 5: Final answer.
Since options B, C, and D are true and only option A is false, the answer to this NOT true question is

More common on the right side

Quick Tip: Varicocele is more common on the left side, not the right, due to venous drainage anatomy.

168. What is NOT true about torsion of the testes?

(A) Absence of flow on Doppler clinches diagnosis

(B) Presence of pyuria assists the diagnosis

(C) The opposite side testis should be fixed

(D) Raising the testis worsens the pain

Correct Answer: (B) Presence of pyuria assists the diagnosis

Solution:

Step 1: Understand the question format again.
This is another NOT true question about testicular torsion, so three statements are accurate and one is the false one we need to pick.

Step 2: Recall the basics of testicular torsion.
Torsion happens when the testis twists on its spermatic cord, cutting off its own blood supply. It is a surgical emergency, since the testis can become non viable within a few hours if not untwisted.

Step 3: Check the Doppler statement.
Option A says absent flow on Doppler ultrasound clinches the diagnosis. This is true, colour Doppler showing no blood flow to the testis in a patient with sudden severe scrotal pain is strong evidence of torsion, though a clinically obvious torsion should not wait for imaging if it delays surgery.

Step 4: Check the pyuria statement.
Option B says presence of pyuria, pus cells in urine, assists the diagnosis of torsion. This is false. Torsion is a purely vascular, mechanical problem, it does not cause pus cells in the urine. Pyuria instead points toward an infective cause of scrotal pain, such as epididymo-orchitis or a urinary tract infection, and is actually one of the ways doctors try to tell torsion apart from infection. So pyuria argues against torsion, it does not assist its diagnosis.

Step 5: Check the remaining two statements.
Option C, fixing the opposite testis, is true and important, since the anatomical defect, a bell clapper deformity, that allowed one testis to twist is usually present on both sides, so the other testis is fixed at the same operation to prevent it twisting later. Option D, that raising the testis worsens the pain, is also a recognised clinical sign, this is the basis of a negative Prehn's sign, where lifting the scrotum does not relieve pain in torsion, unlike in epididymitis, where lifting the testis, a positive Prehn's sign, tends to relieve pain.

Step 6: Final answer.

Since A, C, and D are all true and only B is false, the answer is

Presence of pyuria assists the diagnosis

Quick Tip: Pyuria points away from torsion, not toward it, since torsion is a vascular, not infective, problem.

169. What is NOT true about congenital PUJ (pelviureteric junction) obstruction?

(A) It is due to compression by an aberrant vessel rather than due to intrinsic causes

(B) Retrograde pyelography is useful to find the site of obstruction

(C) Whitaker's formula is useful for classification and treatment assessment

(D) Antenatal diagnosis is possible

Correct Answer: (A) It is due to compression by an aberrant vessel rather than due to intrinsic causes

Solution:

Step 1: Understanding the Question:

The question asks which statement about congenital pelviureteric junction (PUJ) obstruction is FALSE. PUJ obstruction blocks urine flow from the renal pelvis into the ureter, and it can come from a defect in the ureter wall itself (intrinsic) or from pressure by an outside structure (extrinsic), such as a lower pole crossing vessel.

Step 2: Key Formula or Approach:

In children, most cases of congenital PUJ obstruction come from an intrinsic problem, a segment of ureter with poor muscle development (an adynamic segment) that fails to carry urine forward normally. An aberrant crossing vessel compressing the PUJ is a real cause too, but it is seen less often than the intrinsic type, and it is more typical in older children and adults.

Step 3: Detailed Explanation:

Check each statement against this fact.

"Due to compression by an aberrant vessel rather than due to intrinsic causes": this reverses the real pattern. Intrinsic causes are more common in congenital PUJ obstruction, not vascular compression. This statement is false.

"Retrograde pyelography is useful to find the site of obstruction": true. Dye passed up the ureter outlines the pelvis and ureter from below and marks exactly where the narrowing starts.

"Whitaker's formula is useful for classification and treatment assessment": true. The Whitaker test measures pressure and flow across the PUJ and helps decide if the obstruction is bad enough to need surgery.

"Antenatal diagnosis is possible": true. PUJ obstruction is one of the most common causes of antenatal hydronephrosis picked up on routine fetal ultrasound.

Step 4: Final Answer:

Three of the four statements are correct facts about congenital PUJ obstruction. Only the claim that vascular compression outweighs intrinsic causes is wrong, since intrinsic narrowing is the more common cause in children.

Due to compression by an aberrant vessel rather than due to intrinsic causes

Quick Tip: Congenital PUJ obstruction is mostly intrinsic, not from vessel compression.

170. What is best for the diagnosis of a firm, hard, mobile nodule in the right breast of a post menopausal woman?

(A) FNAC

(B) Excision biopsy

(C) Mammography

(D) Needle biopsy

Correct Answer: (B) Excision biopsy

Solution:

Step 1: Understanding the Question:

A postmenopausal woman has a firm, hard, mobile lump in the breast. We need the best way to reach a definite diagnosis, out of FNAC, excision biopsy, mammography, and needle biopsy.

Step 2: Key Formula or Approach:

Any breast lump work up rests on getting actual tissue or cells and checking them under the microscope. Some tests sample only a few cells or part of the lump, so they can miss cancer sitting in an unsampled area, giving a false negative. A test that removes the whole lump gives the surgeon complete tissue to examine, so nothing is missed.

Step 3: Detailed Explanation:

Fine needle aspiration cytology (FNAC) draws out a small sample of cells with a thin needle. It is quick and useful as a first screening step, but because it samples only a tiny part of the lump, it can give a false negative in a hard, fibrous lesion where cells are hard to pull out.

Mammography is an imaging test. It shows the size, shape, and calcification pattern of a lump and raises or lowers suspicion, but it cannot give a tissue diagnosis on its own.

Needle biopsy (core biopsy) takes a thin core of tissue rather than loose cells, which is better than FNAC, but it still samples only part of a hard lump and can miss an area of cancer within a mostly benign looking mass.

Excision biopsy removes the whole lump along with a margin of normal tissue. The complete lump is then checked under the microscope, so no part is left unsampled. This gives the most reliable diagnosis, and for a small, localized lump it treats it in the same step.

In current practice, triple assessment with a core needle biopsy is usually tried first, but when full tissue is still needed to be sure, excision biopsy remains the definitive step.

Step 4: Final Answer:

Because it examines the entire lump rather than a sample, excision biopsy gives the most complete and dependable diagnosis for a discrete, firm, hard nodule in this setting.

Excision biopsy

Quick Tip: A firm, hard, discrete breast lump is best diagnosed by removing and checking the whole lump.

171. Which should NOT be done in a testicular tumor?

(A) High inguinal orchidectomy

(B) High inguinal orchidectomy with chemotherapy

(C) High inguinal orchidectomy and RT

(D) Transcrotal biopsy for tissue diagnosis

Correct Answer: (D) Transcrotal biopsy for tissue diagnosis

Solution:

Step 1: Understanding the Question:
We need to pick the step that should NOT be done when managing a testicular tumor.

Step 2: Key Formula or Approach:
A testicular tumor is removed and diagnosed through the inguinal canal, never through the scrotum. The scrotum drains lymph differently from the retroperitoneal nodes that a testicular tumor actually spreads to, so cutting through the scrotum risks seeding tumor cells into scrotal skin and inguinal lymphatics that were never meant to be exposed to it.

Step 3: Detailed Explanation:
High inguinal orchidectomy is the standard first step for any suspected testicular tumor. The spermatic cord is clamped and divided high near the internal ring, and the testis is delivered and removed through a groin incision, keeping the tumor and its lymphatic pathway intact for safe removal.
Depending on the tumor type and stage, chemotherapy or radiotherapy is added after this operation, so "high inguinal orchidectomy with chemotherapy" and "high inguinal orchidectomy

and RT" are both accepted parts of management, not something to avoid.

Transcrotal biopsy means going through the scrotal skin to sample the tumor. This breaches the barrier between the testis and the scrotal skin and its separate lymphatic drainage, and can seed tumor cells into the scrotal wall and inguinal nodes. If that happens, the surgeon then has to remove the scrotal skin and treat the inguinal nodes too, turning a simple operation into a much bigger one. For this reason a transcrotal approach is avoided, and tissue diagnosis is obtained only through the high inguinal route.

Step 4: Final Answer:

Transcrotal biopsy is the step that should be avoided in a testicular tumor, because of the real risk of local tumor seeding.

Transcrotal biopsy for tissue diagnosis

Quick Tip: Testicular tumor tissue diagnosis and removal go through the groin, never the scrotum.

172. What is NOT likely to be the cause of stridor occurring 2 hours after a thyroidectomy?

(A) Hypocalcemia

(B) Wound hematoma

(C) Tracheomalacia

(D) RLN injury bilaterally

Correct Answer: (A) Hypocalcemia

Solution:

Step 1: Understanding the Question:

Stridor appears just 2 hours after a thyroidectomy, and we need the cause that is NOT likely to explain it at this early time point.

Step 2: Key Formula or Approach:

Post thyroidectomy stridor has several causes, and each has a different typical timing. Matching the cause to how soon it appears after surgery is the key to this question.

Step 3: Detailed Explanation:

Wound hematoma: bleeding into the closed neck space can build up within the first few hours after surgery, pressing on the trachea and causing sudden stridor. This fits a 2 hour timeline, it is an early, even immediate complication.

Bilateral recurrent laryngeal nerve (RLN) injury: if both nerves are cut or badly stretched during surgery, both vocal cords lie close to the midline right after extubation, and stridor from airway narrowing can show up as soon as the patient wakes up. This also fits an early timeline.

Tracheomalacia: a trachea long weakened by a large goiter can cave in once the supporting mass is removed, and this too can show itself early, in the recovery period soon after surgery.

Hypocalcemia: the parathyroid glands are often bruised or removed with the thyroid, and blood calcium drifts down gradually as parathyroid hormone falls after surgery. Symptoms like tingling, cramps, and laryngospasm from hypocalcemia usually appear from about 24 to 72 hours after surgery, not within the first 2 hours, since it takes time for calcium levels to fall low enough to cause symptoms.

Step 4: Final Answer:

Hypocalcemia takes a day or more to drop low enough to cause laryngospasm, so it is not a likely cause of stridor only 2 hours after surgery.

Hypocalcemia

Quick Tip: Hypocalcemia after thyroidectomy takes a day or more to cause symptoms, not 2 hours.

173. What is the likely cause of central cyanosis and oligemic lung fields in a neonate with a normal sized heart?

(A) Pulmonary atresia

(B) TOF

(C) TGA

(D) VSD

Correct Answer: (B) TOF

Solution:

Step 1: Understanding the Question:

A neonate has central cyanosis, oligemic (reduced blood flow) lung fields on X-ray, and a normal sized heart. We need the diagnosis that matches all three findings together.

Step 2: Key Formula or Approach:

Cyanotic congenital heart disease can be sorted by two X-ray clues: heart size, and whether the lung fields look full (plethoric, more blood

flow) or empty (oligemic, less blood flow). Matching these clues narrows the diagnosis quickly.

Step 3: Detailed Explanation:

Tetralogy of Fallot (TOF) causes obstruction to blood flow out of the right ventricle into the lungs (pulmonary stenosis), so less blood reaches the lungs and the fields look oligemic. Because the right ventricle pumps against this obstruction rather than dilating hugely, the heart size on X-ray usually stays normal, giving the classic boot shaped but normal sized heart. This matches the question exactly.

Pulmonary atresia usually shows a more abnormal heart silhouette, and depending on the tricuspid valve findings, the heart is often enlarged rather than staying a normal size.

Transposition of the great arteries (TGA) generally shows increased, plethoric lung fields, not oligemic ones, since the parallel circulation still pushes a large blood volume through the lungs, and the heart classically looks like an "egg on a string," a different shape from a normal sized heart.

Ventricular septal defect (VSD) is a left to right shunt lesion. It causes plethoric lung fields with an enlarged heart from volume overload, and it does not cause cyanosis on its own unless it has progressed to Eisenmenger physiology, which is not the picture here in a neonate.

Step 4: Final Answer:

Cyanosis with oligemic lungs and a normal sized heart is the classic picture of Tetralogy of Fallot.

TOF

Quick Tip: Cyanosis with oligemic lungs and a normal sized heart in a neonate suggests TOF.

174. A child has fever, jaundice, clay colored stools, and biopsy suggests giant cell hepatitis. What is the clinical diagnosis?

(A) Viral hepatitis

(B) Neonatal jaundice and EHBA

(C) Neonatal jaundice and IHBA

(D) Non cirrhotic portal fibrosis

Correct Answer: (B) Neonatal jaundice and EHBA

Solution:

Step 1: Understanding the Question:
A child has fever, jaundice, and clay colored (pale) stools, and the liver biopsy shows giant cell hepatitis. We need the clinical diagnosis that fits this whole picture, not just the biopsy report on its own.

Step 2: Key Formula or Approach:
Giant cell transformation on liver biopsy is a common, nonspecific reaction pattern seen in many causes of neonatal cholestasis, so the biopsy alone does not pin down the exact cause. The clinical clue that narrows it down is the stool color: clay colored, pale (acholic) stools mean bile is not reaching the gut, which points toward a blocked bile drainage pathway rather than a purely inflammatory liver process.

Step 3: Detailed Explanation:
Viral hepatitis in a child usually causes dark urine with normal or only

mildly pale stools, since bile can still reach the gut through an open biliary tree, and it does not typically show true acholic stools. This does not fit the picture here.
Neonatal jaundice with extrahepatic biliary atresia (EHBA) blocks the bile ducts outside the liver, so bile cannot reach the intestine at all, giving persistently clay colored stools. Giant cell hepatitis is a well recognized biopsy finding in this setting, since the liver reacts to the blocked bile flow with giant cell change. Fever and jaundice fit the cholestatic, often complicated course seen in EHBA. This matches the full picture.
Neonatal jaundice with intrahepatic biliary atresia (IHBA) affects the smaller bile ducts inside the liver. Stool color here is usually variable, sometimes normal, sometimes pale, rather than the persistently clay colored stools described in the question.
Non cirrhotic portal fibrosis presents later in life with signs of portal hypertension, such as splenomegaly and variceal bleeding, not with a neonatal picture of fever, jaundice, and clay stools.

Step 4: Final Answer:
Persistent clay colored stools with giant cell hepatitis on biopsy is the classic picture of neonatal jaundice due to extrahepatic biliary atresia.

Neonatal jaundice and EHBA

Quick Tip: Persistent clay colored stools with giant cell hepatitis points to biliary atresia.

175. What is the diagnosis in a case of a 30 year old male with jaundice, unconjugated bilirubinemia, increased urine urobilinogen, negative urine bilirubin, normal direct bilirubin, and normal alkaline phosphatase?

(A) Hemolytic jaundice

(B) Viral hepatitis

(C) Obstructive jaundice

Correct Answer: (A) Hemolytic jaundice

Solution:

Step 1: Understanding the Question:
A 30 year old man has jaundice with a raised unconjugated (indirect) bilirubin, increased urine urobilinogen, negative urine bilirubin, normal direct bilirubin, and normal alkaline phosphatase. We need to name the type of jaundice this pattern fits.

Step 2: Key Formula or Approach:
Jaundice is sorted into hemolytic, hepatocellular, and obstructive types by checking which bilirubin fraction is high, whether bilirubin appears in urine, and whether liver enzymes like alkaline phosphatase are raised. Unconjugated bilirubin is bound to albumin in blood and is not water soluble, so it never appears in urine, while conjugated bilirubin is water soluble and does spill into urine when raised.

Step 3: Detailed Explanation:
In this man, only the unconjugated bilirubin is high, while the direct (conjugated) bilirubin is normal. Since it is unconjugated bilirubin that is raised, and unconjugated bilirubin cannot pass into urine, the urine bilirubin is correctly negative.
More red cells are being broken down than usual, so more bilirubin is

sent to the liver, processed, and excreted into the gut as normal, then turned by gut bacteria into more urobilinogen than usual, some of which is reabsorbed and shows up as increased urine urobilinogen. Normal alkaline phosphatase argues against any blockage of bile flow, since obstruction to bile ducts raises alkaline phosphatase along with conjugated bilirubin, neither of which is seen here.

Viral hepatitis (hepatocellular jaundice) usually raises both conjugated and unconjugated bilirubin, since liver cells that both process and excrete bilirubin are damaged, so conjugated bilirubin and urine bilirubin would typically be raised too. This does not fit, since direct bilirubin here is normal.

Obstructive jaundice raises conjugated bilirubin and alkaline phosphatase, and produces a positive urine bilirubin with reduced urine urobilinogen, since bile cannot reach the gut. This is close to the opposite of the pattern seen here.

Hemolytic jaundice fits perfectly: raised unconjugated bilirubin, normal direct bilirubin, negative urine bilirubin, increased urine urobilinogen, and no bile duct blockage, so alkaline phosphatase stays normal.

Step 4: Final Answer:

This lab pattern, raised unconjugated bilirubin with negative urine bilirubin, increased urobilinogen, and normal direct bilirubin and alkaline phosphatase, is classic for hemolytic jaundice.

Hemolytic jaundice

Quick Tip: Raised unconjugated bilirubin with negative urine bilirubin points to hemolysis.

176. What is the diagnosis in a 65 year old patient with fever, flank pain, and calculi with fat densities?

(A) Xanthogranulomatous pyelonephritis

(B) Renal abscess

(C) Chronic pyelonephritis

(D) Tuberculous kidney

Correct Answer: (A) Xanthogranulomatous pyelonephritis

Solution:

Step 1: Understanding the Question:
A 65 year old patient has fever, pain in the flank, and kidney stones that show fat density on imaging. We need to name the disease that fits this exact picture.

Step 2: Key Concept:
Xanthogranulomatous pyelonephritis (XGP) is a rare, severe form of chronic kidney infection. It destroys the kidney tissue and replaces it with lipid laden (fat filled) macrophages, called foam cells. This fat content is what shows up as fat density areas on CT scan. XGP is almost always linked to an obstructing stone, often a staghorn calculus, along with long standing infection, commonly by Proteus or E. coli.

Step 3: Detailed Explanation:
The three clues, fever, flank pain, and a stone with fat density, point straight to XGP. On CT, XGP classically shows an enlarged kidney with a central stone, dilated calyces filled with pus and debris, and surrounding fat dense material, sometimes called the "bear paw" sign. Renal abscess also causes fever and flank pain, but it does not

typically show a fat containing mass around a stone, it shows a fluid filled cavity with a thick wall.
Chronic pyelonephritis causes scarring and a small shrunken kidney with cortical thinning, not a fat density mass.
Tuberculous kidney causes calcification, cavitation, and a "putty kidney" in advanced cases, but not fat density change.

Step 4: Final Answer:
The fat density change around an obstructing calculus with fever and flank pain is the hallmark of xanthogranulomatous pyelonephritis.

Quick Tip: Fat-dense areas around an obstructing stone with chronic infection point to a specific destructive kidney disease.

177. What is the likely diagnosis in a case with renal calcification, irregular bladder wall outline, and hematuria?

(A) Tuberculosis

(B) Schistosomiasis

(C) Amyloidosis

(D) Hunner's cystitis

Correct Answer: (B) Schistosomiasis

Solution:

Step 1: Understanding the Question:
The findings given are calcification in the renal tract, an irregular outline of the bladder wall, and blood in the urine. We need the disease that produces this triad.

Step 2: Key Concept:
Urinary schistosomiasis (bilharzia), caused by Schistosoma haematobium, is a parasitic infection that deposits calcified eggs in the wall of the bladder and lower ureters. Over years this produces patchy calcification, a thickened and irregular bladder wall, and chronic hematuria (blood in urine) from mucosal ulceration.

Step 3: Detailed Explanation:
On imaging, chronic schistosomiasis gives a classic "calcified bladder" sign, a fine rim of calcium outlining an irregular, sometimes contracted bladder, plus calcification that can extend up the ureters and into the kidney collecting system. Hematuria is a very common early symptom, caused by the eggs eroding through the bladder mucosa.
Renal or urinary tuberculosis also causes calcification and hematuria, but the bladder change in TB is usually a small, thick walled, contracted "thimble bladder" rather than the fine eggshell calcification of schistosomiasis, and TB more often shows calyceal or ureteric strictures.
Amyloidosis of the urinary tract does not typically calcify the bladder wall in this pattern.
Hunner's cystitis (interstitial cystitis) causes bladder ulcers and pain but not this kind of calcification.

Step 4: Final Answer:
The combination of renal and bladder calcification with an irregular bladder outline and hematuria is the classic picture of urinary schistosomiasis.

Quick Tip: Chronic parasitic egg deposits in the bladder wall cause calcification and hematuria over years.

178. In India, sentinel surveillance is done for the following diseases/conditions, EXCEPT

(A) Hepatitis B

(B) Diarrhea

(C) Acute Flaccid Paralysis

(D) HIV

Correct Answer: (B) Diarrhea

Solution:

Step 1: Understanding the Question:
We need to pick the one condition among the four that is NOT tracked through India's sentinel surveillance system.

Step 2: Key Concept:
Sentinel surveillance means collecting disease data from a fixed network of selected reporting sites (sentinel sites), rather than from every health facility in the country. India runs sentinel surveillance for a specific set of diseases where routine reporting is not detailed enough, mainly HIV, hepatitis B and C, other sexually transmitted infections, and vaccine preventable diseases tracked for eradication or elimination goals, such as Acute Flaccid Paralysis (AFP) for polio.

Step 3: Detailed Explanation:
HIV sentinel surveillance is one of the oldest and best known sentinel

systems in India, run through antenatal clinics, blood banks, and high risk group clinics.
Hepatitis B (and C) sentinel surveillance is carried out at selected sites to track trends in viral hepatitis.
AFP surveillance is a mainstay of the polio eradication program, every case of sudden floppy weakness in a child under 15 is reported and investigated as a sentinel event.
Diarrheal disease, in contrast, is tracked through routine disease surveillance under programs like the Integrated Disease Surveillance Programme (IDSP), which collects case counts from all reporting units, not from a limited sentinel network built specifically for diarrhea.

Step 4: Final Answer:
Among the four, diarrhea is the condition that is not part of India's dedicated sentinel surveillance system.

Quick Tip: Sentinel surveillance uses select fixed sites for a few specific diseases, not routine reporting.

179. What is NOT true with regard to 'triple' stones?

(A) Struvite stones are composed of triple phosphate

(B) They are called staghorn calculi when they are formed in the renal pelvis

(C) Form in acidic urine

(D) Associated with UTI

Correct Answer: (C) Form in acidic urine

Solution:

Step 1: Understanding the Question:
"Triple" stones is another name for struvite stones. We need the one statement about them that is false.

Step 2: Key Concept:
Struvite stones are made of magnesium ammonium phosphate, which is why they are called triple phosphate stones (three components: magnesium, ammonium, and phosphate). They form only in the presence of urease producing bacteria, most commonly Proteus, which split urea into ammonia and raise the urine pH. This means struvite stones need alkaline urine to form, not acidic urine.

Step 3: Detailed Explanation:
Statement (A), struvite stones are composed of triple phosphate, is a correct definition of the stone.
Statement (B), they are called staghorn calculi when formed in the renal pelvis, is also correct. Because urease splitting bacteria keep making alkaline, ammonia rich urine, the stone keeps growing to fill the renal pelvis and calyces, forming a branching staghorn shape.
Statement (C), form in acidic urine, is false. Struvite crystals are unstable in acidic urine and only precipitate when the urine pH rises above about 7, which happens because of urease producing organisms.
Statement (D), associated with UTI, is correct, since struvite stones cannot form without a urease producing urinary tract infection driving the alkaline environment.

Step 4: Final Answer:

The false statement is that triple stones form in acidic urine, they actually need alkaline urine to form.

Quick Tip: Struvite (triple phosphate) stones need alkaline urine from urease producing bacteria to form.

180. A tumor/mass lesion of the kidney extends into the IVC, with Gerota's fascia intact. All the following are true, EXCEPT

(A) IVC invasion is a contraindication for surgery

(B) Chest X-ray to rule out pulmonary metastases

(C) Pre-op radiotherapy is not indicated

(D) Pre-op biopsy is not indicated

Correct Answer: (A) IVC invasion is a contraindication for surgery

Solution:

Step 1: Understanding the Question:
A kidney tumor has grown into the inferior vena cava (IVC), but the fibrous covering of the kidney, Gerota's fascia, is still intact, meaning the tumor has not spread outside the kidney's capsule of fat and fascia. We need the one statement that is false about managing this case.

Step 2: Key Concept:
This picture describes locally advanced renal cell carcinoma (RCC) with a tumor thrombus in the IVC but no extracapsular spread. RCC has a well known tendency to grow along veins as a solid tumor thrombus rather than through blood borne spread, so even a large

IVC thrombus can often be removed surgically along with the kidney, especially when Gerota's fascia keeps the disease contained.

Step 3: Detailed Explanation:
Statement (A), IVC invasion is a contraindication for surgery, is false. Radical nephrectomy with IVC thrombectomy (removing the tumor thrombus from the vena cava, sometimes needing cardiopulmonary bypass for very high thrombi) is standard treatment and can be curative, so IVC involvement alone does not rule out surgery.
Statement (B), chest X-ray to rule out pulmonary metastases, is correct practice. RCC commonly spreads to the lungs, so a chest X-ray or CT chest is a routine part of staging before planning surgery.
Statement (C), pre-op radiotherapy is not indicated, is correct. RCC is relatively radioresistant, and radiotherapy has no proven role in shrinking the tumor before surgery.
Statement (D), pre-op biopsy is not indicated, is correct in most cases. When imaging is typical for RCC and the plan is surgical removal anyway, a biopsy adds risk (bleeding, tumor seeding) without changing management, so it is usually skipped.

Step 4: Final Answer:
The false statement is that IVC invasion is a contraindication for surgery, since IVC thrombectomy along with nephrectomy is the standard curative approach.

Quick Tip: RCC with an IVC thrombus, but no spread outside Gerota's fascia, is still resectable with thrombectomy.

181. Which of these is a criterion for conservative treatment in a ureteric calculus?

(A) Infection and hydronephrosis present

(B) Size under 6 mm

(C) No movement for 2 weeks

(D) Highly symptomatic

Correct Answer: (B) Size under 6 mm

Solution:

Step 1: Understanding the Question:
We need to pick the feature of a ureteric stone that supports watching and waiting (conservative treatment) instead of an active procedure.

Step 2: Key Concept:
Conservative, or expectant, management of a ureteric stone means giving pain relief, fluids, and sometimes medical expulsive therapy, and waiting for the stone to pass on its own. The main factor that decides whether a stone is likely to pass by itself is its size, since small stones can travel through the narrow parts of the ureter while larger ones get stuck.

Step 3: Detailed Explanation:
Stones under about 5 to 6 mm have a high chance, roughly 70 percent, of passing on their own within a few weeks, so small size is the classic criterion for conservative treatment.
Infection with hydronephrosis (option A) is actually a warning sign for urgent drainage, an infected obstructed kidney is a urological emergency that needs prompt decompression, not watchful waiting.
No movement for 2 weeks (option C) suggests the stone is impacted

and not progressing, which favors intervention rather than continued conservative care.
Being highly symptomatic (option D) with severe, uncontrolled pain also pushes toward active treatment rather than conservative management.

Step 4: Final Answer:
A stone size under 6 mm is the recognized criterion favoring conservative treatment of a ureteric calculus.

Quick Tip: Small stones under about 6 mm have a high chance of passing on their own.

182. What is NOT true about a urinary bladder calculus?

(A) Primary stones rare in Indian children

(B) Transurethral removal is possible

(C) Most are radio opaque

(D) KUB clinches the diagnosis

Correct Answer: (A) Primary stones rare in Indian children

Solution:

Step 1: Understanding the Question:
We need the one statement about bladder stones that is false.

Step 2: Key Concept:
Bladder stones are grouped as primary (forming directly in the bladder, usually from diet related factors) or secondary (forming

because of an underlying cause such as outlet obstruction, a foreign body, or infection). India, especially the northern "stone belt" regions, has long been known for a high rate of primary bladder stones in children, linked to a low protein, low phosphate diet in early childhood.

Step 3: Detailed Explanation:

Statement (A), primary stones are rare in Indian children, is false. In fact, endemic primary vesical (bladder) calculus disease in children is a well documented problem in parts of India, historically one of the more common urological problems in young boys from certain regions.

Statement (B), transurethral removal is possible, is correct. Many bladder stones, especially smaller ones, can be fragmented and removed endoscopically through the urethra (cystolitholapaxy), avoiding open surgery.

Statement (C), most are radio opaque, is correct. The majority of bladder stones contain calcium and show up on a plain X-ray, unlike pure uric acid stones which are radiolucent.

Statement (D), KUB (kidney, ureter, bladder X-ray) clinches the diagnosis, is correct, since a plain KUB film readily shows most bladder calculi given their radio-opaque nature.

Step 4: Final Answer:

The false statement is that primary stones are rare in Indian children, since primary bladder stones are in fact a recognized, relatively common problem in Indian children in endemic regions.

Quick Tip: Endemic primary bladder stone disease in children is well documented in parts of India.

183. What investigation should be done for a prostatic nodule in a 60 year old man?

(A) Expressed prostatic secretion analysis

(B) CT scan pelvis

(C) Transrectal ultrasound (TRUS)

(D) MRI

Correct Answer: (C) Transrectal ultrasound (TRUS)

Solution:

Step 1: Understand the clinical scenario.
A prostatic nodule found on digital rectal exam in an older man raises concern for prostate cancer. The next step is imaging that can show the nodule clearly and also guide a biopsy.

Step 2: Recall the imaging options for the prostate.
CT scan and MRI can show the prostate but do not give the fine local detail needed to target a small nodule, and CT is poor at showing the zonal anatomy of the gland.

Step 3: Explain why transrectal ultrasound is preferred.
Transrectal ultrasound (TRUS) places the probe close to the prostate through the rectum, so it gives a clear, high resolution picture of the gland. It shows the nodule directly and lets the surgeon guide a needle into it for biopsy in the same sitting, so it works as both a diagnostic tool and a biopsy guide.

Step 4: Rule out the other options.

(A) Expressed prostatic secretion analysis: This checks for infection (prostatitis), not cancer, and cannot assess a nodule.

(B) CT scan pelvis: Poor at showing prostate zonal detail and cannot guide an accurate targeted biopsy.

(D) MRI: Useful for staging once cancer is already confirmed, but it is not the first test for a newly found nodule, and it does not double as a biopsy guide the way TRUS does.

Step 5: Final answer.
The correct first investigation for a prostatic nodule is transrectal ultrasound, since it images the gland closely and guides a needle biopsy in one visit.

Transrectal ultrasound (TRUS)

Quick Tip: Think about which test both images the prostate closely and can guide a biopsy needle.

184. What is NOT true about carcinoma penis?

(A) Circumcision any time before puberty is 100% protective

(B) Erythroplasia of Queyrat is premalignant

(C) Occurs in unhygienic conditions

(D) Presents with inguinal node enlargement in 50% of the cases

Correct Answer: (A) Circumcision any time before puberty is 100% protective

Solution:

Step 1: Recall the risk factors for carcinoma penis.
Carcinoma penis is strongly linked to phimosis, poor hygiene, and the buildup of smegma under the foreskin. It is rare in men circumcised at birth.

Step 2: Understand the true protective effect of circumcision.
Neonatal circumcision, done at birth, removes the foreskin before smegma and chronic irritation can act on the glans, and this gives close to complete protection from carcinoma penis.
Circumcision performed later, even before puberty, does not carry this same near total protection, since some exposure to smegma and chronic irritation may already have occurred by then.

Step 3: Analyze option (A).
The statement claims circumcision at any time before puberty is 100% protective. This is not accurate, since only circumcision done at birth carries near complete protection. Doing it later, though still helpful, does not carry that same guarantee.
So option (A) is the false statement.

Step 4: Analyze option (B).
Erythroplasia of Queyrat is a red, velvety patch on the glans, and it is a recognized premalignant lesion (a form of carcinoma in situ) that can turn into invasive carcinoma penis. This statement is true.

Step 5: Analyze option (C).
Poor hygiene lets smegma collect under the foreskin, and this chronic irritation is a well known risk factor for carcinoma penis. This statement is true.

Step 6: Analyze option (D).
Inguinal lymph nodes are the first site of spread for carcinoma penis, and enlarged inguinal nodes are found in about half of patients at presentation, though not all enlarged nodes mean actual tumor spread since some are just reactive from infection. This statement is true.

Step 7: Final answer.
The false statement is that circumcision any time before puberty is 100% protective, since only circumcision at birth carries that level of protection.

Circumcision any time before puberty is 100% protective - FALSE

Quick Tip: Only circumcision at birth gives strong protection; later circumcision does not carry the same guarantee.

185. Which type of malignancy occurs in longstanding multinodular goitre?

(A) Papillary

(B) Follicular

(C) Anaplastic

(D) Medullary

Correct Answer: (B) Follicular

Solution:

Step 1: Recall what a longstanding multinodular goitre is.
A multinodular goitre (MNG) is an enlarged thyroid gland with many nodules, often from long term iodine deficiency or chronic stimulation of the gland. Over many years, a small number of these glands develop cancer within one of the nodules.

Step 2: Recall the malignancy linked with longstanding MNG.
Follicular carcinoma is the thyroid cancer classically described as arising in a background of longstanding multinodular goitre, especially in areas of iodine deficiency. The chronic overstimulation of follicular cells over years favors this pattern of malignant change.

Step 3: Contrast with the other thyroid cancers.

(A) Papillary carcinoma: This is the most common thyroid cancer overall and is linked mainly to radiation exposure, not to longstanding goitre. It usually arises in an otherwise normal gland.

(C) Anaplastic carcinoma: This is a rare, aggressive cancer seen mostly in elderly patients, and while it can rarely follow a longstanding goitre, it is not the classic association being tested here.

(D) Medullary carcinoma: This arises from the parafollicular C cells and is linked to MEN 2 syndromes, not to iodine deficient multinodular goitre.

Step 4: Final answer.
The malignancy classically linked with longstanding multinodular goitre is follicular carcinoma.

Follicular carcinoma

Quick Tip: Think of the thyroid cancer linked with iodine deficient, chronically enlarged multinodular glands.

186. Which condition may feature pulsatile varicose veins?

(A) Tricuspid regurgitation

(B) Deep vein thrombosis

(C) Klippel-Trenaunay syndrome

(D) Right ventricular failure

Correct Answer: (C) Klippel-Trenaunay syndrome

Solution:

Step 1: Understand what pulsatile varicose veins mean.
Ordinary varicose veins are dilated, tortuous veins with no true pulsation, since venous pressure is low. A pulsatile varicose vein means the vein wall expands and relaxes with each heartbeat, which points to some abnormal high pressure flow reaching the venous system.

Step 2: Recall Klippel-Trenaunay syndrome.
Klippel-Trenaunay syndrome is a congenital vascular malformation with a triad of a port wine capillary stain, varicose or malformed veins, and overgrowth (hypertrophy) of the affected limb. Some of these malformations carry small abnormal connections between arteries and veins, and this abnormal arterial pressure can be transmitted into the varicose veins, making them pulsatile.

Step 3: Rule out the other options.

(A) Tricuspid regurgitation: This can transmit a pulse wave down the inferior vena cava into the femoral vein and cause a pulsatile saphena varix at the groin. That is a separate, specifically named clinical sign rather than the general cause of pulsatile varicosities asked for here.

(B) Deep vein thrombosis: This causes swelling and pain in the leg from an obstructed deep vein, not pulsatile varicosities.

(D) Right ventricular failure: This raises venous pressure and causes swelling of the legs and neck veins, but it does not make superficial varicose veins pulsatile.

Step 4: Final answer.
The condition among these options that may feature pulsatile varicose veins is Klippel-Trenaunay syndrome, through its abnormal vascular connections.

Klippel-Trenaunay syndrome

Quick Tip: Look for the congenital vascular malformation syndrome with limb overgrowth and abnormal vessel connections.

187. What may cause local gigantism with varicosities?

(A) Arteriovenous (AV) fistulae

(B) Deep vein thrombosis

(C) Acromegaly

(D) Osteosarcoma

Correct Answer: (A) Arteriovenous (AV) fistulae

Solution:

Step 1: Understand local gigantism.
Local gigantism means one limb or body part grows larger than normal because of extra blood flow or extra growth signal reaching that part alone, unlike acromegaly which affects the whole body.

Step 2: Recall how an arteriovenous (AV) fistula causes this.
An AV fistula is an abnormal direct connection between an artery and a vein, bypassing the capillary bed. This sends high pressure, high flow arterial blood straight into the venous system of that limb.
The extra blood flow feeds the growing bones and soft tissue of the limb, so the limb grows larger than the other side, a state called local gigantism.
The same high flow also distends and dilates the superficial veins draining the limb, producing varicosities, so both features, limb overgrowth and varicose veins, are explained by one underlying AV fistula.

Step 3: Rule out the other options.

(B) Deep vein thrombosis: This blocks outflow and causes a swollen, painful leg, but it does not cause true bony or soft tissue overgrowth.

(C) Acromegaly: This is caused by excess growth hormone and enlarges the whole body symmetrically, including hands, feet, and jaw, not one limb alone.

(D) Osteosarcoma: This is a bone tumor that causes a local mass and pain, not diffuse limb overgrowth with varicose veins.

Step 4: Final answer.
The cause of local gigantism with varicosities among these options is an arteriovenous fistula.

Arteriovenous (AV) fistula

Quick Tip: Think of what abnormal vessel connection sends extra arterial flow into just one limb.

188. All the following are used as sclerosing agents EXCEPT

(A) Alcohol

(B) Acetic acid

(C) Crysolate

(D) Polidocanol

Correct Answer: (B) Acetic acid

Solution:

Step 1: Recall what a sclerosing agent does.
A sclerosing agent is injected into a varicose vein to irritate its inner

lining, which then triggers clotting and fibrosis, closing the vein permanently. This is the basis of sclerotherapy for varicose veins.

Step 2: List the known sclerosing agents.
Common agents used clinically include absolute alcohol, hypertonic saline, sodium tetradecyl sulphate, sodium morrhuate, chromated glycerin type solutions, and polidocanol. Each of these directly damages the vein wall lining to close it off.

Step 3: Analyze each option.

(A) Alcohol: Absolute alcohol is a recognized sclerosing agent, used to damage the vein lining and induce fibrosis. This is a true sclerosant.

(B) Acetic acid: Acetic acid is not used as a venous sclerosing agent. It is used in other clinical settings instead, such as applying it to the cervix during screening to highlight abnormal tissue, or as an antiseptic wash, not to close off varicose veins.

(C) Crysolate: This agent is used clinically as a sclerosing solution for varicose veins.

(D) Polidocanol: This is one of the most widely used modern sclerosing agents for varicose veins and spider veins. This is a true sclerosant.

Step 4: Final answer.
The agent that is NOT used as a sclerosing agent is acetic acid.

Acetic acid

Quick Tip: Think about which of these agents is used for cervical screening or as an antiseptic, not for closing off veins.

189. Pelviureteric obstruction on the left side in a 33 year old male who presents with fever and infection. All the following are correct EXCEPT

(A) Dismembered pyeloplasty is the treatment of choice

(B) Endoscopic pyeloplasty is contraindicated

(C) Most common cause is an aberrant vessel

(D) Tuberculosis can be a cause

Correct Answer: (C) Most common cause is an aberrant vessel

Solution:

Step 1: Understand pelviureteric junction (PUJ) obstruction.
PUJ obstruction is a block at the point where the renal pelvis narrows into the ureter, so urine cannot drain freely from the kidney. It can be congenital, present from birth, or acquired later from stones, tumors, or infection.

Step 2: Analyze option (A), dismembered pyeloplasty.
The Anderson-Hynes dismembered pyeloplasty is the gold standard operation for PUJ obstruction. The narrow segment is cut out, the pelvis is trimmed, and a fresh wide anastomosis is made to the ureter. This statement is true.

Step 3: Analyze option (B), endoscopic pyeloplasty being contraindicated.

Endoscopic treatment (endopyelotomy) cuts the narrow segment from inside, without removing it or repositioning nearby vessels. It needs a clean, uninfected urinary tract to heal safely, since cutting through infected, inflamed tissue raises the risk of bleeding and urosepsis.
This patient has an obstructed kidney with active fever and infection, so the endoscopic approach is not safe as a first step here, and calling it contraindicated in this setting is a true statement.

Step 4: Analyze option (C), aberrant vessel as the most common cause.
The most common cause of PUJ obstruction is actually an intrinsic problem, an abnormal, poorly contractile (aperistaltic) segment of muscle at the PUJ that fails to push urine forward, present from birth in most congenital cases.
A crossing or aberrant lower pole vessel pressing on the PUJ from outside is a well recognized cause, and it matters because it changes how the pyeloplasty is done, but it is not the single most common cause overall.
So the claim that an aberrant vessel is the most common cause is false, and this is the statement that does not fit with the rest.

Step 5: Analyze option (D), tuberculosis as a cause.
Genitourinary tuberculosis can scar and narrow the ureter at the PUJ as it heals, causing a secondary, acquired PUJ obstruction. This statement is true.

Step 6: Final answer.
Note on the source key: the original answer key for this question was left blank or unclear in the source paper. Working through each statement on current medical grounds, the one that is NOT correct is

that an aberrant vessel is the most common cause of PUJ obstruction, since an intrinsic aperistaltic segment is actually the more common cause.

Most common cause is an aberrant vessel - INCORRECT statement

Quick Tip: Recall whether an intrinsic narrow segment or an outside vessel is the more common cause of PUJ obstruction.

190. A 60 year old hypertensive patient comes with abdominal pain and fusiform dilatation of the abdominal aorta. What could be the most probable etiology?

(A) Marfan's syndrome

(B) Syphilis

(C) Atherosclerosis

(D) Cystic medial necrosis

Correct Answer: (C) Atherosclerosis

Solution:

Step 1: Read the clinical picture.
The patient is 60 years old, has high blood pressure, and has a fusiform (spindle shaped, uniform) swelling of the abdominal aorta. This is the classic picture of an abdominal aortic aneurysm (AAA).

Step 2: Recall the usual cause of AAA.
Most abdominal aortic aneurysms, especially fusiform ones in an elderly hypertensive patient, form because of atherosclerosis. Fatty

plaques build up in the aortic wall, weaken the middle layer, and the artery bulges out over the years. Age, smoking, and high blood pressure all speed this process up.

Step 3: Check why Marfan's syndrome does not fit.
Marfan's syndrome is a connective tissue disorder that weakens the aortic wall through cystic medial necrosis, but it mainly affects the ascending (thoracic) aorta and causes aneurysms at a much younger age, not a 60 year old with a fusiform abdominal swelling.

Step 4: Check why syphilis does not fit.
Syphilitic aortitis damages the vasa vasorum of the aorta and classically causes a saccular aneurysm of the ascending aorta or arch, not the abdominal aorta. This diagnosis has also become rare since antibiotics came into use.

Step 5: Check why cystic medial necrosis does not fit.
Cystic medial necrosis is the tissue change seen in Marfan's syndrome and related disorders, and it too points to the thoracic aorta, not a fusiform abdominal aneurysm in a hypertensive elderly patient.

Step 6: Final answer.
The most probable cause of a fusiform abdominal aortic aneurysm in a 60 year old hypertensive patient is atherosclerosis.

Quick Tip: Fusiform abdominal aortic aneurysm in an elderly hypertensive patient points to a degenerative wall change from fat plaque build up.

191. Which of these is most often secreted by a pheochromocytoma?

(A) Epinephrine

(B) Norepinephrine

(C) Dopamine

(D) Serotonin

Correct Answer: (B) Norepinephrine

Solution:

Step 1: Recall what a pheochromocytoma is.
A pheochromocytoma is a tumor of the chromaffin cells of the adrenal medulla. These are the same cells that normally make and release catecholamines, so the tumor keeps making them, often in large and uncontrolled amounts.

Step 2: Recall which catecholamine dominates.
The normal adrenal medulla makes mostly epinephrine, using the enzyme PNMT to convert norepinephrine into epinephrine. In a pheochromocytoma, tumor cells often lose this efficient conversion step, so norepinephrine builds up and becomes the catecholamine secreted in the largest amount overall, causing sustained high blood pressure.

Step 3: Check why epinephrine is not the best answer.
Epinephrine is still released by many pheochromocytomas and causes the classic episodic palpitation and sweating attacks, but taken across all pheochromocytomas, norepinephrine is secreted more often and in greater total amount, and this is what the question is testing.

Step 4: Check why dopamine is wrong.
Dopamine is the catecholamine precursor and is raised mainly in

malignant or extra-adrenal pheochromocytomas (paragangliomas), not in typical benign adrenal tumors, so it is not the usual main secretion.

Step 5: Check why serotonin is wrong.
Serotonin is not a catecholamine at all. It is the hormone secreted by carcinoid tumors of the gut, which cause flushing and diarrhea, a completely different tumor and a different clinical picture.

Step 6: Final answer.
The catecholamine most often secreted by a pheochromocytoma is norepinephrine.

Quick Tip: Pheochromocytoma cells often skip the last conversion step, so this catecholamine builds up and dominates.

192. In a surgery ward, what is the best method of prevention of post op wound infection in patients, and hence preventing their spread to other patients?

(A) Hand washing prior to and in between patient examination and dressings

(B) Fumigation of the ward

(C) Cleaning of the floor with Ceramide

(D) Vancomycin prophylaxis

Correct Answer: (A) Hand washing prior to and in between patient examination and dressings

Solution:

Step 1: Identify what actually spreads infection between patients.
In a ward, the main route by which one patient's wound germs reach another patient is the hands of the staff examining and dressing wounds. Every time hands touch a wound, dressing, or bed and then touch the next patient, germs can travel along with them.

Step 2: State the single most effective control measure.
Hand washing before touching a patient and again between patients breaks this chain of spread. It is simple, cheap, and backed by strong evidence as the single most effective step in preventing hospital acquired and cross patient wound infection.

Step 3: Check why fumigation of the ward is wrong.
Fumigating a ward with chemical vapors does not reliably lower infection rates, is not proven to reduce surgical wound infection, and exposes staff and patients to unnecessary toxic fumes. It has been abandoned as an infection control method.

Step 4: Check why floor cleaning is wrong.
The floor is a low risk surface for spreading wound infection, since it is not directly touched during wound care. Cleaning it matters for general hygiene, but it does not stop the person to person spread that happens through hands.

Step 5: Check why routine vancomycin prophylaxis is wrong.
Giving vancomycin to every patient as a routine measure is not standard practice. It does not address the actual route of spread, and overusing a strong antibiotic like this only breeds resistant bacteria, making infections harder to treat later.

Step 6: Final answer.

Hand washing before and between patient contact is the best method to prevent and stop the spread of post op wound infection.

Quick Tip: Think about what actually travels from one wound dressing to the next patient.

193. What is the preferred treatment for a solitary thyroid nodule?

(A) Hemithyroidectomy

(B) Total thyroidectomy

(C) Subtotal thyroidectomy

(D) Enucleation

Correct Answer: (A) Hemithyroidectomy

Solution:

Step 1: Understand what a solitary thyroid nodule needs.
A solitary nodule may be benign or malignant, and this usually cannot be settled with complete certainty before surgery even after needle aspiration. So the operation chosen has to both treat the lump and give a clear tissue diagnosis.

Step 2: State the preferred operation.
Hemithyroidectomy, removing the entire lobe carrying the nodule along with the isthmus, is the preferred initial surgery. It removes the whole area of concern with a clear margin and gives the pathologist the full lobe to examine, so the diagnosis is reliable.

Step 3: Explain what happens after histology.

If the final report shows a benign nodule, hemithyroidectomy alone is enough treatment. If it turns out to be a cancer needing more surgery, a completion thyroidectomy can be done as a planned second operation.

Step 4: Check why total thyroidectomy is wrong here.
Removing the whole gland straight away is too aggressive as a first step when the nature of a single nodule is not yet known, and it carries added risk to both parathyroid glands and both recurrent laryngeal nerves for no proven benefit at this stage.

Step 5: Check why subtotal thyroidectomy is wrong here.
Subtotal thyroidectomy, leaving behind a remnant of tissue on both sides, is used for conditions like multinodular goiter or Graves' disease, not for a single well defined nodule, and it would leave abnormal tissue behind on the side of the nodule.

Step 6: Check why enucleation is wrong here.
Simply shelling out the nodule without taking a surrounding margin of normal lobe risks leaving tumor cells behind if it is malignant, and it is not accepted practice for thyroid surgery.

Step 7: Final answer.
Hemithyroidectomy is the preferred treatment for a solitary thyroid nodule.

Quick Tip: Pick the operation that removes the whole nodule with a margin and also gives a full tissue diagnosis.

194. All of the following pass behind the ischial spine EXCEPT?

(A) Obturator nerve

(B) Pudendal nerve

(C) Internal pudendal vessels

(D) Nerve to obturator internus

Correct Answer: (A) Obturator nerve

Solution:

Step 1: Recall the exit route through the greater sciatic foramen.
Several pelvic nerves and vessels leave the pelvis through the greater sciatic foramen, below the piriformis muscle, and then hook around the back of the ischial spine and the sacrospinous ligament before entering the perineum through the lesser sciatic foramen.

Step 2: List the structures that make this hook around the ischial spine.
The pudendal nerve, the internal pudendal vessels, and the nerve to obturator internus all follow this exact path, passing behind the ischial spine on their way into the pudendal (Alcock's) canal in the perineum.

Step 3: Trace the obturator nerve separately.
The obturator nerve comes from the lumbar plexus and runs along the lateral pelvic wall, then leaves the pelvis through the obturator canal at the upper part of the obturator foramen, at the front of the pelvis, nowhere near the ischial spine.

Step 4: Compare the two exit routes.
The first three structures share one exit route around the ischial

spine, while the obturator nerve uses a completely different anterior exit, so it is the odd one out.

Step 5: Final answer.
The obturator nerve does not pass behind the ischial spine.

Quick Tip: Three of these structures re-enter the perineum through the lesser sciatic foramen; one leaves the pelvis through the obturator canal instead.

195. Injury to which nerve during a herniorrhaphy may cause paresthesia at the root of the scrotum and base of the penis?

(A) Ilioinguinal

(B) Pudendal

(C) Genitofemoral

(D) Iliohypogastric

Correct Answer: (A) Ilioinguinal

Solution:

Step 1: Recall the course of the ilioinguinal nerve.
The ilioinguinal nerve runs through the inguinal canal alongside the spermatic cord and leaves through the superficial inguinal ring. It lies quite superficial in the operative field used for inguinal hernia repair, so it is easily caught in a suture or stretched by a retractor.

Step 2: Recall what it supplies.
It carries sensation from the skin at the root of the penis, the upper part of the scrotum (or the mons pubis and labia majora in a woman),

and the adjoining upper inner thigh. Damage to it during herniorrhaphy shows up as numbness, tingling, or burning pain in exactly this area.

Step 3: Check why the pudendal nerve is wrong.
The pudendal nerve travels through the pudendal canal in the perineum and supplies the deeper perineal structures and external genitalia through a different route altogether. It is not exposed in the field of a standard inguinal hernia operation.

Step 4: Check why the genitofemoral nerve is wrong.
Its genital branch runs with the spermatic cord and mainly supplies the cremaster muscle, so injury to it classically causes loss of the cremasteric reflex, not the sensory change described here at the root of the scrotum and penis.

Step 5: Check why the iliohypogastric nerve is wrong.
This nerve supplies the skin over the lower abdomen just above the pubic bone, a region above and separate from the root of the scrotum and penis, so it does not match the symptom described.

Step 6: Final answer.
Injury to the ilioinguinal nerve during herniorrhaphy causes paresthesia at the root of the scrotum and base of the penis.

Quick Tip: This nerve runs in the inguinal canal itself and exits through the superficial ring, right in the surgical field of a hernia repair.

196. Which of these statements regarding the kidney is NOT correct?

(A) Right kidney is preferred to the left for transplantation

(B) Right kidney is at a lower level than the left

(C) Right kidney is related to the duodenum in the anteromedial aspect

(D) Right renal vein is shorter than the left

Correct Answer: (A) Right kidney is preferred to the left for transplantation

Solution:

Step 1: Check the statement about kidney level.
The right kidney sits slightly lower than the left kidney because the large right lobe of the liver pushes down on it from above. This statement is correct.

Step 2: Check the statement about the duodenum.
The second (descending) part of the duodenum lies against the anteromedial surface of the right kidney, close to its hilum. This statement is correct.

Step 3: Check the statement about renal vein length.
The inferior vena cava lies close to the right side of the body, so the right renal vein only has a short distance to travel and is shorter than the left renal vein, which has to cross the midline. This statement is correct.

Step 4: Check the statement about transplantation.
For a live kidney transplant, the left kidney is usually the preferred one to remove and use, mainly because its longer renal vein makes the vessel much easier to stitch onto the recipient's vessels. Preferring the right kidney over the left is therefore not correct as a general

statement.

Step 5: Final answer.
The statement that is NOT correct is that the right kidney is preferred to the left for transplantation. In practice, the opposite is true.

Quick Tip: Surgeons prefer the kidney with the longer renal vein for an easier vascular join in the recipient.

197. All of the following enzyme deficiencies, except one, may cause lens opacities and mental retardation in a child. Which one is the exception?

(A) Galactokinase

(B) Galactose UDP 1 transferase

(C) Galactose 4 epimerase

(D) Lactase

Correct Answer: (D) Lactase

Solution:

Step 1: Understanding the Question.
The question lists four enzymes and asks which one, when deficient, does NOT cause lens opacities (cataracts) and mental retardation in a child. Three of the four enzymes work in galactose metabolism; only one has nothing to do with galactose handling.

Step 2: Trace the galactose pathway.
Dietary lactose is split into glucose and galactose. Galactose is then processed in three steps: galactokinase turns galactose into galactose-

1-phosphate, galactose-1-phosphate uridyltransferase swaps it onto UDP-glucose to make UDP-galactose, and UDP-galactose-4-epimerase converts UDP-galactose back to UDP-glucose so it can enter normal glucose metabolism.

Step 3: Look at what happens when each galactose enzyme is missing.

A block at the transferase step (classic galactosemia) lets galactose-1-phosphate and galactose pile up. Some of that galactose is turned into galactitol by aldose reductase, and galactitol collects in the lens and draws in water, causing cataracts. The same buildup damages the liver and brain, causing mental retardation if the child stays on milk. A block at the epimerase step, in its severe generalized form, causes a similar pattern of cataracts and mental delay, because galactose and galactose-1-phosphate still build up. A block at the galactokinase step also raises blood galactose and lets galactitol form, so cataracts appear, and severe or late-treated cases carry some neurological risk too.

Step 4: Look at lactase.

Lactase is a different kind of enzyme. It sits on the brush border of the small intestine and splits lactose into glucose and galactose in the first place. If lactase is missing, lactose is never broken down and never absorbed, so galactose never even enters the blood in any extra amount. The child gets bloating, gas, and diarrhea from unabsorbed lactose reaching the colon, not lens opacities or mental retardation.

Step 5: Final Answer.

Since galactokinase, the transferase, and the epimerase all sit inside the galactose pathway and can each raise galactose or galactitol enough to affect the lens and brain, lactase is the odd one out.

Lactase deficiency does not cause lens opacities and mental retardation

Quick Tip: Only lactase sits outside the galactose metabolism pathway; the other three enzymes all handle galactose itself.

198. Dietary fibers are degraded by colonic bacteria to form which of the following?

(A) Butyrates

(B) Glycerol

(C) Sucrose

(D) Free radicals

Correct Answer: (A) Butyrates

Solution:

Step 1: Understanding the Question.
Dietary fiber (mostly non-starch polysaccharides like cellulose, hemicellulose, and pectin) cannot be digested by human enzymes in the small intestine. The question asks what colonic bacteria turn this fiber into once it reaches the large bowel.

Step 2: Recall what colonic bacteria do to fiber.
Bacteria in the colon ferment undigested fiber without oxygen. This fermentation breaks the long fiber chains down into short-chain fatty acids (SCFAs), mainly acetate, propionate, and butyrate, along with gases like hydrogen, methane, and carbon dioxide.

Step 3: Focus on butyrate.

Butyrate is the SCFA that matters most for the colon itself. Colonocytes (the cells lining the large intestine) use butyrate as their main fuel, ahead of glucose. Butyrate also helps keep the colon lining healthy and is linked to lower colon cancer risk.

Step 4: Rule out the other options.

(B) Glycerol: Glycerol comes from fat breakdown, not from fiber fermentation. Incorrect.

(C) Sucrose: Sucrose is a simple sugar absorbed in the small intestine; it is not a fermentation product of fiber. Incorrect.

(D) Free radicals: Free radicals are reactive oxygen species from oxidative processes, not a normal fermentation output of colonic bacteria. Incorrect.

Step 5: Final Answer.
Colonic bacterial fermentation of dietary fiber mainly yields short-chain fatty acids, of which butyrate is the key one used by colon cells.

Butyrates

Quick Tip: Colonic bacteria ferment fiber into short-chain fatty acids; butyrate feeds the colon cells.

199. What is the possible cause for gout in a patient who has a glucose-6-phosphatase deficiency?

(A) Increased synthesis of pentoses

(B) Increased accumulation of sorbitol

(C) Increased synthesis of glycerol

(D) Decreased function of Krebs cycle

Correct Answer: (A) Increased synthesis of pentoses

Solution:

Step 1: Understanding the Question.

Glucose-6-phosphatase deficiency is Von Gierke disease (glycogen storage disease type I). This enzyme normally converts glucose-6-phosphate to free glucose in the liver, the last step before glucose leaves the liver into the blood. The question asks why patients with this deficiency develop gout, that is too much uric acid.

Step 2: See what happens to glucose-6-phosphate when the enzyme is missing.

Since glucose-6-phosphate cannot be turned into free glucose, it piles up inside liver cells. This trapped glucose-6-phosphate gets pushed down other available pathways instead, mainly glycolysis and the pentose phosphate pathway (also called the hexose monophosphate shunt).

Step 3: Follow the pentose phosphate route to uric acid.

The pentose phosphate pathway converts glucose-6-phosphate into ribose-5-phosphate. Extra ribose-5-phosphate is then used to make more phosphoribosyl pyrophosphate (PRPP), the starting material for purine synthesis. More PRPP drives more purine synthesis, and purines break down into uric acid. This raises blood uric acid and causes gout.

Step 4: Add the second contributing factor.
Because the liver cannot release free glucose, the body also runs more glycolysis for energy, and this raises lactate. High lactate competes with uric acid for the same excretion route in the kidney tubules, so less uric acid is cleared out. Both the extra production, through pentose and purine synthesis, and the reduced clearance, through lactic acidosis, push uric acid levels up together.

Step 5: Rule out the other options.

(B) Increased accumulation of sorbitol: Sorbitol buildup is tied to the aldose reductase pathway seen in diabetes and galactosemia, not to glucose-6-phosphatase deficiency. Incorrect.

(C) Increased synthesis of glycerol: Glycerol synthesis is not the driver of high uric acid in this disease. Incorrect.

(D) Decreased function of Krebs cycle: The Krebs cycle is not the pathway linked to purine overproduction here. Incorrect.

Step 6: Final Answer.
The trapped glucose-6-phosphate gets shunted into the pentose phosphate pathway, raising ribose-5-phosphate and PRPP, which drives extra purine synthesis and uric acid production.

Increased synthesis of pentoses

Note: the source answer key for this question did not list a lettered answer (it was left blank on the page). Option (A) is filled in here

based on the standard, well established mechanism of high uric acid in glucose-6-phosphatase deficiency (Von Gierke disease).

Quick Tip: Trapped glucose-6-phosphate is shunted into the pentose phosphate pathway, raising PRPP and purine and uric acid production.

200. Consider a chain reaction with the sequence S1 to S2 to S3 to S4, where S4 is converted to P1 and P1 is converted to P2. The enzymes catalyzing the three reactions S1 to S2, S2 to S3, and S3 to S4 are EA, EB, and EC respectively. Enzyme EA is under positive feedback and enzyme EB is under negative feedback. If enzyme EC is absent, which of the following is true?

(A) S1 accumulates

(B) S2 accumulates

(C) P1 accumulates

(D) P2 accumulates

Correct Answer: (B) S2 accumulates

Solution:

Step 1: Understanding the Question.

The pathway runs S1 to S2 (enzyme EA), S2 to S3 (enzyme EB), and S3 to S4 (enzyme EC), with S4 going on to P1 and then P2. EA is switched on by positive feedback, and EB is switched off by negative feedback. The question asks what builds up if EC is missing.

Step 2: Trace what happens right after EC is lost.

Without EC, S3 cannot move forward to S4. This means S3, the

product of the EB reaction, starts to rise because it has nowhere to go.

Step 3: Apply the negative feedback on EB.
Negative feedback on EB means that as its own product (S_3) climbs, S_3 acts back on EB and slows it down. So once S_3 starts to build up from the missing EC step, EB is switched off before S_3 has a chance to keep climbing without limit. This stops the flow of S_2 into S_3.

Step 4: Apply the positive feedback on EA.
EA does not have this brake. Its positive feedback keeps it running, or even running harder, so S_1 keeps being turned into S_2 at a steady or increased pace.

Step 5: Put the two effects together.
S_2 keeps being made by EA, but it cannot move on to S_3 because EB has been throttled by the S_3 buildup triggering its negative feedback. With production continuing and the exit route blocked, S_2 is the one that piles up.

Step 6: Rule out the other options.

(A) S_1 accumulates: S_1 is still being used up normally by the unblocked, positively driven EA reaction. Incorrect.

(C) P_1 accumulates: P_1 sits downstream of the missing EC step, so with S_3 to S_4 blocked, little to no new S_4 or P_1 is even being formed. Incorrect.

(D) P_2 accumulates: P_2 is even further downstream than P_1, so it is affected the same way and does not build up. Incorrect.

Step 7: Final Answer.

With EA still active from positive feedback and EB shut down by negative feedback once S3 rises, the substrate stuck between them, S2, is what accumulates.

$$\boxed{\text{S2 accumulates}}$$

Quick Tip: Loss of EC lets S3 rise, which switches off EB by negative feedback; EA keeps feeding S2, so S2 backs up.