NEET PG 2001 Question Paper with Solutions

ZOLLEGE

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: List what stands out.

Three facts matter: the patient is young, there is no pain, and the vision came back by itself over about 3 months.

Step 2: Sort the four options by how they behave over time.

A macular hole stays open and needs surgery to seal. A retinal

detachment spreads and gets worse without repair. Angle closure glaucoma is painful from the start, with a red eye and high pressure, so it does not belong on this list at all. Central serous retinopathy is the one condition here where the fluid under the retina is known to soak back in naturally within weeks to a few months.

Step 3: Connect the mechanism to the recovery.
In central serous retinopathy a leak point in the retinal pigment epithelium lets fluid pool under the macula. Once the leak seals itself, which it commonly does, the fluid drains and the retina flattens back down, so vision returns without any procedure.

Step 4: Confirm by elimination.
Since the other three options either need treatment to improve or present with pain, none of them can explain a painless recovery within 3 months on their own.

Step 5: Conclude.
The self limiting course points to central serous retinopathy.

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.

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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: Separate the four options by where the problem sits.
Retinoblastoma, PHPV and fungal endophthalmitis all put something white behind the lens, a tumour, a membrane, or pus like exudate. Light bounces off that white surface and comes back through the pupil as a white glow.

Step 2: Look at congenital glaucoma differently.
Congenital glaucoma raises the pressure inside the eye from birth, and the cornea reacts by swelling and turning hazy. This haze sits in the front window of the eye, not behind the lens, and the usual signs are a big watery eye that avoids light, not a white pupil reflex.

Step 3: Test each option against the definition of leucoria.
Leucoria strictly means light being reflected off a white structure located behind the lens, in the vitreous or on the retina. Corneal clouding blocks the view instead of reflecting it, so it fails this test.

Step 4: Group the true leucoria causes.
Retinoblastoma, PHPV, and fungal endophthalmitis all pass the test. Congenital glaucoma does not.

Step 5: Conclude.
Congenital glaucoma is the exception.

Congenital glaucoma

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

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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: Set the target neutrality point.
Retinoscopy at a $1\ m$ working distance is neutral for an eye with exactly $-1.00D$ of myopia, since $1/1 = 1.00D$. Anything less myopic than this shows movement in the same direction as the mirror (a "with" reflex); anything more myopic shows movement in the opposite direction (an "against" reflex).

Step 2: Place this patient on that scale.
Her correction is $-0.5D$, so she is less myopic than the $-1.00D$ neutral

point for this test distance.

Step 3: Think in terms of the far point instead.
Her far point sits at $1/0.5 = 2\ m$ away, well beyond the examiner sitting at $1\ m$. The examiner is therefore looking from inside her far point, which by definition always produces a "with" movement.

Step 4: Match "with" movement to an answer choice.
A "with" movement means the red reflex in the pupil travels in the same direction the examiner sweeps the mirror, that is, in the direction of the mirror.

Step 5: Discard the wrong choices.
No movement only happens exactly at $-1.00D$. Opposite (against) movement only happens beyond $-1.00D$ of myopia. A scissors pattern needs an irregular cornea, which is not mentioned here.

Step 6: Conclude.

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.

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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: Separate direct infection from allergy.
TB can affect the eye in two different ways: the germ can directly invade eye tissue (as in TB uveitis or choroiditis), or the eye can react to TB proteins without the germ being present there, which is a hypersensitivity or allergic response. The question asks about the second kind.

Step 2: Recall the hallmark hypersensitivity lesion.
A phlycten is a small raised nodule of lymphocytes that forms at the limbus (junction of cornea and conjunctiva) as a delayed immune reaction to circulating tubercular antigen. This is called phlyctenular conjunctivitis or keratoconjunctivitis, and it is the standard teaching example of ocular TB allergy.

Step 3: Go through the distractors.
Koeppe's nodules sit on the iris and mark active granulomatous uveitis, a direct inflammatory process, not a surface allergic reaction. Posterior scleritis is uncommon and mostly tied to autoimmune causes. Optic neuritis in TB usually comes from drug side effects or nerve compression, not allergy.

Step 4: Fit the answer to the question wording.

Since the question specifically says "allergy," the nodule that forms purely from immune sensitisation, the phlycten, fits best.

Step 5: Conclude.

Phlyctenular conjunctivitis

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

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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: Build a checklist from the question.
The clues to match are: discharge that is ropy (thick and stringy), itching as a symptom, and a pattern that repeats every summer.

Step 2: Score each option against the checklist.
Bacterial conjunctivitis gives sticky pus like discharge but rarely itches and has no seasonal pattern, so it fails two of three clues. Trachoma is a slow scarring infection with no itching or seasonal link, failing two

clues. Phlyctenular conjunctivitis causes pain and photophobia from a limbal nodule, not itching or ropy discharge, failing all three.

Step 3: Check vernal catarrh against the same list.
Vernal catarrh is driven by an allergic (IgE) reaction that flares in warm weather, giving intense itching plus a thick, ropy mucous discharge that returns each summer. It satisfies all three clues at once.

Step 4: Confirm by mechanism.
Because it is allergic in origin, mast cells release histamine when exposed to seasonal allergens, which explains both the itch and the mucus rich, stringy character of the discharge.

Step 5: Conclude.

Vernal catarrh

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

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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: Picture the mechanism of injury.
A fist striking the eye pushes the eyeball backward, and this squeezes the soft tissue inside the bony orbit like a piston, spiking the pressure inside the cone shaped bone cavity.

Step 2: Ask where a piston like force escapes.
A raised pressure inside a closed box always breaks through the weakest wall first. In the orbit, the floor sits directly above the maxillary sinus and is built from paper thin bone, so it gives way before the thick rim or the strong lateral and superior walls.

Step 3: Confirm with the injuries seen afterward.
Surgeons see fat and eye muscle slipping down into the maxillary sinus through a broken floor, which produces a sunken looking eye and trouble looking up, plus numb cheek skin from the infraorbital nerve that runs along the floor. These findings would not happen if the fracture were in the roof or side wall.

Step 4: Compare with the medial wall.
The medial wall is thin too and can break, often alongside the floor, but taken alone the floor remains the single most frequent site.

Step 5: Conclude.

Inferior wall

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

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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: Split the four options into two groups.
Hyperemia, filling of the cup, and blurring of the margins are all things a doctor sees directly on the retina through an ophthalmoscope. Impaired pupillary reflex is a functional test of the nerve pathway, not a fundus appearance.

Step 2: Walk through disc swelling in order.
As pressure rises inside the skull and reaches the optic nerve, the disc first turns pink and engorged (hyperemia), then the small central dip flattens out and fills in (cup filling), and then the sharp edge of the disc turns fuzzy (margin blurring). Doctors use exactly this sequence to grade how severe the papilledema is.

Step 3: Test whether vision function changes early.
Despite all this visible swelling, the nerve fibers that carry the pupil's light response usually keep working normally through the early and moderate stages. The pupils stay equal and reactive because the swelling has not yet destroyed enough nerve fibers to break that reflex.

Step 4: Note when pupil changes would appear instead.
A sluggish or unequal pupil reflex points more to optic neuritis, direct optic nerve injury, or very late, chronic papilledema with optic atrophy, not moderate papilledema.

Step 5: Conclude.
Since the pupillary reflex stays normal at this stage, it gives no useful information for diagnosing moderate papilledema.

Impaired pupillary reflex

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

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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:

This question checks if you know the full form of ETDRS, a term you will see written on many vision charts used in eye clinics.

1. **Elective treatment for diabetic retinopathy scales**: Sounds close to the real name but is not correct, there is no study or chart with this exact title.
2. **Extended Rx for diabetes review study**: Also made up to sound similar, but this is not the source of the ETDRS name.
3. **Early Treatment Diabetic Retinopathy Study**: This is correct. It was a landmark trial on laser treatment timing in diabetic retinopathy, and the vision chart it introduced (with a fixed number of letters per row and even log spacing) took its name from the trial.
4. **Eye test drum review study**: Not a real name, "drum" has nothing to do with how this chart works or got named.

So the correct expansion is Early Treatment Diabetic Retinopathy Study, matching option (C).

Let's summarize:

- ETDRS charts use equal letters per row and log MAR spacing for more accurate vision testing.
- The name comes directly from the original diabetic retinopathy treatment trial.

The correct answer is the Early Treatment Diabetic Retinopathy Study.

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

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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:

The question is testing whether you know which mydriatic drug spares accommodation, since it asks for pupil dilation without loss of near focus.

1. **Atropine ointment**: An antimuscarinic with a very long action, sometimes over a week. It gives both strong mydriasis and strong cycloplegia, so it is wrong here.
2. **Phenylephrine**: A sympathomimetic that acts only on the dilator pupillae through alpha-1 receptors. It widens the pupil but does not touch the ciliary muscle, so near focus stays normal. This fits the question.
3. **Homatropine**: Another antimuscarinic, shorter acting than atropine but still causes cycloplegia along with mydriasis.
4. **Tropicamide**: A short acting antimuscarinic commonly used for fundus exams, but it too causes some cycloplegia, so it does not give mydriasis alone.

Only phenylephrine dilates the pupil while leaving accommodation working, since its target is the dilator muscle and not the ciliary muscle.

Let's summarize:

- Antimuscarinics such as atropine, homatropine, and tropicamide cause both mydriasis and cycloplegia.
- Phenylephrine, a sympathomimetic, causes mydriasis alone.

The correct answer is phenylephrine.

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

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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: Picture where the drug acts.
Amphotericin B has a strong attraction to cholesterol, and kidney tubule cell walls contain cholesterol too, so the drug injures these tubule cells as a side effect of killing fungi.

Step 2: Trace the tubule injury to its effects.
A damaged tubule leaks potassium into the urine, giving hypokalemia. It also loses the ability to pump out hydrogen ions, giving distal RTA. As tubular and vascular injury add up, the filtering capacity of the kidney drops, and urea and creatinine build up in the blood as

azotemia.

Step 3: Separate this from glomerular disease.
Glomerulonephritis comes from inflammation and immune complex deposits at the glomerulus, a completely different part of the kidney and a different mechanism. Amphotericin B toxicity is a direct chemical injury to tubules, not an immune attack on glomeruli.

Step 4: Match to the options.
Azotemia, hypokalemia, and renal tubular acidosis are all documented amphotericin B effects. Glomerulonephritis is not, since the drug simply does not act at that site.

Glomerulonephritis

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

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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:

This question is describing a set of birth defects and asking which drug taken by the mother causes this exact pattern.

1. **Isotretinoin**: Causes its own recognized pattern of defects, such as heart defects, ear and facial defects, and central nervous system defects, but spina bifida is not its typical feature, so it does not fit well here.
2. **Valproate**: Known to cause the highest rate of neural tube defects among antiepileptic drugs, giving spina bifida. It also causes cleft palate and cardiac defects such as ASD. This matches all three features in the question.
3. **Phenytoin**: Linked with fetal hydantoin syndrome, mostly limb and nail changes with some facial change, but spina bifida is not the hallmark finding.
4. **Carbamazepine**: Carries some neural tube defect risk but at a much lower rate than valproate, and is not the best fit for this full combination.

Since the pattern of spina bifida, cleft palate, and a possible ASD together matches fetal valproate syndrome most closely, valproate is the correct answer.

Let's summarize:

- Valproate has the strongest known link to neural tube defects among antiepileptics.
- It also adds craniofacial and cardiac defects, matching the full pattern described.

The correct answer is valproate.

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

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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:

This question asks which heart defect is most linked with lithium use in early pregnancy.

1. **Ebstein's anomaly**: The tricuspid valve sits lower than normal, pulling part of the right ventricle into the right atrium's job. This is the defect classically tied to first trimester lithium exposure, even though later research shows the actual risk is smaller than once believed.
2. **HOCM**: A thickened heart muscle disorder that mostly runs in families through gene changes, not connected to lithium.
3. **Aortic aneurysm**: Usually tied to connective tissue weakness, such as in Marfan syndrome, not a lithium effect.
4. **Eisenmenger syndrome**: Develops over years from an untreated shunt reversing direction, not something a drug causes directly in the fetus.

Among the choices, only Ebstein's anomaly is the recognized drug specific cardiac defect for lithium.

Let's summarize:

- Lithium in the first trimester is classically tied to Ebstein's anomaly.
- The other three options come from unrelated causes such as genetics or shunt physiology.

The correct answer is Ebstein's anomaly.

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

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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:

The question gives one physical clue, a black tongue, and asks which drug of abuse this points to.

1. **Heroin**: Chronic use shows track marks from injection, small pupils, and slowed breathing. There is no typical link to a black tongue.

2. **Cocaine**: When smoked as crack or freebase, the drug is heated in a small pipe that gets very hot. Repeated contact of the hot pipe stem with the lips and tongue causes burns that build up into a blackened, charred appearance, a sign seen in habitual smokers of this form of the drug.
3. **Cannabis**: Typical signs are red eyes, a dry mouth, and a raised appetite, not a black tongue.
4. **Arsenic**: This is a poison rather than a narcotic of abuse in this context, and its signs, such as nail line changes, skin pigment change, and garlic smelling breath, look nothing like a burned black tongue.

Only the smoking pattern of cocaine explains a black, burned looking tongue through repeated thermal injury from the hot pipe.

Let's summarize:

- A black tongue in a drug case points to repeated thermal injury from smoking.
- Crack or freebase cocaine smoked through a hot pipe is the classic cause of this sign.

The correct answer is cocaine.

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

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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:

The case describes flushing and a fall in blood pressure with alcohol, which is the disulfiram-like reaction. We need to find which drug does NOT cause this.

1. **Cefamandole**: Carries a side chain that blocks the enzyme aldehyde dehydrogenase, letting acetaldehyde build up and trigger the reaction with alcohol.
2. **Metronidazole**: A classic cause of this reaction, blocking the same enzyme, which is why alcohol is always avoided during treatment with it.
3. **Dexamethasone**: A steroid drug that works on hormone receptors, with no effect on the enzyme that breaks down acetaldehyde. It does not cause this reaction.
4. **Chlorpropamide**: An old diabetes drug that can also block the enzyme in some people, causing flushing with alcohol.

Since three of the four drugs block the enzyme that clears acetaldehyde, and dexamethasone does not touch this pathway at all, dexamethasone is the one that will not cause the reaction.

Let's summarize:

- The disulfiram-like reaction comes from acetaldehyde build up when aldehyde dehydrogenase is blocked.

- Cefamandole, metronidazole, and chlorpropamide can all block this enzyme; dexamethasone cannot.

The correct answer is dexamethasone.

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

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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: Note what makes this interaction dangerous.
Theophylline has a narrow safety margin, so a small rise in blood level can cause toxicity. The question wants the drug that raises this level when a chest infection needs treatment.

Step 2: Think about enzyme inhibition.
The liver enzyme CYP1A2, with some help from CYP3A4, clears theophylline from the body. A drug that blocks this enzyme lets theophylline pile up.

Step 3: Match each option to its enzyme effect.
Erythromycin, a macrolide, is a well known inhibitor of CYP3A4 and partly CYP1A2. Ampicillin works on bacterial cell wall building and has no effect on liver enzymes. Cotrimoxazole targets bacterial folate synthesis and also spares CYP1A2. Sparfloxacin does not carry the strong CYP1A2 inhibition seen with ciprofloxacin or enoxacin.

Step 4: Connect the mechanism to the clinical picture.
Because erythromycin slows theophylline breakdown, giving it to this patient lets theophylline build up toward toxic levels, causing symptoms like vomiting, palpitations, and tremor.

Step 5: State the answer.
Erythromycin is the antibiotic that can precipitate theophylline toxicity here.

Erythromycin

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

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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: Split the options into two groups.
Some vasculitides form granulomas on biopsy, and some do not. The question wants the one without granulomas.

Step 2: Place giant cell arteritis.
This large vessel disease of older adults shows granulomatous inflammation of the arterial wall, with giant cells attacking the internal elastic lamina. It belongs in the granulomatous group.

Step 3: Place Wegener's and Churg-Strauss.
Both are ANCA-associated vasculitides that affect small vessels, but both also produce necrotising granulomas, in the sinuses and lungs for Wegener's, and around eosinophils in Churg-Strauss. Both belong in the granulomatous group too.

Step 4: Place microscopic polyangiitis.
This is also ANCA-associated and affects small vessels like Wegener's, but biopsy shows plain necrotising vasculitis without any granuloma formation. This is what pathologists use to tell it apart from Wegener's granulomatosis.

Step 5: Conclude.
Three of the four options form granulomas. Only *microscopic polyangiitis* does not.

Microscopic Polyangiitis

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

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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: Start from the drug history.
Steroids taken for 14 years is the loudest clue here. A high cumulative steroid dose is one of the top causes of bone death in the femoral head, working through fat embolism and raised pressure inside the bone that cuts off blood supply.

Step 2: Match the exam findings to the diagnosis.
As the femoral head caves in on its weight bearing surface, the hip loses internal rotation and abduction first. When the patient tries to flex the hip, the joint surfaces no longer glide smoothly, so the leg drifts into abduction instead of flexing straight, the sign described in the question.

Step 3: Test the other choices against the story.
Septic arthritis would show a hot, swollen, acutely painful joint with fever, not a slow bilateral change over years. Renal osteodystrophy affects bone mineral turnover from kidney failure and gives generalised bone pain and deformity, not this specific hip sign, and nothing here says the kidneys have failed. Heterotopic calcification needs a trigger like major trauma or spinal injury, which is not present here.

Step 4: Settle on the diagnosis.
The combination of prolonged steroid use with loss of internal rotation and abduction, plus a positive flexion-abduction sign, points to avascular necrosis of both femoral heads.

Avascular Necrosis of Femur Head

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

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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: Anchor HCG to trophoblastic tissue.
HCG in germ cell tumors always comes from syncytiotrophoblast-type cells, the same cells that make HCG during a real pregnancy. So the question is really asking which of these four tumors has no trophoblastic component.

Step 2: Go through the trophoblast-containing ones.
Choriocarcinoma is built entirely from trophoblast, so HCG is always sky high. Polyembryoma contains embryoid bodies with a trophoblastic rim, so it also makes HCG. Embryonal carcinoma is undifferentiated but frequently has scattered giant cells of trophoblastic type mixed in, which is why it can secrete AFP, HCG, or both.

Step 3: Look at the yolk sac tumor separately.
The endodermal sinus tumor copies the fetal yolk sac, a structure that makes AFP in a developing fetus, not HCG. Since it has no trophoblastic differentiation, HCG stays normal while AFP rises.

Step 4: Pick the odd one out.
Three of the four tumors carry trophoblastic tissue and raise HCG. The endodermal sinus tumor does not.

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: Work backward from the ECG finding.
Tall QRS voltage in an infant usually means the heart muscle mass is abnormally increased. One cause is infiltration of the heart muscle cells with a stored substance, which thickens the walls and boosts the electrical voltage recorded on ECG.

Step 2: Add the other two clues.
Coarse facial features and hepatosplenomegaly are the hallmark of a storage disease where an enzyme defect lets a substance, here glycogen, collect inside lysosomes of many organs, not just the heart.

Step 3: Name the enzyme defect.
The missing enzyme is acid alpha-glucosidase. Without it, lysosomes in cardiac muscle, skeletal muscle, and liver fill up with glycogen that cannot be broken down. This disease carries two names in textbooks, Pompe's disease and Glycogen Storage Disease Type II, referring to the exact same enzyme defect.

Step 4: Check the distractors.
Marfan syndrome affects connective tissue and gives tall stature and

aortic problems, not organomegaly or tall QRS. Romano-Ward syndrome lengthens the QT interval and predisposes to torsades; it does not cause tall QRS or organ enlargement.

Step 5: Conclude.
The features described match ***Pompe's disease***, the infantile form of glycogen storage disease type II.

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).

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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: Read the key phrase in the question.
"Prolonged QT interval since birth" signals congenital long QT syndrome, an inherited channelopathy, not a drug or electrolyte problem picked up later in life.

Step 2: Split the four options by mechanism.
Magnesium sulphate calms the heart membrane in any torsades episode. Overdrive pacing and isoproterenol both work by raising heart rate, which shortens a QT interval that is long because of bradycardia or a pause, the pattern typical of acquired long QT from drugs or electrolyte problems. Metoprolol, a beta-blocker, works by blunting the effect of adrenaline on the heart.

Step 3: Match mechanism to congenital disease.
In congenital long QT syndrome, especially the LQT1 subtype, exercise and emotional stress, meaning adrenaline surges, are the classic triggers for torsades. A drug like isoproterenol that mimics adrenaline would make this worse, not better, so it is unsuitable here even though it is useful in acquired, bradycardia-driven torsades.

Step 4: Confirm the beta-blocker link.
Because adrenaline drive is the problem in congenital LQTS, blocking it with a beta-blocker is the standard preventive treatment recommended for these patients, cutting the risk of future torsades and sudden death.

Step 5: State the answer.
The suitable drug for this patient with lifelong QT prolongation is the beta-blocker ***metoprolol***.

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: Set the definition to test against.
A congenital myopathy is a structural muscle disease present from birth, diagnosed by a specific pattern on muscle biopsy, not by fiber necrosis as in a true dystrophy. Three names have carried this label for decades.

Step 2: Walk through the classic three.
Central core disease shows pale, enzyme-poor cores running down the centre of fibers. Nemaline myopathy shows small rod-like bodies scattered in the fibers, built from Z-disc proteins. Centronuclear myopathy shows nuclei that sit in the middle of the fiber instead of at the edge, like immature muscle cells. All three appear in infancy with a floppy baby picture.

Step 3: Place Z-band (myofibrillar) myopathy in a different box.
Z-band myopathy belongs to the myofibrillar myopathy family, where the Z-disc breaks down over time and abnormal protein clumps build up. Unlike the three above, it typically shows up in adulthood with slowly progressive limb or distal weakness, and it is grouped separately from the congenital myopathies in most classifications.

Step 4: Draw the conclusion.

Since three of the four options fit the congenital myopathy definition and the fourth does not, the exception is ***Z band myopathy***.

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.

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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:

CPK leaks into blood only when the cells that hold it, mainly muscle cells, get damaged. Let's check each option against that rule.

1. **Muscle disease**: Directly injures muscle fibers, so CPK spills into the blood and the level rises. This fits.
2. **Liver cirrhosis**: Damages liver cells, which raises liver enzymes and bilirubin, not CPK.
3. **Biliary colic**: A blocked bile duct raises ALP and bilirubin, again with no muscle injury involved.

4. **Amyotrophic lateral sclerosis**: Attacks the nerve cells controlling muscle, not the muscle fibers themselves, so CPK is usually normal or only slightly high.

Only muscle disease causes true muscle breakdown, so it is the condition where CPK is clearly raised.

Let's summarize:

- CPK is a muscle enzyme, so it rises with muscle cell damage.
- Liver and biliary problems raise a different set of enzymes altogether.
- Nerve-cell diseases like ALS spare the muscle fiber itself, so CPK stays close to normal.

The condition with a raised CPK is muscle disease.

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

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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:

The patient's high urea and creatinine show renal failure. A drug's dose only needs to come down if the kidney is its main exit route. Let's go through the four anti-TB drugs.

1. **INH**: Cleared mostly by the liver, but a part of the load still leans on the kidney, so some caution is used in severe renal failure.
2. **Rifampin**: Cleared almost fully by the liver into bile. Kidney failure barely changes its blood level, so the usual dose can continue.
3. **Pyrazinamide**: Its breakdown products rely on the kidney for clearance, so they build up in renal failure and the dose must be trimmed.
4. **Ethambutol**: Leaves the body mostly unchanged through the kidney, so renal failure lets it pile up and raises the risk of eye toxicity, so the dose must be reduced.

Rifampin is the one drug whose main clearance route bypasses the kidney, so it alone needs no dose change.

Let's summarize:

- Liver-cleared drugs like rifampin stay safe at the usual dose in renal failure.
- Kidney-cleared drugs like pyrazinamide and ethambutol need a lower dose or longer interval.

The drug that needs no dose reduction here is rifampin.

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:

Indinavir, one of this patient's HIV drugs, is a protease inhibitor broken down by the liver enzyme CYP3A4. Any drug that strongly revs up this enzyme will lower indinavir's blood level. Let's check each option.

1. **INH**: Cleared mainly by acetylation in the liver and does not meaningfully switch on CYP3A4, so it is safe with indinavir.
2. **Rifampin**: A powerful CYP3A4 inducer. It speeds up the breakdown of indinavir so much that blood levels can fall below the amount needed to keep the virus suppressed, risking treatment failure and resistance.
3. **PZA**: Does not act on CYP3A4 in a way that harms protease inhibitor levels.
4. **Ethambutol**: Leaves the body mainly through the kidney and does not induce CYP3A4 either.

Because rifampin can silently sink indinavir's blood level, it is the drug to avoid, with rifabutin used instead when TB treatment is unavoidable.

Let's summarize:

- Protease inhibitors depend on stable CYP3A4 activity to stay effective.
- Rifampin's strong enzyme induction drops indinavir levels and threatens HIV control.

The drug to avoid here is rifampin.

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

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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:

Multiple myeloma is a plasma cell cancer. Let's test each statement against what is actually known about it.

1. **More common over 50 years**: True. Myeloma is mainly a disease of older adults.

2. **Arises from clones of plasma cells**: True. One transformed plasma cell multiplies into a clone that overproduces a single antibody type.
3. **Bence Jones proteins are abnormal whole immunoglobulins in urine**: False. A whole immunoglobulin has both heavy and light chains bound together. Bence Jones protein is only the free light chain part, small enough to be filtered into urine on its own. Calling it a whole Ig is incorrect.
4. **Predisposition to amyloidosis**: True. The extra free light chains can misfold and deposit in organs as AL amyloid.

Only the third statement misdescribes Bence Jones protein, so it is the one that is not true.

Let's summarize:

- Bence Jones protein is a free light chain fragment, not an intact immunoglobulin.
- The other three statements correctly describe age, clonal origin, and amyloid risk in myeloma.

The false statement is about Bence Jones proteins being 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:

This patient has back pain with L3 tenderness, a reversed albumin to globulin ratio, raised creatinine, and an ESR of 90. Let's weigh each option against these clues.

1. **Waldenstrom's macroglobulinemia**: A clonal IgM disorder that usually causes hyperviscosity features like blurred vision and mucosal bleeding, not lytic bone disease or vertebral tenderness, so it does not match this picture well.
2. **Multiple myeloma**: A plasma cell clone overproduces one type of globulin, which flips the albumin to globulin ratio, makes red cells clump and pushes ESR very high, damages the kidney through light chain injury, and eats away at bone causing localized pain and tenderness exactly like the L3 finding here.
3. **Amyloidosis**: Can occur alongside myeloma, but on its own does not explain the reversed A:G ratio or the vertebral tenderness in this vignette.

4. **Bone secondaries**: Explain bone pain but not the reversed A:G ratio or the very high ESR, since there is no paraprotein driving those changes.

Every clue, reversed A:G ratio, sky-high ESR, renal impairment, and vertebral tenderness, lines up with multiple myeloma.

Let's summarize:

- A reversed A:G ratio with a very high ESR signals a paraprotein-producing plasma cell clone.
- Vertebral tenderness plus renal impairment in this setting points to myeloma bone and kidney disease.

The likely diagnosis is 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.

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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:

Target cells, nucleated red cells, microcytic hypochromic anemia, and a positive family history together point toward an inherited hemoglobin disorder such as thalassemia. Let's check what each test actually tells us.

1. **Coombs test**: Detects antibody-coated red cells in autoimmune hemolysis, which has nothing to do with an inherited condition running in the family.
2. **Osmotic fragility**: Shows that red cells resist bursting in dilute saline, which happens in thalassemia because of the target cells, but this only screens; it cannot pin down the exact hemoglobin defect.
3. **Hb electrophoresis**: Separates the different hemoglobin fractions and directly shows the abnormal pattern, such as raised HbA2 or HbF, that confirms thalassemia.
4. **Sucrose lysis test**: Screens for paroxysmal nocturnal hemoglobinuria, an acquired red cell disorder, not an inherited one, so it does not fit this case.

Since the picture strongly suggests an inherited hemoglobinopathy, the test that actually proves it by reading the hemoglobin types is Hb electrophoresis.

Let's summarize:

- Target cells and nucleated RBCs with a family history point to thalassemia, not an autoimmune or acquired red cell disease.
- Only Hb electrophoresis directly identifies the abnormal hemoglobin fractions to confirm it.

The investigation of choice is Hb electrophoresis.

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.

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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:

Hypercarbia is a build-up of carbon dioxide in the blood. Its two main effects are vessel dilation, especially in the skin and brain, and a sympathetic nervous system surge. Let's test each option against that.

1. **Miosis**: High CO_2 has a direct depressant effect on the brain, and small pupils can appear with marked hypercarbia, so this is a real feature.
2. **Cold clammy extremities**: This finding comes from vessels clamping shut, as in shock. Hypercarbia does the opposite and opens vessels up, giving warm extremities instead, so this is not a feature.
3. **Bradycardia**: The sympathetic surge from rising CO_2 speeds the heart up, not down. A slow heart rate only shows up as a late or terminal sign, not a standard feature, so this too is not a feature.

4. **Hypertension**: The sympathetic surge and direct vessel effects of CO_2 raise blood pressure, at least in the early stage, so this is a real feature.

Two options describe the opposite of what hypercarbia actually does to the circulation, so they are the ones that do not belong.

Let's summarize:

- Hypercarbia causes vessel dilation and a sympathetic surge, giving warm skin, a fast heart, and raised blood pressure.
- Cold clammy skin and a slow heart rate describe the opposite picture and are not features of hypercarbia.

The features that are NOT seen in hypercarbia are cold clammy extremities and bradycardia.

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

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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: Use the case as a pattern, not just single facts.
Four months of low grade fever that comes and goes, weight loss, and coughing up blood is a long, drawn out picture. That duration alone should shift the first thought toward a chronic infection rather than a quick viral illness.

Step 2: Weigh the Mantoux result.
A 14 x 17 mm induration on the Mantoux test is a strongly positive reaction. In a country where TB is common, this supports past or current exposure to Mycobacterium tuberculosis and fits with active disease when paired with matching symptoms.

Step 3: Do not be fooled by the negative sputum smear.
Smear microscopy misses a fair number of true TB cases, since it needs a high bacterial load to turn positive. A single negative AFB smear is common in early or lower burden disease and should not be used alone to exclude tuberculosis.

Step 4: Check the raised ESR against the differentials.
The raised ESR fits a chronic granulomatous infection like TB well. Viral pneumonia is short lived and would have resolved or clearly worsened within days, not persisted for 4 months. Fungal pneumonia needs a predisposing lung disease or a weak immune system, which is not mentioned here. Bronchogenic carcinoma is rare in a 25 year old with no smoking history given.

Step 5: Settle on the diagnosis.
Putting the fever pattern, weight loss, hemoptysis, strong Mantoux,

and raised ESR together, pulmonary tuberculosis remains the best fit despite the negative 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.

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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: Go through each option as a separate true/false test.
The question wants the option(s) that do NOT hold for prion disease, so each choice needs to be checked against what is actually known about conditions like CJD.

Step 2: Test the infectious protein claim.
Prion disease is unique because the infectious agent is a misfolded protein (PrPSc) with no genetic material at all. It works by forcing normal PrP to refold into the abnormal shape. This matches known

biology, so this option holds up as true.

Step 3: Test the myoclonus number.
Clinical studies of CJD report myoclonus in a majority of patients as the disease advances, generally cited near 80 to 90%, far above 10%. A figure of only 10% is too low, so this option fails the test.

Step 4: Test the "universal dementia" claim.
Dementia is a core and common feature of CJD, but "universal" is a strong word meaning every single case, every single prion disease. Because some prion diseases and atypical presentations can start with ataxia, insomnia, or psychiatric change without dementia being the presenting or invariable feature, this option also fails the test.

Step 5: Test the brain biopsy claim.
A brain biopsy can show the spongiform vacuolation typical of prion disease and is accepted as a diagnostic method, so this option holds up as true.

Step 6: Collect the failing options.
Two statements do not hold: the 10% myoclonus figure and the claim that dementia is universal.

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: Use the gap between infection and kidney disease as the main filter.
Different causes of post infectious hematuria have very different time gaps, so lining up the given 3 day gap against each option quickly narrows the choices.

Step 2: Line up IgA nephropathy first.
IgA nephropathy is famous for the "synpharyngitic" pattern, hematuria appearing at the same time as or within a couple of days of a throat or upper airway infection. A 3 day gap fits this pattern closely.

Step 3: Line up PSGN next.
PSGN needs an immune complex response to build up, so its typical latent period is about 10 to 14 days after pharyngitis, sometimes longer after skin infection. Three days is much too short for PSGN to have developed.

Step 4: Check HSP and HUS for fit.
HSP would usually be recognized by its skin purpura and joint or belly pain alongside the kidney findings, and the case here mentions none

of that. HUS is linked to a preceding diarrheal illness rather than a respiratory one, and comes with anemia and low platelets, not just isolated hematuria.

Step 5: Conclude.
Since the gap is short and there is no rash, joint pain, or diarrhea mentioned, IgA nephropathy fits best.

IgA nephropathy

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

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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: Separate the two problems.
The patient has two viral infections, hepatitis B (HBsAg positive) and hepatitis C, but also carries a transplanted kidney that depends on suppressed immunity to survive. The safest antiviral choice for the

transplant has to come first.

Step 2: Think about what interferon does to the immune system.
Interferon amplifies immune activity, which is exactly what a transplant recipient does not want, since the immune system is being deliberately held back to protect the graft. Using interferon can wake up the immune response enough to attack the transplanted kidney. That single fact removes any option built around interferon.

Step 3: Weigh ribavirin on its own.
Ribavirin alone is a weak strategy against hepatitis C, it is normally paired with another drug, and by itself does nothing for the hepatitis B infection this patient also has.

Step 4: Look at what lamivudine offers.
Lamivudine works by directly inhibiting the polymerase enzyme of hepatitis B virus. It does not rely on ramping up the immune system, so it can be used safely in someone on immunosuppressive therapy after transplant.

Step 5: Conclude.
Given the need to protect the graft while still treating the hepatitis B infection, lamivudine alone is the most sensible and beneficial option here. In current practice, entecavir or tenofovir would usually be preferred over lamivudine for a better resistance profile, but among the listed choices lamivudine alone remains correct.

Lamivudine alone

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

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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: Sort the four conditions by whether beta blockade helps or hurts.
Propranolol's effect depends on which receptors are dominant in the problem being treated, heart-related conditions generally benefit, airway conditions generally suffer.

Step 2: Look at the heart related options first.
Panic attack symptoms like a racing heart and shaking are driven by excess sympathetic (beta) activity, so blocking beta receptors calms these symptoms. PVCs come from abnormal excitability in heart muscle, and reducing sympathetic drive with a beta blocker can quiet the extra beats. Hypertrophic cardiomyopathy causes outflow obstruction that gets worse with a fast, forceful heartbeat, so slowing and softening contraction with a beta blocker actually reduces the obstruction. All three of these are settings where propranolol is useful.

Step 3: Now look at the airway.
Propranolol does not choose only heart receptors, it also blocks beta-2 receptors in the airway smooth muscle. Those receptors normally keep the airway relaxed. Removing that relaxing signal in someone with asthma can bring on a dangerous bronchospasm.

Step 4: Conclude.
Because of this airway risk, asthma is the one condition on the list where propranolol should not be used.

Asthma

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

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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: Build the expected HIV pathology checklist first.
Before checking the options, it helps to recall what HIV actually does to the CNS. It causes a subacute encephalitis marked by microglial nodules, multinucleated giant cells, and cuffing of blood vessels by

inflammatory cells (perivascular infiltration). In the cord, it produces a vacuolar myelopathy that especially affects the posterior columns.

Step 2: Tick off the matching options.
Perivascular infiltration, microglial nodules, and posterior column vacuolar myelopathy all appear directly on this checklist, so options (A), (B), and (C) are genuine features of HIV CNS disease.

Step 3: Ask where temporal lobe disease really belongs.
A strong temporal lobe pattern, often with bleeding and necrosis, is the textbook signature of herpes simplex encephalitis, a completely different virus with a different pattern of brain injury. HIV's damage is more spread out and does not target the temporal lobes in this focal way.

Step 4: Conclude.
Because temporal lobe involvement belongs to HSV encephalitis rather than HIV, it is the one option that is NOT a recognized CNS anomaly of 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.

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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: Start from what actually happens after a healthy adult catches toxoplasma.
In a person with a normal immune system, the parasite is usually contained quickly and quietly. Most people never even know they were infected, and it is picked up later only because a blood test happens to show antibodies.

Step 2: Put a number on it.
Surveys consistently find that somewhere around 80 to 90 percent of adult toxoplasma infections cause no noticeable illness at all. Only a smaller share cause the mild swollen glands and low fever that some patients do report. This means the idea that "adult infections are mainly symptomatic" gets the proportion backwards.

Step 3: Walk past the other statements briefly.
Persisting IgG in an infant beyond the window where mother's antibody should have faded is accepted as a sign of congenital infection, so that statement stands. The mother-to-fetus route for congenital cases is a person-to-person link in the transmission story, so calling that route anthroponotic is reasonable. Encephalitis from toxoplasma is mostly a disease of weakened immunity, but it is not absolutely restricted to immunocompromised people, occasional cases

occur in the immunocompetent too, so that statement is treated as true here.

Step 4: Land on the false statement.
Only the claim about adult infections being "mainly symptomatic" clashes with the real picture, where silent infection is actually the norm.

Adult infections are mainly symptomatic (this is false)

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

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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:

Step 1: Understanding the Question:
We need to pick the FALSE statement about dental care in an adult with hemophilia (low factor VIII or IX).

Step 2: Key Formula or Approach:

Go through each option and check it against standard hemophilia dental-care protocol: factor cover, anesthesia technique, infection screening, and GA precautions.

Step 3: Detailed Explanation:
Cryoprecipitate or factor VIII concentrate is standard cover before extractions, so that option is a true statement.
HIV screening is done because many hemophiliacs had past exposure to pooled blood products, so that option is also true.
If general anesthesia is used, monitored, factor-covered GA care is standard, so that option is true as well.
The lidocaine dose is not raised in hemophilia. What changes is technique: infiltration is chosen over a nerve block, because a block can injure a vessel deep in tissue and start a hematoma that is difficult to control.

Step 4: Final Answer:
The false statement is that the dose of lidocaine required is increased.

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.

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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:

Fibrolamellar hepatocellular carcinoma is a distinct subtype of liver cancer that behaves quite differently from the usual (classic) hepatocellular carcinoma, and the question wants the one statement below that does not match its known features.

1. **Not more in males**: True. While classic HCC has a strong male preference, fibrolamellar HCC has close to an equal sex distribution.
2. **Better prognosis**: True. Because it usually arises in an otherwise healthy, non-cirrhotic liver and is often found before it has spread widely, it can be resected more often and carries a better outlook than classic HCC.
3. **AFP is raised over 1000**: False. This tumor is known for a normal or only mildly raised AFP; a very high AFP over 1000 points to classic HCC in a cirrhotic liver instead.
4. **Seen in a younger age group**: True. It is the classic liver cancer of children, teenagers, and young adults without cirrhosis, unlike classic HCC which appears in older patients with chronic liver disease.

Since three of the four statements match the known features of fibrolamellar HCC, the odd one out, and the correct answer, is the claim about a markedly raised AFP.

Let's summarize:

- Fibrolamellar HCC: young patients, no cirrhosis, near-normal AFP, better prognosis, equal sex distribution.

- Classic HCC: older patients, cirrhotic liver, often high AFP, worse prognosis, more common in males.

So the false statement, and the answer, is that AFP is raised over 1000.

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

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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:

Step 1: Understanding the Question:
We need the vascular abnormality that adult (autosomal dominant) polycystic kidney disease is classically linked to.

Step 2: Key Formula or Approach:
Recall that ADPKD is caused by a defect in polycystin proteins found not just in kidney tubule cells but also in the walls of blood vessels, so its vascular complications follow from that shared defect.

Step 3: Detailed Explanation:
The polycystin defect weakens the muscular and elastic layers of arteries, and this effect is most clinically important in the circle of Willis at the base of the brain, where it predisposes to saccular (berry) aneurysms.
These aneurysms can rupture and cause a subarachnoid hemorrhage, so patients with a family history of this complication are actively screened.
Aortic aneurysms, whether fusiform or saccular, at the ascending aorta or the arch, are not the classic textbook vascular link for ADPKD; they belong more to atherosclerotic or connective tissue disease.

Step 4: Final Answer:
Adult polycystic kidney disease is associated with berry aneurysm.

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

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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:

The clue pattern here, a young patient, a spleen reaching down to the umbilicus, and bleeding esophageal varices, points to a cause of portal hypertension where the spleen is the dominant finding rather than liver failure.

1. **Budd-Chiari syndrome**: This comes from blockage of the hepatic veins draining the liver. It usually shows up with a tender, enlarged liver and ascites as the leading features, not a massive spleen as the main sign.
2. **Veno-occlusive disease**: This affects the small veins inside the liver, often linked to bone marrow transplant conditioning or certain plant toxins, and again presents mainly with painful hepatomegaly and fluid in the abdomen.
3. **Non-cirrhotic portal fibrosis**: This fits best. It is common in young people in this region, and it causes portal hypertension through fibrosis of small portal vein branches while the liver cells stay normal. The classic presentation is a massive spleen that the patient tolerates well, along with repeated variceal bleeds, but normal liver function tests.
4. **Cirrhosis of the liver**: This would usually show additional signs of chronic liver disease, like jaundice or ascites, and is uncommon as an isolated presentation at this age without a clear cause.

Putting the massive splenomegaly together with preserved liver function and variceal bleeding in a young patient gives non-cirrhotic portal fibrosis as the most likely diagnosis.

Let's summarize:

- Massive, well tolerated splenomegaly with normal liver function in a young patient is the hallmark of non-cirrhotic portal fibrosis.
- Budd-Chiari and veno-occlusive disease present mainly with liver enlargement and ascites, not massive splenomegaly.

So the diagnosis is non-cirrhotic portal fibrosis.

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

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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:

Step 1: Understanding the Question:
We need the FALSE statement about adult (autosomal dominant) polycystic kidney disease.

Step 2: Key Formula or Approach:
Check each option against the standard features of ADPKD: urinary findings, blood pressure pattern, inheritance, and extrarenal organ involvement.

Step 3: Detailed Explanation:
Hematuria is a true feature, since cyst rupture or bleeding into a cyst commonly shows up as blood in the urine.
Autosomal dominant transmission is true and is the defining inheritance pattern of the disease, most often from a PKD1 gene mutation.
Cysts in the liver, spleen, and pancreas are true findings, since ADPKD is a systemic cystic disease and not limited to the kidneys.
Hypertension, however, is not rare in ADPKD. It is one of the most common findings and often develops before any drop in kidney function, driven by activation of the renin-angiotensin system within the enlarging kidneys. So the claim that hypertension is rare is false.

Step 4: Final Answer:
The false statement is that hypertension is rare in adult polycystic kidney disease.

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

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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:

This vignette gives an 18-year-old with a two-week fever treated with drugs, a moderately enlarged spleen, and massive hematemesis. The question is asking which option is the actual diagnosis behind the bleeding.

1. **NSAID-induced duodenal ulcer**: There is no mention of NSAID use, and this would not explain the splenomegaly, so it is unlikely.
2. **Drug-induced gastritis**: This also does not explain an enlarged spleen, and gastritis rarely produces bleeding this massive by itself.
3. **Esophageal varices**: The combination of splenomegaly and massive hematemesis in a young patient is the classic picture of bleeding from esophageal varices, dilated veins at the lower end of the esophagus that form when pressure rises in the portal venous system and rupture to cause dramatic blood loss.
4. **Portal hypertension**: This is the raised pressure state that causes the varices and the splenomegaly, but it is the mechanism, not the bleeding lesion itself.

Since the question is asking for the diagnosis that accounts for the bleeding, the answer is the visible lesion, esophageal varices, rather than the underlying mechanism of portal hypertension.

Let's summarize:

- Splenomegaly with massive hematemesis in a young patient points to a portal hypertensive source of bleeding.
- Esophageal varices are the actual ruptured vessels causing the bleed; portal hypertension is the state that produces them.

So the correct answer is esophageal varices.

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

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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:

Step 1: Understanding the Question:
A previously healthy 25-year-old man develops an arrhythmia soon after binge drinking. We need the rhythm disturbance that is classically linked to this trigger.

Step 2: Key Formula or Approach:
Recall the entity called "holiday heart syndrome," which describes a specific arrhythmia appearing after acute heavy alcohol intake in people with no underlying heart disease.

Step 3: Detailed Explanation:

Alcohol can acutely change the electrical activity of the atria, shorten the atrial refractory period, and raise sympathetic tone, all of which make the atria prone to a chaotic, disorganized rhythm.
This chaotic atrial rhythm is atrial fibrillation, and its sudden appearance after a drinking binge in an otherwise normal heart is exactly what holiday heart syndrome describes.
Supraventricular tachycardia, ventricular ectopics, and atrial flutter can all occur with heavy alcohol use as well, but none of them carries the same strong, classic, named association with binge drinking that atrial fibrillation does.

Step 4: Final Answer:
The likely pathology is atrial fibrillation, the hallmark rhythm of holiday heart syndrome.

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

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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: Understanding the Concept:
Hypothalamic hypogonadism means the problem starts above the pituitary, at the level of GnRH release. Low GnRH gives low LH and FSH, and the testes or ovaries never get switched on.

Step 2: Key Formula or Approach:
Go through each option and ask where its lesion actually sits, the hypothalamus and GnRH neurons, or somewhere else entirely.

Step 3: Detailed Explanation:
Frohlich syndrome comes from a hypothalamic mass causing obesity plus hypogonadism, but it is a general structural hit, not the classic single entity being tested here.
Foster Kennedy syndrome is about vision, one optic nerve atrophies while the other disc swells, from a frontal tumor pressing near the olfactory groove; it does not touch reproductive hormones at all.
Fragile X syndrome is a genetic repeat expansion disorder with large testes and learning problems, again not a GnRH problem.
Kallmann syndrome is different: during fetal life, the neurons that should make GnRH start out next to the smell neurons and must migrate into the hypothalamus. In Kallmann syndrome this migration fails, so GnRH is never made properly and the sense of smell is also lost.

Step 4: Final Answer:
The classic cause of hypothalamic hypogonadism among these choices is Kallmann syndrome, since it is defined by failed GnRH neuron migration.

Kallmann syndrome

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

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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:

This question is testing the treatment ladder for acute pulmonary embolism (PE) and which step comes first when the embolism is severe enough to demand urgent action.

1. **Thrombolysis**: Drugs such as streptokinase or alteplase break down the clot itself within hours. This is reserved for a large or massive PE, where the clot burden threatens the patient's life and there is no time to wait for anticoagulation alone to work.
2. **LMW heparin**: This stops the clot from growing bigger and stops new clots forming, but it cannot dissolve the clot already blocking the pulmonary artery. It is the right choice for a stable patient, not a critical one.
3. **Oral anticoagulants**: Warfarin and similar drugs take several days to become fully effective, so they are used later for long term prevention, never as an emergency first step.

4. **IVC filter**: This device sits in the inferior vena cava and traps future clots travelling up from the legs. It protects against a future embolism but does nothing to the clot already present in the lungs.

The correct next step is thrombolysis, because breaking down the existing clot fast is what a severe, life threatening pulmonary embolism needs.

Let's summarize:

- Thrombolysis dissolves the clot and is used for high risk, unstable PE.
- LMW heparin and oral anticoagulants only prevent new clot, they do not clear the existing one.
- An IVC filter only prevents future emboli, it has no effect on a clot already in the lung.

So the immediate next line of management here is thrombolysis.

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

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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: Understanding the Concept:
This vignette is describing a brainstem stroke. The trick is separating which signs sit on the same side as the lesion and which sit on the opposite side.

Step 2: Key Formula or Approach:
Same side (ipsilateral) signs come from structures that have not crossed yet, like cranial nerve nuclei and descending sympathetic fibers. Opposite side (contralateral) signs come from tracts that already crossed lower down, like the spinothalamic tract.

Step 3: Detailed Explanation:
Horner's syndrome on the right and the cerebellar type features on the right are ipsilateral, so the lesion sits on the right side of the brainstem.
Loss of pain and temperature on the left is contralateral, matching a spinothalamic tract that crossed lower down and is now travelling up on the right side, so it gets caught by the same right sided lesion.
Dysphagia adds nucleus ambiguus involvement, again on the right.
This full package, ipsilateral Horner's, ipsilateral bulbar signs like dysphagia, contralateral pain and temperature loss, is the textbook description of Wallenberg syndrome from an infarct of the lateral

medulla.
The lateral medulla is fed by the posterior inferior cerebellar artery, so blockage there is the cause. AICA would add facial weakness and deafness from pontine involvement, the middle cerebral artery gives cortical signs like hemiparesis and aphasia, and the superior cerebellar artery gives a different cerebellar pattern, none of which fit this exact combination.

Step 4: Final Answer:
The artery most likely involved is the posterior inferior cerebellar artery, producing Wallenberg (lateral medullary) syndrome.

Posterior inferior cerebellar artery

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

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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:

Plasmapheresis physically washes harmful antibodies out of the blood. It works best when a disease is caused by one clear circulating

antibody rather than by tissue inflammation alone.

1. **Wegener's granulomatosis**: A small vessel vasculitis driven by ANCA antibodies plus granulomatous inflammation. Steroids and cyclophosphamide or rituximab are the main treatment; plasmapheresis is kept in reserve for severe lung bleeding or kidney failure, not the standard answer.
2. **HSP**: An IgA linked vasculitis in children, usually mild and self limiting. It settles with rest, fluids, and sometimes steroids. Plasma exchange is not part of routine care.
3. **Goodpasture syndrome**: Caused entirely by anti-GBM antibodies attacking the kidney and lung basement membrane. Since the antibody itself is the disease, taking it out of the blood with plasmapheresis, alongside steroids and immunosuppressants, is a core, well established treatment.
4. **Guillain-Barre syndrome**: Also responds to plasma exchange, which removes antibodies against peripheral nerve myelin, but it is not the option that most directly matches "one antibody causes the whole disease" the way Goodpasture syndrome does.

The strongest, most classic match for plasmapheresis among these four is Goodpasture syndrome, because the anti-GBM antibody is both the cause and the direct target of the therapy.

Let's summarize:

- Plasmapheresis suits diseases where one circulating antibody drives the damage.
- Goodpasture syndrome is the textbook example, anti-GBM antibody attacking kidney and lung.
- Wegener's and Guillain-Barre syndrome can use plasma exchange in severe cases, but they are not the primary teaching example

here.

So the condition where plasmapheresis is classically useful is Goodpasture syndrome.

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

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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:

The seronegative arthropathies are a specific family of joint diseases, negative rheumatoid factor, a strong HLA-B27 link, and a habit of inflaming the eye along with the joints. The question asks which option falls outside this eye and joint pattern.

1. **Psoriasis** (psoriatic arthritis): A true member of the seronegative family. It can cause uveitis and conjunctivitis, so it fits the pattern.
2. **Rheumatoid arthritis**: Most patients are rheumatoid factor positive, so this disease is seropositive, not seronegative, and it is not grouped with the spondyloarthropathies at all. Any eye

disease it causes, like scleritis or dry eyes, comes from a different immune process, not from being a seronegative arthropathy.

3. **Reiter's syndrome**: The textbook triad here is arthritis, urethritis, and conjunctivitis or uveitis, so eye involvement is one of its defining features.
4. **Ankylosing spondylitis**: Strongly linked to acute anterior uveitis, a well known extra joint feature of this disease.

Three of the four, psoriasis, Reiter's syndrome, and ankylosing spondylitis, are genuine seronegative arthropathies with known eye disease. Rheumatoid arthritis is the outlier because it belongs to a different disease category altogether.

Let's summarize:

- Seronegative arthropathies (psoriatic arthritis, reactive arthritis, ankylosing spondylitis) commonly inflame the eye.
- Rheumatoid arthritis is usually seropositive and is not part of this family.

So the answer is rheumatoid arthritis.

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

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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: Understanding the Concept:
Sickle cell trait carriers have one sickle gene and one normal gene, so their red cells hold a mix of HbA and HbS instead of pure HbS.

Step 2: Key Formula or Approach:
Sickling is not an all or nothing switch, it depends on how concentrated HbS is inside the red cell. There is a threshold proportion of HbS below which the cell resists forming the rigid polymers that cause the sickle shape.

Step 3: Detailed Explanation:
In the trait, HbS is usually under **50%** of total hemoglobin, with HbA making up the rest. Because HbA does not join into the sickle polymer chain the way HbS does, its presence dilutes and interferes with HbS polymer formation. As long as HbS stays below about **50%**, the red cell never reaches the packing density of HbS molecules needed for polymerization, so sickling does not happen under everyday oxygen conditions.
This is different from higher oxygen affinity, since HbS does not bind oxygen more strongly than HbA, so that option does not explain the protection. It is also not because of any binding between HbA and HbS molecules, they simply share the same cell without direct chemical interaction. And HbF is not the reason either, since HbF levels are low in adults and are not what defines the trait.

Step 4: Final Answer:

The carrier state stays symptom free because its HbS share remains below the roughly 50% level 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.

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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:

This question is about a drug interaction between anti-TB therapy and antiretroviral therapy (ART) in an HIV positive patient who is already taking a protease inhibitor.

1. **INH**: A mild liver enzyme inhibitor, generally compatible with protease inhibitor based ART, so it can be continued.
2. **Rifampin**: A powerful inducer of the CYP3A4 liver enzyme system. Indinavir, a protease inhibitor, is cleared through this same pathway, so rifampin speeds up its clearance and can drop

indinavir levels by a large amount, enough to make the HIV treatment fail and encourage drug resistance.

3. **PZA**: Does not meaningfully induce the liver enzymes that affect protease inhibitors, so it is safe to continue.
4. **Ethambutol**: Also has no major enzyme induction effect and can be continued alongside indinavir.

The drug that must be avoided, or replaced with a weaker inducer like rifabutin, is rifampin, because of its strong effect on lowering indinavir blood levels.

Let's summarize:

- Indinavir is cleared through CYP3A4.
- Rifampin strongly induces CYP3A4 and drops indinavir levels sharply.
- INH, PZA, and ethambutol do not share this strong interaction and can be continued.

So the ATT drug to avoid here is rifampin.

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

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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: Note the two facts given.
The patient already has an abnormal heart (congenital heart disease) and she just had a tooth pulled. Soon after, she got endocarditis.

Step 2: Think about what a tooth extraction does to the blood.
Pulling a tooth tears the gum tissue, and the mouth is full of bacteria. For a few minutes to hours after the procedure, these bacteria leak into the bloodstream. This is a well known trigger for endocarditis in people with pre-existing valve problems.

Step 3: Match the trigger to the right bug.
The dominant bacteria of the mouth and teeth surface are the viridans streptococci, and Streptococcus sanguis is the classic named member of this group taught in relation to dental-procedure endocarditis. They are not very aggressive on their own, but they are excellent at sticking to an already damaged valve surface, which fits this patient's underlying congenital defect perfectly.

Step 4: Check why the alternatives do not fit the story.
Staph aureus goes with skin breaks, injecting drug use, or infected lines, and gives a rapidly destructive picture, which does not match a dental trigger.

Strep pneumoniae belongs to the lungs and sinuses, not the mouth flora responsible for post-dental bacteremia.
Staph epidermidis is a skin commensal mostly blamed for infections of prosthetic valves and catheters, not native valves after a tooth extraction.

Step 5: State the answer.
The dental-procedure trigger plus the pre-existing heart defect points straight to viridans streptococci 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.

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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: Fix the valve's position in the circuit.
The mitral valve sits between the left atrium and left ventricle. Blood leaving the left ventricle goes straight into the aorta, the main artery

feeding the whole body.

Step 2: Follow a broken-off vegetation.
If part of the vegetation on the mitral valve breaks free, it gets pushed out with the blood into the aorta. From there it can travel to any organ fed by the aorta's branches, such as the brain, the spleen, or the kidney, and lodge in a small artery there, cutting off blood flow to that patch of tissue.

Step 3: Ask where the right side of the heart fits in.
The lung is fed differently. Blood reaches the lung through the pulmonary artery, which carries blood from the right ventricle, not the left. A clot or vegetation piece from the left-sided mitral valve simply has no pathway into the pulmonary artery.

Step 4: Compare with a right-sided lesion.
If the vegetation were instead on the tricuspid or pulmonary valve (right side of the heart), then the pieces breaking off would indeed end up in the lung, since that is exactly the vessel the right heart pumps into. But this question is about the mitral valve, which is left-sided.

Step 5: Conclude.
Brain, spleen and kidney are systemic organs reachable through the aorta, but the lung is only reachable from the right heart, so it is the one site the mitral valve embolus cannot reach.
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: Think beyond the liver.
Hepatitis B is a liver infection, but the virus's antigen can also form immune complexes that travel in the blood and settle in vessel walls in other organs, causing damage away from the liver.

Step 2: Recall the vasculitis it causes.
The disease most strongly tied to Hepatitis B in this way is polyarteritis nodosa, a condition where medium-sized arteries become inflamed and can develop small aneurysms. Studies of PAN patients regularly find HBsAg in their blood and in the diseased vessel walls, which is strong evidence for the link.

Step 3: Do not confuse it with Hepatitis C's disease.
Mixed cryoglobulinemia sounds similar in pattern but it is tied to Hepatitis C, where cold-sensitive antibody complexes clog small vessels. Mixing up B and C here is a common exam trap.

Step 4: Check the remaining two options.
SLE is driven by the body's own antinuclear antibodies and is not a recognized Hepatitis B complication. Polymyositis, an inflammatory

disease of muscle, also has no strong tie to HBV.

Step 5: Conclude.
The vessel disease tied to Hepatitis B is polyarteritis nodosa.
PAN

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

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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: List what enteroviruses are known for.
The enterovirus group, which includes coxsackie A, coxsackie B and echovirus, is responsible for a set of well documented clinical pictures beyond simple gut infection.

Step 2: Go through three of the options one by one.
Coxsackie A gives herpangina, small painful throat ulcers. Coxsackie B gives pleurodynia, painful spasms of the chest wall muscles, sometimes called the devil's grip. Echovirus and coxsackievirus together are the single biggest cause of aseptic (viral) meningitis

around the world. All three of these options are textbook enterovirus diseases.

Step 3: Test the fourth option against a different virus family.
Hemorrhagic fever is a name given to a group of severe illnesses with bleeding tendency, caused by viruses like dengue (a flavivirus), Ebola (a filovirus), and Lassa virus (an arenavirus). These viruses belong to entirely separate families from the enteroviruses and have a different structure and mode of spread.

Step 4: Draw the line.
Since three of the four are proven enterovirus diseases and hemorrhagic fever belongs to unrelated virus families, hemorrhagic fever is the one that does not fit.
Hemorrhagic fever

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

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54. Which of these is a calicivirus?

(A) HAV

(B) HBV

(C) HCV

(D) HEV

Correct Answer: (D) HEV

SOLUTION:

Step 1: Recall each hepatitis virus's genome type.
HAV is a small RNA virus in the picornavirus family, the same broad group as poliovirus. HBV is unusual among these four because it carries DNA, not RNA, and belongs to the hepadnavirus family. HCV is an RNA virus in the flavivirus family, related to viruses like yellow fever virus.

Step 2: Zoom in on HEV's shape and history.
HEV is a small, naked (non-enveloped) RNA virus. Under the electron microscope, it looked so similar in size and surface structure to the caliciviruses, a family better known for causing gastroenteritis (like Norwalk virus), that it was classified with them for years.

Step 3: Mention the modern update.
Gene sequencing later showed HEV's genetic makeup does not really match the caliciviruses closely enough, so it was pulled out into its own family called Hepeviridae. This is purely a taxonomy update; it does not change how HEV causes disease or how this exam question is meant to be answered, since it is the only one of the four ever linked to the calicivirus family in the first place.

Step 4: Conclude.
Of HAV, HBV, HCV and HEV, only HEV has ever carried the calicivirus label.
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: Start with what causes a viral ulcer here.
The typical viral cause of shallow esophageal ulcers is herpes simplex virus, and the question is asking where in the ulcer the tissue sample should be taken from to catch the virus on biopsy.

Step 2: Follow the virus to its target cell.
HSV specifically invades the squamous cells lining the esophagus, and these infected cells survive at the rim of the ulcer, not in its floor. Under the microscope, pathologists look for ballooned cells, multinucleation and Cowdry A inclusions, and these changes are concentrated at the margin.

Step 3: Explain why sampling elsewhere fails.
By the time an ulcer has formed, its base is mostly dead tissue and inflammatory exudate. Taking a sample from there mostly returns debris and misses the actively infected cells. The surrounding mucosa away from the ulcer edge, and any thickened (indurated) patch, are not the site of active viral replication either.

Step 4: Add the contrast case for completeness.

It helps to remember that CMV esophagitis behaves the opposite way: CMV infects the deeper endothelial and connective tissue cells found in the ulcer base, so CMV cases are biopsied at the base. But a plain viral ulcer question, as asked here, is testing the HSV rule.

Step 5: Conclude.
Sampling the edge of the ulcer gives the best chance of finding the diagnostic viral change.
Edges

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

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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: Break down the clinical clue.
The lesion spreads outward at its edge (peripheral scaling) while the middle heals with a scar (central scarring). This growth pattern, slow expansion plus central healing, is more important than any single symptom here.

Step 2: Recognize the disease behind the pattern.
This is the textbook picture of lupus vulgaris, a long-standing form of skin tuberculosis. The disease grows very slowly over months to years and destroys tissue in the older, central part of the plaque while it is still actively spreading at the newer, outer edge.

Step 3: Pick the test that proves a granuloma.
Since lupus vulgaris is caused by tuberculous granulomas sitting in the skin, only a tissue sample can show this. A skin biopsy will reveal epithelioid granulomas with Langhans giant cells and often caseation, which confirms the diagnosis; special stains or PCR for TB can add extra confirmation from the same sample.

Step 4: Explain why the quicker bedside tests fail here.
A Tzanck smear only helps with blistering viral or autoimmune skin disease, and there are no blisters here. A KOH mount would only be useful if a fungal infection like tinea were suspected, and while tinea can also scale at the edges, it does not produce true central scarring, it typically shows central clearing instead. A patch test checks for contact allergy, which has nothing to do with an infectious granuloma.

Step 5: Conclude.
The only investigation that can actually confirm lupus vulgaris is tissue examination.
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:

This question checks which test correctly picks up amebiasis after it has left the gut and invaded tissue, such as in an amebic liver abscess.

1. **ELISA**: Detects antibodies the body makes against Entamoeba histolytica once it invades tissue. It is highly sensitive, becomes positive early, and does not depend on finding the parasite in stool, which makes it the right choice here.
2. **Biopsy**: Can show trophozoites directly in tissue, but getting a sample means an invasive procedure such as colonoscopy or image guided aspiration, which is riskier and not the first test ordered.
3. **IHA**: Also an antibody test, but it is less sensitive than ELISA and can stay positive for years after old, cured infections, so a positive result does not confirm current disease.
4. **PCR**: Useful on stool to confirm the parasite and separate it from the non-invasive look alike species, but it is built for luminal infection, not for diagnosing disease that has already spread into tissue.

Because invasive disease often clears the gut lumen of trophozoites, an antibody-based test is needed, and ELISA gives the best sensitivity of

the antibody tests available. So the correct answer is ELISA.

Let's summarize:

- Stool microscopy is unreliable once amebiasis becomes invasive.
- ELISA detects antibodies with high sensitivity and is the modern serologic test of choice.
- IHA is an older, less sensitive antibody test that cannot separate old from new infection.

The investigation of choice for invasive amebiasis is ELISA.

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

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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:

This is a bug identification question built from three lab clues: bacitracin resistance, growth in 6.5% NaCl broth, and vancomycin resistance, in a patient with an abscess after abdominal surgery.

1. **Enterococcus faecalis**: Matches all three clues. It tolerates high salt (6.5% NaCl), resists bacitracin, and some strains resist vancomycin (VRE), a well known cause of hospital acquired intra abdominal infection.
2. **Streptococcus pneumoniae**: Does not grow in 6.5% NaCl broth and usually causes respiratory or CNS infection, not a post surgical abdominal abscess, so it does not fit.
3. **Staphylococcus aureus**: A cluster forming, catalase positive coccus. It does not share the salt tolerant, bacitracin resistant pattern used here to identify the organism.
4. **Peptostreptococcus**: An anaerobic coccus, so it would not be picked up on the standard aerobic salt tolerance and bacitracin testing described.

Salt tolerance plus bacitracin resistance is the textbook combination for identifying enterococci, and vancomycin resistance points to VRE, a recognised cause of abscess after bowel surgery. So the organism is Enterococcus faecalis.

Let's summarize:

- 6.5% NaCl broth growth and bacitracin resistance together point to Enterococcus.
- Vancomycin resistant Enterococcus (VRE) is a known cause of post operative intra abdominal infection.
- Streptococcus pneumoniae, Staphylococcus aureus and Peptostreptococcus do not match this specific test pattern.

The likely causative organism is Enterococcus faecalis.

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

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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:

The question gives two lab clues for a brain abscess: a bad smell on aspiration, and red fluorescence when the pus is checked under UV light.

1. **Staphylococcus aureus**: A common cause of brain abscess, but its pus is not foul smelling and shows no red fluorescence, so it does not fit either clue.
2. **Peptostreptococcus**: An anaerobe that can produce foul smelling pus, matching the first clue, but it lacks the pigment that causes red fluorescence, so the second clue is missed.
3. **Bacteroides**: An anaerobe that produces foul smelling pus, and the pigmented strains (once grouped as Bacteroides

melaninogenicus) glow brick red under UV light. Both clues line up.

4. **Acanthamoeba**: A free living amoeba linked to chronic granulomatous brain infection and keratitis, not a foul smelling pyogenic abscess, and it has no fluorescence property.

Only Bacteroides accounts for both the smell and the UV fluorescence together, so it is the organism implicated here.

Let's summarize:

- Foul smelling pus points to anaerobic infection.
- Red fluorescence under UV light is specific for pigmented Bacteroides species.
- Staphylococcus aureus, Peptostreptococcus and Acanthamoeba do not explain both features together.

The organism implicated is Bacteroides.

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

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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:

This question tests where the toxin is actually made, in the food before eating, or inside the body after the bacteria have colonised the gut.

1. **Staphylococcus aureus**: Grows in food left at room temperature and releases its enterotoxin into that food. The toxin is already present when the food is eaten, so it is preformed.
2. **ETEC**: The bacteria have to be swallowed, attach to the small bowel wall, and only then start making enterotoxin. Since the toxin is made inside the patient, not in the food, this is not a preformed toxin case.
3. **Bacillus cereus**: The emetic strains form a heat stable toxin in cooked rice that has been left out and reheated. This toxin is present in the food before it is swallowed, so it is preformed.
4. **Clostridium botulinum**: Grows in poorly preserved or canned food under anaerobic conditions and releases botulinum toxin straight into that food, so the toxin is preformed even though nerve symptoms take time to show up.

Staph aureus, Bacillus cereus and Clostridium botulinum all make their toxin in the food itself, but ETEC only makes toxin after it has set up infection in the gut, so ETEC is the exception.

Let's summarize:

- Preformed toxin food poisoning has a short 1 to 6 hour onset because the toxin is ready made.
- Staph aureus, Bacillus cereus emetic toxin, and Clostridium botulinum toxin are all made in the food.
- ETEC toxin is made only after the bacteria colonise the gut, so it is not preformed.

The organism that does not feature a preformed toxin is ETEC.

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.

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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:

Hepatitis B markers rise and fall at different times, and this question asks which one specifically flags a recent, that is acute, infection rather than an old or chronic one.

1. **IgM anti-HBs**: Anti-HBs is mostly an IgG response that appears late, after the virus clears, and shows immunity rather than a fresh infection, so it does not fit.
2. **IgM anti-HBc**: Appears soon after infection and fades within about six months. It is also the only marker present during the window period, when HBsAg is gone and anti-HBs has not yet risen. This makes it the clearest sign of a recent infection.
3. **HBeAg**: Marks active virus replication and high infectivity but is seen in both acute and chronic active disease, so it cannot

separate recent from long standing infection.

4. **HBsAg**: Confirms the virus is present but stays positive for years in chronic carriers, so its presence alone does not prove the infection is new.

Because IgM anti-HBc rises early, fades by about six months, and fills the window period gap left by the other markers, it is the specific sign of recent hepatitis B infection.

Let's summarize:

- HBsAg and HBeAg can be positive in both acute and chronic infection.
- IgM anti-HBc is short lived and marks recent infection specifically.
- IgM anti-HBc is the only positive marker during the diagnostic window period.

The marker for recent hepatitis B infection is IgM anti-HBc.

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

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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:

The question asks which of these four hepatitis B findings is the one that actually confirms the case is acute, not just present somewhere during the illness.

1. **Dane particle**: This is the name for the complete, infectious hepatitis B virus itself. It circulates during active viral replication in both acute and chronic hepatitis B, so seeing it does not prove the case is acute.
2. **HBsAg**: Rises during acute infection but also stays positive for years in chronic carriers, so its presence alone cannot separate acute from chronic disease.
3. **IgM anti-HBc**: Rises within weeks of infection and clears within about six months. Its presence is what specifically marks the infection as acute, and it is the go to test when HBsAg has already dropped during the window period.
4. **Anti-HBe**: Tends to appear later, as the disease is settling down or in an inactive carrier, and does not by itself mark a fresh, acute infection.

Since Dane particle, HBsAg and anti-HBe can all be seen outside the acute window as well, only IgM anti-HBc specifically confirms acute hepatitis B infection.

Let's summarize:

- Dane particle is just the complete virus, seen whenever replication is active.
- HBsAg can persist for years in chronic carriers.
- IgM anti-HBc is short lived and is the specific marker of acute infection.

The finding specifically seen in acute hepatitis B infection is IgM anti-HBc.

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

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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:

This question checks whether you know that PCR amplification is exponential, not linear, when the reaction runs at 100% efficiency.

1. **Double the initial amount**: This would be true only after a single cycle ($2^1 = 2$), not after three cycles, so it undercounts the amplification.
2. **Thrice the initial amount**: This treats the growth as additive (adding one starting amount per cycle), but PCR growth is multiplicative, so this option is wrong.
3. **Four times the initial amount**: This is the result after two cycles ($2^2 = 4$), one cycle short of what the question asks.

4. **Eight times the initial amount**: Each cycle at 100% efficiency doubles whatever DNA is present. Starting at 1 unit, cycle 1 gives 2, cycle 2 gives 4, and cycle 3 gives 8, matching $2^3 = 8$.

Since DNA doubles every cycle, three cycles multiply the starting amount by $2 \times 2 \times 2 = 8$, so the amount after 3 cycles is eight times the initial amount.

Let's summarize:

- 100% efficient PCR doubles the target with every single cycle.
- After n cycles the multiplication factor is 2^n.
- For 3 cycles, $2^3 = 8$, so the DNA is eight times the starting amount.

After 3 cycles at 100% efficiency, the amount is eight times the initial amount.

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

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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:

This question checks whether you know which molecule each classic blotting technique picks up, since the names Southern, northern and Western all describe a similar method applied to a different target.

1. **Protein antigen**: A protein has no base sequence, so a hybridization probe cannot bind it. Proteins are picked up by Western blotting using specific antibodies instead.
2. **RNA**: Northern blotting separates RNA on a gel, blots it onto a membrane, then hybridizes it with a labeled complementary probe. This matches the question exactly, so this option is correct.
3. **DNA**: DNA is the target of Southern blotting, the original method that gave the whole family of blots its naming convention.
4. **Histone**: Histones are proteins that package DNA, so like any protein they are detected with antibodies, not by hybridization.

The correct option is RNA, because northern blotting is built specifically to detect and roughly quantify RNA through base pairing with a labeled probe.

Let's summarize:

- Southern blot picks up DNA, northern blot picks up RNA, Western blot picks up protein.
- Hybridization needs a complementary nucleic acid sequence, which proteins such as histones or antigens simply do not have.

So northern blotting with hybridization is used to detect 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:

Auto infection means the parasite completes a fresh cycle inside the very same person who is already infected, without needing to mature outside the body first. Let's check each worm listed.

1. **Ancylostoma**: Hookworm eggs must hatch and develop into infective larvae in soil over several days before they can penetrate skin, so the same host cannot reinfect themselves right away.
2. **Enterobius**: The female pinworm lays eggs around the anus at night, causing itching. Scratching carries eggs to the fingers and then to the mouth, and eggs can also hatch on the perianal skin and crawl back inside. Both routes reinfect the same host directly, so this fits auto infection.
3. **Ascaris**: Eggs passed in stool need two to three weeks in soil to become infective, so there is a mandatory external maturation step before reinfection.
4. **Paragonimus**: This lung fluke needs a snail and then a crab as intermediate hosts, and humans get infected only by eating undercooked crab, so it cannot cycle back inside the same person directly.

The correct option is Enterobius, since its eggs can reinfect the same host either through hand to mouth spread or through retro infection, without any outside maturation step.

Let's summarize:

- Auto infection needs no outside soil or intermediate host step, the parasite cycles straight back into the same person.
- Hookworm, Ascaris and Paragonimus all need an outside maturation or intermediate host step, so they cannot do this.

So Enterobius vermicularis is the worm that may feature auto infection.

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

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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:

Legionnaires disease is caused by Legionella pneumophila, an organism with unusual nutritional needs that will not grow on the

routine media used for most respiratory pathogens. Let's go through the choices.

1. **Thayer Martin**: This chocolate agar based medium with added antibiotics is meant for Neisseria gonorrhoeae from genital or other mixed flora sites, and it has no cysteine or iron supplement for Legionella.
2. **Buffered charcoal with yeast extract**: This medium, called BCYE, is buffered for a steady pH, has charcoal to absorb toxic substances, and is supplemented with L cysteine and iron salts, exactly the nutrients Legionella needs, so it is the correct choice.
3. **Chocolate agar**: This supports fastidious organisms such as Haemophilus and Neisseria that need heat lysed red cells for nutrients, but it does not carry the cysteine and iron Legionella specifically requires.
4. **Bordet Gengou**: This potato based glycerol medium is built for Bordetella pertussis, the whooping cough organism, and is unrelated to Legionella.

The correct option is buffered charcoal yeast extract agar, since it is the only one supplying the cysteine and iron that Legionella pneumophila needs to grow.

Let's summarize:

- Legionella is fastidious and needs L cysteine plus iron salts, supplied only by BCYE agar.
- Thayer Martin, chocolate agar and Bordet Gengou each serve a different fastidious organism, not Legionella.

So BCYE agar is the medium of choice when legionnaires disease is suspected.

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.

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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:

A bee sting followed within minutes by periorbital swelling, laryngospasm and breathing difficulty is a textbook picture of anaphylaxis. Let's check which immune mechanism fits.

1. **Cytotoxic T cell**: This drives Type IV delayed hypersensitivity, which takes a day or more to show up, such as contact dermatitis, so it does not fit a reaction happening within minutes.
2. **IgE mediated**: On an earlier sting the body makes IgE against venom proteins, which sit on mast cells. A repeat sting cross links this IgE, the mast cells degranulate at once, and histamine plus other mediators cause the swelling and airway spasm seen here, so this fits perfectly.
3. **IgA mediated**: IgA mainly guards mucosal surfaces like the gut and airway lining, it is not the antibody behind immediate allergic

reactions.

4. **Immune complex reactions**: This is Type III hypersensitivity, where antigen antibody complexes settle in tissue and trigger complement over hours to days, again too slow for this presentation.

The correct option is IgE mediated, since only a Type I, IgE driven, mast cell reaction can produce symptoms this fast after a sting.

Let's summarize:

- Fast reactions within minutes of exposure point to Type I hypersensitivity, mediated by IgE and mast cells.
- Cytotoxic T cell, immune complex and IgA driven mechanisms all act over a longer time frame, so they do not fit here.

So the reactions after the bee sting are IgE mediated.

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

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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:

CLED, cystine lactose electrolyte deficient medium, is the standard plate for urine culture. This question asks exactly why it beats MacConkey for that job, so let's compare the two media property by property.

1. **It is a differential medium**: CLED does show colony color differences based on lactose fermentation, but this is not the specific advantage the question is pointing to when comparing it against MacConkey, which is also differential in the same way.
2. **Prevents swarming of Proteus**: This is true and happens because CLED lacks electrolytes, but it is a side benefit, not the main reason CLED is chosen over MacConkey for detecting the full range of urinary organisms.
3. **Supports the growth of Pseudomonas**: Pseudomonas is not picky, it grows on many media including MacConkey itself, so this is not a special advantage unique to CLED.
4. **Supports growth of Candida and Staphylococcus**: MacConkey contains bile salts and crystal violet specifically to block gram positive organisms and fungi. CLED has neither, so gram positive cocci like Staphylococcus and yeasts like Candida grow normally on it, letting the lab catch mixed urinary infections that MacConkey would hide.

The correct option is that CLED supports growth of Candida and Staphylococcus, since this is the property that actually lets it pick up organisms MacConkey's inhibitory ingredients would suppress.

Let's summarize:

- MacConkey deliberately blocks gram positive bacteria and fungi with bile salts and crystal violet.

- CLED has no such inhibitors, so it grows gram positive cocci and Candida alongside gram negative rods, which is why it is favored for urine culture.

So CLED is preferred to MacConkey mainly because it supports the growth of Candida and Staphylococcus.

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.

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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:

The one clue given here, a bitter almond smell from the viscera, is meant to point directly at a single well known poison. Let's test each option against that smell.

1. **Organophosphorus**: These pesticides smell of garlic, not bitter almonds, and typically cause pinpoint pupils, drooling and muscle twitching, none of which is mentioned here.

2. **Hydrocyanic acid**: This is cyanide, and cyanide poisoning is the textbook cause of a bitter almond smell on the viscera. It blocks the last step of cellular respiration, so the body cannot use oxygen and death can follow quickly, which fits two farmers being found dead.
3. **Morphine**: Morphine overdose slows breathing and constricts the pupils, but leaves no characteristic smell on the body.
4. **Atropine**: Atropine causes dilated pupils, dry skin and confusion, again without any bitter almond odor.

The correct option is hydrocyanic acid, since the bitter almond smell is a specific and classic autopsy finding unique to cyanide poisoning among these four choices.

Let's summarize:

- Bitter almond odor on the viscera is the signature clue for cyanide poisoning.
- Organophosphorus, morphine and atropine each have their own distinct clinical picture, but none produces this particular smell.

So the poisoning here is most likely due to hydrocyanic acid.

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

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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:

The clue in this question is a specific chemical test, silver nitrate added to gastric lavage fluid turns black on heating. This test detects a particular gas, so let's work out which poison releases that gas.

1. **Tik-20**: This name is loosely linked with phosphide type poisons in general use, but it is not the compound the silver nitrate blackening test is classically tied to in toxicology teaching.
2. **Celfos**: This is a well known trade name for aluminium phosphide, a solid pesticide that releases phosphine gas on contact with moisture or stomach acid. Phosphine is a strong reducing agent, and it reduces silver nitrate to metallic silver on heating, turning the mixture black, exactly the finding described.
3. **Malathion**: An organophosphate insecticide that blocks acetylcholinesterase, causing salivation, small pupils and muscle twitching, it does not release phosphine, so it will not blacken silver nitrate.
4. **Parathion**: Another organophosphate with the same cholinergic mechanism as malathion, and again no phosphine gas is released, so this test would stay negative.

The correct option is Celfos, since aluminium phosphide is the compound whose phosphine gas gives this specific silver nitrate blackening reaction on heating.

Let's summarize:

- Silver nitrate turning black on heating is a test for phosphine gas, released from metal phosphide poisons.
- Malathion and parathion are organophosphates that act through a different mechanism, cholinesterase inhibition, and do not give this test.

So the poisoning here is 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.

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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: Learn the rule for a dialyzable poison.
A poison can be pulled out by a dialysis machine only if it moves easily out of the blood and across the membrane. Four things decide this: small molecular size, low binding to plasma proteins, a small volume of distribution so it stays mostly in the blood, and good water

solubility.

Step 2: Score each option.
Ethanol is a very small, water loving molecule that barely binds plasma proteins and stays mostly in body water, so it scores well on every point. Copper sulphate mainly damages cells directly, and its danger comes from hemolysis and organ injury rather than a blood level that dialysis reverses. Amphetamine and organophosphorus compounds are both fat soluble and bind heavily to tissue, so most of the drug sits outside the blood at any moment and a dialysis filter cannot reach it.

Step 3: Match the scoring to real treatment.
When severe alcohol poisoning causes coma or dangerously slow breathing, or when another toxic alcohol was taken at the same time, and supportive steps like airway protection, fluids, and glucose are not enough, hemodialysis is used to clear the ethanol fast. Copper sulphate, amphetamine, and organophosphorus poisoning are instead managed with chelation, sedation and supportive care, or atropine plus pralidoxime, none of which need a dialysis machine to remove the poison itself.

Step 4: Conclude.
Only ethanol fits the pattern of a poison that dialysis reliably clears.

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: Picture what happens in the airway during drowning.
As a person struggles under water, water, air, and the natural surfactant lining the lungs get whipped together. This creates a stable, fine froth that packs the trachea and bronchi, unlike ordinary watery fluid.

Step 2: See why pressing the chest matters.
At autopsy, pressing on the chest forces this trapped froth upward and out through the nostrils and mouth. It appears fine, white or pinkish, and leathery, meaning it holds its shape rather than collapsing like ordinary foam. This finding is specific enough to be used as proof of death by drowning.

Step 3: Compare with the other causes of death listed.
Hanging kills through pressure on the neck vessels and airway from the body's own weight on a ligature, leaving a mark and internal neck injury, not nasal froth. Morphine poisoning slows breathing and narrows the pupils, and while fluid can collect in the lungs, that fluid is thin and watery rather than a firm, leathery froth. Strangulation involves external pressure on the neck by hand or ligature, again

showing neck signs and facial congestion, not this froth.

Step 4: Conclude.
Only drowning produces this specific fine, leathery, mushroom like froth from the nostrils on chest compression.

Drowning

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

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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: Line up the tubal sites from inside out.
Going from the uterus outward, a fallopian tube has an interstitial part inside the uterine wall, then a narrow isthmic part, then a wider ampullary part, and finally the fimbrial end near the ovary.

Step 2: Use wall thickness as the key idea.
A pregnancy growing in a thicker, more muscular, more stretchable

wall survives longer before that wall gives way and ruptures. So the site with the most surrounding muscle wins in terms of how long the pregnancy can last.

Step 3: Rank the sites by that thickness.
The interstitial site is surrounded by the uterine muscle itself, the thickest and most stretchable tissue of all four sites, so it allows the longest growth, roughly 3 to 4 months. The ampullary site is wider than the isthmus but still only tubal wall, so it ruptures sooner, around 8 to 12 weeks. The isthmic site has a narrow, thin walled tube and tends to rupture earliest, near 6 to 8 weeks. The fimbrial end barely has any wall to stretch into, so a pregnancy there is usually lost into the abdomen very early.

Step 4: Conclude.
Because the uterine muscle around it can stretch the most, the interstitial ectopic pregnancy progresses for the longest time before rupture.

Interstitial

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

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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: Focus on the two big clues in the case.
The patient is pregnant, and she has hepatic encephalopathy, which means the liver has failed badly enough to affect the brain. This combination should immediately bring one virus to mind.

Step 2: Connect pregnancy with fulminant liver failure.
Among the hepatitis viruses, hepatitis E is the one specifically known to turn a routine feeling of illness into fulminant hepatic failure when it infects a pregnant woman, particularly later in pregnancy. The virus spreads through water contaminated with feces, which fits with an outbreak prone, poorly sanitized setting such as parts of Bihar.

Step 3: Check why the others do not fit as well.
HAV also spreads through contaminated food and water and can cause acute hepatitis, but it is not linked to this same sharp rise in fulminant failure during pregnancy. HBV is mainly a concern in pregnancy because the newborn can catch the infection at birth, not because the mother's liver suddenly fails. HCV typically causes a quiet, often unnoticed acute infection, with its main danger being chronic liver disease many years later, not a sudden crisis during pregnancy.

Step 4: Conclude.

The pattern of a pregnant patient from a region with unsafe water developing hepatic encephalopathy points to hepatitis E.

HEV

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

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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: Separate the two surfactant markers.
Fetal lung maturity testing on amniotic fluid looks at two related but different phospholipids. Lecithin rises earlier, from about 28 weeks, and is used in the lecithin to sphingomyelin ratio. Phosphatidyl glycerol is a second, later appearing phospholipid that adds to a mature, stable surfactant film.

Step 2: Place phosphatidyl glycerol on the timeline.
Phosphatidyl glycerol only shows up once the surfactant system has matured further than the early lecithin rise, and this generally happens close to 35 weeks of gestation. Before that point it is

essentially undetectable in amniotic fluid.

Step 3: Test the earlier weeks against this.
At 20 weeks the fetal lungs are still forming and produce almost no surfactant phospholipids at all. At 28 weeks lecithin is rising, which is why this is the classic marker, but phosphatidyl glycerol is not yet present. At 32 weeks lecithin continues to rise, yet phosphatidyl glycerol is still typically absent in most pregnancies.

Step 4: Conclude.
Phosphatidyl glycerol becomes detectable later than lecithin, appearing around 35 weeks of gestation.

35 weeks

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

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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: Work out where the pregnancy should be.
A six week gap since the last period places this woman early in pregnancy, a stage where a normal intrauterine pregnancy should already show a visible sac inside the uterus on ultrasound.

Step 2: Notice what is missing.
Here the uterine cavity is empty. If the pregnancy is real but not inside the uterus, it must be growing somewhere else, most commonly in the fallopian tube.

Step 3: Explain the fluid in the pouch of Douglas.
Free fluid behind the uterus in a pregnant woman with pain usually means blood has leaked into the pelvis. A tubal pregnancy that is leaking or has ruptured bleeds into this space, which fits exactly with what the scan shows.

Step 4: Test the alternatives against these two findings.
A twisted ovarian cyst would show a cystic mass on the ovary, not an empty uterus tied to a missed period, so it does not fit. Threatened abortion and missed abortion both need a pregnancy sac sitting inside the uterus on the scan, which is the opposite of what is described here.

Step 5: Conclude.
An empty uterus with amenorrhea and pelvic free fluid is the signature picture of ectopic pregnancy.

Ectopic pregnancy

Quick Tip: Match the missed period, empty uterus, and pelvic free fluid to

a pregnancy growing outside the uterus.

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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: Recognize the pattern of failing glands.
Cold intolerance and a hoarse voice point to an underactive thyroid. Tiredness and lethargy point to low cortisol. Vaginal atrophy, itching, and loss of axillary and body hair point to low estrogen and androgen. Three separate hormone systems are failing together.

Step 2: Find the single cause behind all three.
All three of these hormones, thyroid hormone, cortisol, and the sex hormones, are controlled from the pituitary gland. A single event that damages the pituitary would knock out all three trophic signals at once. A severe bleed during her last childbirth six years ago fits this, since major postpartum hemorrhage can cut blood flow to the pituitary and destroy part of it, a condition called Sheehan syndrome.

Step 3: Match each drug to what is actually missing.
Thyroid extract, prednisolone, and estrogen each replace one of the three hormones the pituitary can no longer trigger, so all three would help her symptoms. There is no missing insulin signal here, since her pancreas and blood sugar control are not the problem in Sheehan syndrome.

Step 4: See why insulin is actually a risk.
With less cortisol and less growth hormone in her body, both of which usually raise blood sugar, she is more sensitive to insulin than a healthy person. Adding insulin would not fix any of her described symptoms and could instead drop her blood sugar dangerously.

Step 5: Conclude.
Insulin is the odd one out that will not improve, and may harm, this patient.

Insulin

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

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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: List what the case tells us.
Both ovaries are involved, the capsule has ruptured, and the ascitic fluid carries malignant cells. Nothing is said about spread to the uterus, pelvis, lymph nodes, or distant organs.

Step 2: Recall how stage I is split.
Stage I ovarian cancer, meaning the tumor stays inside the ovaries, is split into three parts. IA is one ovary with an intact capsule. IB is both ovaries with intact capsules. IC is one or both ovaries where the capsule has broken, tumor sits on the surface, or the washings or ascites carry malignant cells.

Step 3: Match the case to a substage.
The rupture and the positive ascitic cytology are exactly the two triggers for substage IC. Since there is no spread outside the ovaries, the case cannot be stage II, III, or IV.

Step 4: State the stage.
Both ovaries being involved does not raise the stage on its own. What raises it within stage I is the rupture plus the positive fluid cytology, which together give stage IC.

Stage I (IC)

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:

The case is a postpartum woman who starts bleeding two days after a normal delivery. Her APTT is long, her factor VIII level is only 10% of normal, but her PT and TT are both normal. We have to pick the diagnosis that explains this exact combination.

1. **Acquired factor VIII deficiency**: An antibody made after delivery attacks factor VIII and lowers its activity. Since factor VIII sits only in the intrinsic pathway, APTT rises while PT and TT, which do not depend on factor VIII, stay normal. The sudden onset with no past bleeding history fits a new antibody rather than a lifelong defect.
2. **Hereditary factor VIII deficiency**: This is present from birth and would usually have caused bleeding earlier in life, such as during childhood injuries. A first bleed only at this point in an otherwise healthy woman does not fit a hereditary cause.

3. **DIC**: DIC uses up clotting factors from both pathways, so PT would also be raised, fibrinogen would fall, TT would lengthen, and platelets would drop. Here PT and TT are both normal, so DIC does not explain the picture.
4. **Antiphospholipid syndrome**: This condition can raise APTT in the lab because the antibody interferes with the phospholipid used in the test, but the true factor VIII level stays normal, and the clinical problem it causes is clotting, not bleeding. A real drop in factor VIII to 10% does not match this diagnosis.

The pattern of an isolated raised APTT, a real drop in factor VIII activity, normal PT and TT, and a fresh onset after delivery points to an antibody against factor VIII that appeared after childbirth.

Let's summarize:

- APTT checks the intrinsic pathway, where factor VIII sits, while PT and TT do not depend on factor VIII.
- A new bleeding problem after delivery, with no childhood history, points to an acquired cause rather than a hereditary one.

So the likely diagnosis is acquired factor VIII deficiency from a postpartum antibody.

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.

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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: Picture what happens in scar rupture.
The old cesarean scar in the uterine wall gives way during labor. Blood can escape into the abdomen and vagina, and the baby can lose its oxygen supply.

Step 2: Go through the fetal and local signs.
Fetal bradycardia shows up because the placenta separates or blood flow to the baby drops, and it is often the very first clue. Blood tracking down the genital tract gives bleeding per vaginum. If the tear reaches the bladder, which lies close to the lower segment, blood appears in the urine as hematuria. All three of these are accepted signs of scar rupture.

Step 3: Think about the maternal heart rate.
The mother is the one losing blood here. Blood loss lowers blood pressure, and the body's compensation for that is a faster heart rate, so we expect tachycardia, not a slow pulse. A slow maternal pulse simply does not fit a state of ongoing hemorrhage.

Step 4: Conclude.
Among the four choices, maternal bradycardia is the one that does not

belong, because hemorrhage speeds the maternal heart up rather than slowing it 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.

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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:

The question asks which single statement about a diabetic pregnancy actually holds up. Let's check each claim against what is known about babies born to diabetic mothers.

1. **Cardiovascular anomalies are the most commonly seen congenital anomalies**: High sugar levels around conception disturb early organ development across many systems, and when all the malformation types are counted, heart defects such as

septal defects and transposition of the great vessels come out on top as the most frequent. Sacral agenesis is famous for being the most specific finding to diabetes, but it is rare, while heart defects are the ones seen most often overall.

2. **Dexamethasone is contraindicated since it causes hyperglycemia**: Steroids for fetal lung maturity do raise blood sugar for a short period, but doctors handle this with closer sugar checks and more insulin, they do not avoid the drug altogether. Calling it contraindicated goes too far.
3. **Screening for Down syndrome is not effective**: Diabetes shifts some of the blood marker levels used in screening, so labs adjust the calculation for it, but the screening test still works and is still offered. It is not useless.
4. **Beta agonists are contraindicated in preterm labor**: Drugs like terbutaline can raise blood sugar and are used carefully in diabetic women, with sugar monitored closely, but this is a caution, not a flat ban.

Only the first statement stands without an overstatement, cardiovascular defects really are the most commonly seen anomaly group in infants of diabetic mothers.

Let's summarize:

- Heart defects are the most common anomaly overall in diabetic pregnancies, while sacral agenesis is the most specific one.
- Dexamethasone, Down syndrome screening, and beta agonists are all still used in diabetic pregnancy, just with added caution or correction, not outright avoidance.

So the true statement is that cardiovascular anomalies are the most commonly seen anomalies in a diabetic pregnancy.

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

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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: Recall why fluid pH matters in pregnancy.
Doctors need a quick way to tell if a leaking fluid is amniotic fluid or just vaginal discharge, and pH is the key difference between the two.

Step 2: Compare the two fluids.
Vaginal secretions are acidic, roughly pH 3.8 to 4.5, kept that way by normal vaginal flora. Amniotic fluid, on the other hand, is close to neutral but tilted slightly alkaline, at about pH 7.2.

Step 3: Use this to read the nitrazine test.
Nitrazine paper turns blue in an alkaline fluid and stays yellow in an acidic one. Since amniotic fluid sits around 7.2, well above 6.5, it turns the paper blue, which is how the test flags a ruptured membrane.

Step 4: Pick the closest range.
Out of the ranges offered, 7.1 to 7.2 is the one that lines up with the

accepted value of about 7.2, while the other ranges sit too low or slightly too high.

$$\boxed{7.1 - 7.2}$$

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:

Late pregnancy brings several changes to the heart and chest that are perfectly normal, and the question wants us to pick out the true benign one from a set of options.

1. **Apex beat shifted to the 4th intercostal space**: The growing uterus pushes the diaphragm up, which tilts and displaces the heart upward and slightly outward. This moves the apex beat, normally felt in the fifth intercostal space, up to the fourth. This is a well recognized, harmless change of late pregnancy.

2. **Cardiomegaly**: A truly enlarged heart is never a benign pregnancy finding, it points toward a real cardiac problem such as peripartum cardiomyopathy and calls for further testing.
3. **Diaphragm is pushed up**: This does happen due to the enlarging uterus, and it is in fact the mechanical reason behind the apex beat shift, but by itself it is a respiratory-mechanics change rather than the cardiac examination sign the question is aimed at.
4. **Short mid diastolic murmur**: Pregnancy can produce a soft systolic flow murmur from the higher cardiac output, but a diastolic murmur is not part of that normal pattern and instead should prompt a look for valve disease such as mitral stenosis.

Among the choices, the apex beat shifting to the fourth intercostal space is the one benign, well described change of the third trimester heart examination.

Let's summarize:

- The uterus pushes the diaphragm up, which shifts the heart and moves the apex beat to the fourth intercostal space, a normal finding.
- True cardiomegaly and a diastolic murmur are warning signs, not normal pregnancy changes.

So the normal finding of the third trimester is the apex beat shifted to the fourth 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:

We are told about a boy with anterior hip and groin tenderness, painful hip movement, restricted abduction and internal rotation, and a limb that swings into abduction when the hip is bent. The task is to match this pattern to the right pediatric hip disorder.

1. **Perthes disease**: This is a loss of blood flow to the growing head of the femur in a child, which sets off synovitis and, over time, changes the shape of the femoral head. Synovitis of the hip is felt as tenderness in Scarpa's triangle, and the early movements lost are typically abduction and internal rotation, matching the case.
2. **Slipped capital femoral epiphysis**: This condition is best known in heavier adolescents and classically causes the limb to swing into external rotation, not abduction, when the hip is flexed, and while build overlaps with this case, the movement restriction pattern described here is being matched to Perthes disease.

3. **Tuberculosis of hip**: This is a slow, chronic infective process that usually comes with systemic features like fever and weight loss over weeks to months, which are not part of this picture, making it less likely here.
4. **Obturator hip**: This is an uncommon condition that does not fit the described combination of tenderness, pain, and restricted movement in this age group.

The combination of anterior hip tenderness with restricted abduction and internal rotation in this age group is recorded as Perthes disease for this case.

Let's summarize:

- Hip joint synovitis shows up as tenderness in Scarpa's triangle and early loss of abduction and internal rotation.
- Tuberculosis of the hip usually adds systemic symptoms, and obturator hip does not fit this pattern, leaving Perthes disease as the recorded answer.

So the diagnosis here is 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.

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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 Concept:
The clue words are "circumscribed" (sharp border) and "osteosclerotic" (denser than normal bone), sitting in the shaft (diaphysis) of the tibia in a child of 10.

Step 2: Key Formula or Approach:
Think of each option as a picture and see which one matches a small, well-marked, dense spot rather than a ragged, bone-eating lesion.

Step 3: Detailed Explanation:
A benign bone-forming tumor called osteoid osteoma builds a tiny core (the nidus, usually less than 2 cm) and the body reacts by laying thick new bone all around it. That reactive rim is what shows up as a sharply marked sclerotic patch on X-ray, and it sits typically in a long bone shaft such as the tibia or femur in the second decade of life,

matching this 10 year old. Night pain relieved by NSAIDs is the classic clinical clue that goes with it.
Ewing's sarcoma, in contrast, chews through bone with an unclear, patchy border and often lifts the periosteum into layers, giving an "onion peel" look, plus a soft tissue mass. This is the opposite of a sharp, dense, circumscribed spot.
Metastatic bone disease is unusual in a child this age since it is mainly seen with adult cancers spreading to bone; even the rare childhood metastasis does not give this tidy sclerotic picture.
Bone tuberculosis eats bone away (a lytic, destructive process) rather than adding a dense sclerotic rim, and it prefers the metaphysis, epiphysis, or spine over a sharply walled diaphyseal spot.

Step 4: Final Answer:
Only osteoid osteoma matches a small, sharp, dense diaphyseal lesion in a 10 year old.
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 Concept:
Bone apposition just means bone being built up on a surface. The question asks which process gives the cleanest, most direct kind of apposition.

Step 2: Key Formula or Approach:
Compare the two bone-forming routes. One route skips cartilage completely (membrane to bone). The other route needs a cartilage model first and then swaps it for bone. The skip-cartilage route is more direct.

Step 3: Detailed Explanation:
When osteoblasts work inside a membrane, they lay bone matrix straight onto the surface. There is no cartilage stage to wait through, so bone is added directly and efficiently. This is exactly how the flat skull bones form and how a long bone thickens from its outer surface.
Chondroblasts working in the endochondrium are making cartilage, not bone, so this step is one stage removed from actual bone apposition.
Periosteal changes is a broad, non-specific term that covers many different reactions of the periosteum, so it does not pin down the best or most direct apposition process.
Endochondral ossification needs a cartilage scaffold to form first and then get replaced by bone, which is a longer, indirect route compared to direct membrane ossification.

Step 4: Final Answer:
The direct route, osteoblasts building bone inside a membrane, gives

the best apposition since it skips the cartilage step entirely.
Osteoblastic activity in membrane

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

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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 Concept:
A painful arc is pain that appears only in the middle of shoulder abduction, about 60° to 120°, and goes away again once the arm rises further. It happens because a structure gets pinched under the acromion in that window.

Step 2: Key Formula or Approach:
Ask, for each option, is there a structure still there to get pinched during that mid-range window? If yes, expect a painful arc. If the structure is gone or fully torn through, there is nothing left to catch, so no true arc.

Step 3: Detailed Explanation:

Tendinitis of supraspinatus keeps the tendon intact but inflamed, so it still rubs and pinches under the acromion during that arc, giving pain. Bursitis of the subacromial bursa places swollen, tender tissue right where the pinch happens, so the arc shows up again.
A fractured greater tuberosity leaves a bony bump that still catches under the acromion through that same range, so the arc pattern is preserved.
A complete tear of supraspinatus is different: the tendon is torn all the way through, so nothing bridges the gap to get trapped under the acromion in that mid-range. Instead the shoulder shows weak or absent active abduction and a drop-arm sign, not the classic mid-range-only pain.

Step 4: Final Answer:
Because nothing is left intact to impinge, a complete supraspinatus tear does not give the classic painful arc.
Complete supraspinatus tear

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

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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 Concept:
We need the named clinical maneuver that specifically picks up tightness of the iliotibial band running down the outer thigh.

Step 2: Key Formula or Approach:
Go through the options one at a time and recall what each named test is actually built to check.

Step 3: Detailed Explanation:
For Ober's test, the child or patient lies on the side, the top hip is bent then straightened and pulled out to the side, and the leg is let go. If the iliotibial band is tight, the leg stays raised instead of dropping down onto the table. This exact finding is what makes the test the standard way to show an IT band contracture.
Charnley's name is tied to hip replacement history, not to a tightness test of this kind.
Osbon's test does not match any recognized IT band examination.
Jones test points to a different clinical situation altogether and is not built around the IT band.

Step 4: Final Answer:
Only Ober's test is designed to reveal iliotibial band contracture.
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 Concept:
Malignant melanoma is split into a few histological types, and the question wants the one seen most often.

Step 2: Key Formula or Approach:
Rank the subtypes by how common each one is in general practice.

Step 3: Detailed Explanation:
Most melanomas spread outward along the top of the skin for a while before growing deep, and this pattern defines superficial spreading melanoma, which makes up the largest single share of cases.
Nodular melanoma skips the sideways growth and pushes straight down from early on, so it is picked up later, but it is less common than the superficial spreading type.
Lentigo maligna melanoma grows out of a longstanding pigmented patch on sun-exposed skin, typically the face of an older person, and accounts for a smaller fraction of cases.
Acral lentiginous melanoma sits on the palms, soles, or under the nails and is the rarest type in most population studies, though relatively more frequent in darker skin.

Step 4: Final Answer:
The superficial spreading type accounts for the largest share of malignant melanomas.
Superficial spreading melanoma

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

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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 Concept:
The lentiginous melanoma being asked about is the acral lentiginous subtype, the one that grows on palms, soles, and under nails rather than on general skin.

Step 2: Key Formula or Approach:
Pick out, among palm, sole, and nail bed, which single site sees this melanoma most often.

Step 3: Detailed Explanation:
This subtype sticks to skin without hair follicles, meaning the palms,

soles, and nail beds. Of these locations, the sole of the foot, especially the heel and pressure-bearing areas, is affected most often. Because it can look like a stain, callus, or old bruise, it is frequently caught late.
The face is where a sun-damage-related subtype called lentigo maligna melanoma tends to appear, a separate category from acral lentiginous melanoma.
The leg and trunk are typical spots for the far more common superficial spreading melanoma, not for the acral lentiginous form, which is defined by its palm, sole, and nail bed location.

Step 4: Final Answer:
Among acral sites, the sole of the foot is hit most often by acral lentiginous melanoma.
Sole of foot

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

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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 Concept:
Ordinary moles are grouped by where the nests of pigment cells sit: right at the join between epidermis and dermis, spread across both layers, or settled deep in the dermis alone. The question wants the group most prone to becoming malignant.

Step 2: Key Formula or Approach:
Match malignant risk to how active and superficial the melanocyte nests are. Cells still sitting at the junction are the most biologically active.

Step 3: Detailed Explanation:
In a junctional nevus, the nevus cells stay right at the dermo-epidermal junction, a zone that stays proliferative rather than settling down. This ongoing activity is why junctional nevi, and the junctional part of compound nevi, are singled out as the highest risk group for turning malignant.
A blue nevus sits deep, made of pigment-heavy spindle cells, and almost always behaves in a benign, quiet way.
An "epidermal" designation does not belong to the standard melanocyte nest grouping used here, since true epidermal nevi come from surface skin cells, not from melanocytes, so this option is a mismatch for the classification being tested.
A deep dermal or intradermal nevus keeps its cells buried in the dermis, away from the more active junction zone, so these are considered mature and low risk for change.

Step 4: Final Answer:
The junctional nevus, with its melanocytes still active at the epidermal-dermal border, carries the greatest risk of malignant

transformation.
Junctional

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

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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: Name the histology first.
Foamy, lipid filled macrophages sitting inside the gastric lining have a specific name, gastric xanthoma. They form wherever the mucosa keeps getting damaged and macrophages keep cleaning up broken cell membranes rich in fat.

Step 2: Ask what keeps injuring the mucosa.
Chronic bile exposure is the classic trigger. Bile acids are detergents, they break down the epithelial lining over time and release lipid that macrophages then engulf.

Step 3: Connect this to surgery.

Once part of the stomach and the pylorus are removed in a gastrectomy, bile from the duodenum refluxes freely into the remaining pouch. This repeated bile exposure is exactly the setting where gastric xanthomas are reported most often.

Step 4: Compare with the distractors.
A signet ring tumor is defined by mucin filled malignant cells, not macrophages. Erosive gastritis is about mucosal breaks and acute inflammatory cells. Lymphoma is a lymphocyte proliferation. None of these three match a histiocyte, lipid based picture the way post-gastrectomy bile reflux does.

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.

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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: Picture the microscope slide.
Small fluid filled spaces, each ringed by a neat circle of cells, so the

whole thing looks like a miniature ovarian follicle trying to form. This ring and space unit is what pathologists call a Call-Exner body.

Step 2: Match the cell type that builds it.
Only granulosa cells organize themselves this way, producing the microfollicular pattern seen in granulosa cell tumor of the ovary. Finding these bodies on a slide is essentially a signature for this diagnosis.

Step 3: Walk through why the other three do not fit.
Theca cell tumor cells are plump and lipid rich but grow in solid sheets, they do not ring fluid spaces. Dysgerminoma is made of large cells with clear cytoplasm split by fibrous septa carrying lymphocytes, no rosettes at all. Brenner tumor shows nests of urothelial-type cells in dense fibrous stroma, again nothing like a follicle.

Step 4: State the answer.

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: Set the rule for a granuloma.
Whenever macrophages cannot digest and clear an antigen, they wall it off by clumping together into epithelioid cells, sometimes fusing into giant cells. This walling off is the granuloma.

Step 2: Check the three infections that do wall things off.
Tuberculosis is the classic granuloma former, with central caseous necrosis. Tuberculoid leprosy also produces sharp, well formed granulomas because the immune response there is strong. Yersinia enterocolitica infection in the gut can produce granulomatous ileitis and lymphadenitis, closely mimicking Crohn disease.

Step 3: Check Mycoplasma pneumoniae.
This bacterium lacks a cell wall and causes a milder, diffuse interstitial lung inflammation. The reaction is lymphocytic and plasma cell rich, spread through the septa, not a focal macrophage wall. So no granulomas form here.

Step 4: Conclude.

Mycoplasma pneumoniae

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

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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: Compare apoptosis with its opposite, necrosis.
Necrosis is an uncontrolled, messy death where the cell swells, the membrane bursts, and enzymes and cell debris spill into the tissue, drawing in neutrophils and causing visible inflammation. Apoptosis is the tidy, controlled opposite of this.

Step 2: Trace what happens to an apoptotic cell.
The membrane stays sealed the whole time, so nothing leaks out to alarm the immune system. The cell shrinks, its DNA gets chopped by activated endonucleases into regular fragments, giving the classic condensed, clumped chromatin. Caspases drive the whole cascade, cleaving proteins that hold the cytoskeleton and nucleus together.

Step 3: See how the cell disappears.
Once broken into small, membrane wrapped apoptotic bodies, nearby macrophages and even ordinary neighboring cells quietly engulf these fragments. Because nothing spills out, no inflammatory chemicals are ever released.

Step 4: Match this against the four statements.
Caspase activation, endonuclease mediated chromatolysis, and macrophage clearance are all genuine features of apoptosis.

Surrounding inflammation is the one feature that belongs to necrosis instead, so it is the false statement here.

Surrounding inflammation

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

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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: Think about a cell that never divides again.
Neurons and heart muscle cells stay with the body for decades without being replaced. Every bit of oxidative damage they take gets partly cleared by lysosomes, but a residue is always left behind.

Step 2: Name that residue.
That leftover material is lipofuscin, a golden brown granular pigment made of oxidized lipid and protein fragments. Because it cannot be broken down further, it just keeps collecting inside the cell year after year, which is why pathologists treat its presence as a sign of an aged, long lived cell.

Step 3: Test each wrong option against biology.
If oxidative phosphorylation went up with age, cells would get more energetic as they aged, but the opposite is true, mitochondrial output falls with accumulated mutations. Glycogen stores do not rise with age either, metabolic reserves shrink. Nuclear material and mitochondrial number do not increase, aging cells generally show fewer, abnormal mitochondria and an irregular nuclear shape.

Step 4: Settle on the answer.

Lipofuscin accumulation

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

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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: Picture a normal cell versus a cancer cell using glucose.
A normal, well oxygenated cell mostly sends glucose through the mitochondria for oxidative phosphorylation, squeezing out the

maximum ATP per molecule. A cancer cell, even sitting in the same oxygen rich environment, instead pushes most of its glucose through glycolysis and turns it into lactate.

Step 2: Ask why a cell would choose the less efficient path.
Speed and raw material matter more than efficiency here. Glycolysis runs fast and its branch points hand off carbon skeletons to make nucleotides, amino acids and fatty acids, exactly what a cell needs when it is about to divide again and again.

Step 3: Address the tempting wrong answers.
Saying mitochondria or oxidative phosphorylation would be wrong here because these pathways are relatively suppressed in this metabolic switch, not the driver of it. Calling it anaerobic metabolism misses the point of the Warburg effect entirely, since oxygen is not scarce, the cell is choosing glycolysis on purpose despite having oxygen to spare.

Step 4: Conclude.

Glycolysis

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

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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: Start with the shape of the protein.
Clathrin molecules link up into three legged units called triskelions, and many of these fit together into a curved basket-like lattice on the cytoplasmic side of the cell membrane.

Step 2: See what that basket does to the membrane.
As the lattice grows and curves, it drags the membrane in with it, forming a pit. Whatever is bound to receptors sitting in that patch of membrane gets trapped inside the forming pit. Dynamin then snips the neck of the pit free, releasing a coated vesicle inside the cell.

Step 3: Give this process its name.
Because the cargo entering the cell is selected by receptor binding, this whole route is called receptor mediated endocytosis. Classic textbook examples are the uptake of LDL particles through the LDL receptor and the uptake of iron bound transferrin through the transferrin receptor.

Step 4: Explain why the other three do not fit.
Bulk uptake without a receptor generally uses other coats or no coat at all, so clathrin is not its main tool. Exocytosis, receptor based or not, works through SNARE mediated vesicle fusion with the outer

membrane, which is a different machinery from a clathrin coat pulling membrane inward.

Step 5: State the answer.

Receptor mediated endocytosis

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

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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:

Crescentic glomerulonephritis is graded by how widespread the crescent formation is across the biopsy sample, not by any single crescent's look.

1. **Size**: A crescent's size within one glomerulus is not tracked as a grading tool in routine reporting.
2. **Shape**: Crescents can be cellular, fibrocellular, or fibrous, but shape alone is a stage marker for one crescent, not a severity

score for the whole kidney.

3. **Number**: Pathologists count the glomeruli that carry crescents and report this as a percentage of all glomeruli sampled. Above 50 percent, the case is called RPGN, and a higher percentage predicts faster loss of kidney function.
4. **Pattern of distribution**: Whether crescents are focal or diffuse adds detail but the core severity marker stays the count.

So the number, that is the percentage of glomeruli involved, is what drives the severity grade and the decision to treat aggressively.

Let's summarize:

- Crescent count (as percent of glomeruli) is the standard severity parameter in crescentic GN.
- Over 50 percent involvement defines rapidly progressive glomerulonephritis.

The correct parameter is the number of glomeruli showing crescents.

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

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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:

FAP comes from a germline defect in the APC gene. Losing working APC protein frees up beta catenin, and this shift changes several downstream genes, including c-myc. Let's check each statement against that biology.

1. **C-myc gene expression decreased**: This is false. Loss of APC lets beta catenin build up and turn on c-myc, so c-myc expression rises rather than falls.
2. **Antibodies to normal mucin**: FAP patients have been found to carry antibodies against normal colonic mucin, so this statement holds true.
3. **Due to defect in the FAP gene**: True by definition, FAP is caused by a mutated APC/FAP gene inherited in an autosomal dominant pattern.
4. **Proliferation of colonic epithelium**: True, unchecked epithelial growth is exactly what produces the hundreds of adenomatous polyps seen in FAP.

Three of the four statements match accepted FAP biology. Only the claim of decreased c-myc expression goes against the known increase caused by unopposed beta catenin/TCF signalling.

Let's summarize:

- APC loss raises, not lowers, c-myc expression through beta catenin/TCF signalling.
- The other three statements about FAP are accepted as correct.

The statement that is NOT true is the claim of decreased c-myc gene expression.

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

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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:

Minimal change disease gets its name because the glomeruli look almost normal under a light microscope, yet the patient still has heavy proteinuria. The real damage only shows up on electron microscopy.

1. **Loss of foot processes**: True but incomplete on its own, since it does not explain why protein specifically leaks out.
2. **Antigen-antibody complexes**: Wrong for MCD. Immunofluorescence is negative here; immune complex deposits belong to diseases like membranous nephropathy.

3. **Loss of foot processes along with loss of charge across the membrane, causing proteinuria**: This is the complete and correct mechanism. Podocyte foot process effacement plus loss of the membrane's anionic (negative charge) barrier lets albumin cross into the urine.
4. **Destruction of the glomerulus, with minimal tissue remaining**: This describes severe glomerular scarring, not MCD, where the glomerulus stays structurally intact.

Put simply, MCD proteinuria comes from a double hit: the podocyte's foot processes flatten out, and the membrane loses the negative charge that normally repels albumin.

Let's summarize:

- Light microscopy is normal in MCD; the lesion is seen only on electron microscopy.
- Foot process loss plus charge barrier loss together explain the selective proteinuria.

The correct statement is option (C): loss of foot processes with loss of membrane charge, leading to proteinuria.

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

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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:

Cell immortalisation is about beating the natural limit on how many times a cell can divide, and that limit is set by the telomeres at the chromosome ends.

1. **DNA polymerase**: Copies the DNA strand during replication, but it cannot fully replicate the very end of a linear chromosome, which is exactly why telomeres shorten over successive divisions.
2. **mRNA polymerase**: Transcribes DNA into messenger RNA. It has no part in copying or lengthening telomeres.
3. **Telomerase**: Carries its own RNA template and extends the telomere repeats after each round of division. Cancer cells reactivate this enzyme, which is normally switched off in most adult tissues, so their telomeres stop shrinking and the cells can divide without limit.
4. **Topoisomerase**: Cuts and reseals DNA strands to relieve supercoiling ahead of the replication fork. It does not touch telomere length.

Because telomerase is the enzyme that keeps telomeres from wearing down, its reactivation is what gives cancer cells their immortal, endlessly dividing behaviour.

Let's summarize:

- Telomere shortening normally caps how many times a cell can divide.
- Telomerase reactivation in cancer cells removes this cap.

The correct enzyme is telomerase.

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

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

(A) Dysplasia

(B) Metaplasia

(C) Anaplasia

(D) Hyperplasia

Correct Answer: (A) Dysplasia

SOLUTION:

The question is asking which cellular change shows disordered but reversible orientation of cells, that is, a polarity problem.

1. **Dysplasia**: Cells lose their normal size, shape and orderly polarity, along with increased mitotic activity. This disorder is a pre-cancerous change, and importantly it can reverse if the trigger is removed in time.
2. **Metaplasia**: One mature cell type is swapped for another, better suited type, for example columnar to squamous. This is about cell type, not the orientation of individual cells, and it is reversible too, but it does not describe a polarity defect.

3. **Anaplasia**: Cells lose differentiation almost completely and take on a wild, disorganised appearance typical of malignancy. This change is not reversible.
4. **Hyperplasia**: An increase in the number of normally arranged cells in response to a stimulus. The cells keep their normal polarity, so this option does not fit either.

Only dysplasia specifically combines a disturbance of cell polarity with the possibility of reversal once the offending stimulus is gone.

Let's summarize:

- Dysplasia disturbs cell polarity, size and shape, and can reverse early on.
- Metaplasia, anaplasia and hyperplasia do not match a polarity based, reversible change the way dysplasia does.

The correct answer is dysplasia.

Quick Tip: Dysplasia disturbs cell polarity and orientation, and can reverse if the cause is removed early.

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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:

Diapedesis is one specific step in how a neutrophil gets from the blood into inflamed tissue, so each option needs to be checked against where it sits in that journey.

1. **Attachment of neutrophils to blood vessel**: This describes adhesion, an earlier step where integrins on the neutrophil bind ICAM-1 on the endothelium. It happens before diapedesis, not during it.
2. **Escape of neutrophils from capillaries through the capillary endothelium**: This is exactly diapedesis, the neutrophil squeezing between endothelial cells and moving out of the vessel into the tissue.
3. **Stimulation of cytokine secretion by inflammatory cells**: This is chemical signalling that helps recruit and activate cells, not the physical crossing of the vessel wall.
4. **Response of mediator cells to cytokine secretion**: Again a signalling event, describing how cells react to chemical messengers, not a physical movement across the endothelium.

Only the second option describes the actual physical event of a neutrophil leaving the capillary lumen and entering the tissue, which is the definition of diapedesis.

Let's summarize:

- Diapedesis follows adhesion in the leukocyte extravasation sequence.

- It is the physical transmigration of the neutrophil across the endothelium, not a signalling event.

The correct answer is escape of neutrophils from the capillary across the endothelium.

Quick Tip: Diapedesis is the physical step where neutrophils cross the capillary endothelium into tissue, after adhesion.

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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:

The scenario describes one adult cell type in the lung being swapped for a different, tougher adult cell type in response to years of smoke exposure. That swap has a specific name in pathology.

1. **Anaplasia**: Describes cells that have lost their differentiation, typical of malignant tumours. It is not about one normal cell type being replaced by another.
2. **Metaplasia**: The reversible change of one differentiated tissue type into another differentiated type better able to handle the

stress. Smoke driven replacement of respiratory epithelium by stratified squamous epithelium is the classic textbook example.
3. **Dysplasia**: Disordered, atypical growth within a single cell type, which can follow metaplasia if the irritant is not removed, but is a separate, more advanced change than the cell type swap itself.
4. **Hyperplasia**: More cells of the same original type, not a change to a different cell type.

Since the question describes one epithelial type being entirely replaced by another, tougher type, the fitting term is metaplasia.

Let's summarize:

- Metaplasia swaps one differentiated cell type for another, better suited one.
- Chronic smoking classically produces squamous metaplasia of the respiratory epithelium.

The correct answer is metaplasia.

Quick Tip: Chronic smoking classically causes squamous metaplasia, swapping respiratory epithelium for tougher squamous cells.

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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:

This question is testing the classic light microscopy finding of minimal change disease, the commonest cause of steroid-responsive nephrotic syndrome in children under 6 years.

1. **Nothing**: correct. Minimal change disease gets its name because the glomeruli look normal under a light microscope. No thickening, no scarring, no cell proliferation is seen.
2. **Loss of foot processes**: this change is real, but it is only visible on electron microscopy, where the podocyte foot processes appear flattened and fused. A light microscope cannot resolve structures this small.
3. **Tubule atrophy**: this is a feature of chronic, longstanding renal damage, seen in conditions like chronic glomerulonephritis or long-standing obstruction, not in a steroid-responsive nephrotic child.
4. **Crescents**: crescents form in Bowman's space in rapidly progressive glomerulonephritis, a severe and steroid-resistant disease pattern, which does not match this child's good response to steroids.

Since the child is young, steroid-responsive, and the light microscope shows a normal-looking glomerulus, the diagnosis is minimal change disease, and the light microscopy finding is nothing.

Let's summarize:

- Minimal change disease has a normal light microscopy picture; the abnormality is only seen on electron microscopy.

- Steroid responsiveness in a young child with nephrotic syndrome strongly points to minimal change disease.

So the correct answer is that nothing abnormal is seen on light microscopy.

Quick Tip: Steroid-responsive nephrotic syndrome in a young child points to minimal change disease, which looks normal on light microscopy.

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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:

This question tests which vascular skin lesion is permanent versus which ones fade with time.

1. **Salmon patch**: a light pink mark seen at birth, most often on the eyelids or the back of the neck. The ones on the face typically fade within the first one to two years of life.
2. **Strawberry angioma**: a bright red raised lesion that grows for a few months after birth, then slowly shrinks over the next several years, leaving little or no trace by school age in most children.
3. **Portwine stain**: a flat, deep red to purple mark that is a true malformation of small capillaries, not a tumor. It is permanent,

growing with the child and often becoming darker and thicker with age.

4. **Lymphangiomatosis circumscripta**: clusters of small clear or blood tinged vesicles from abnormal lymph channels, which persist but are not the classic teaching example here.

Since a portwine stain is a fixed capillary malformation rather than a growth that involutes, it is the one lesion here that never regresses on its own.

Let's summarize:

- Salmon patch and strawberry angioma are both known for fading or shrinking with time.
- Portwine stain is a permanent capillary malformation that tends to darken, not disappear.

So the lesion that does not regress is the portwine stain.

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:

This question is about why a child with a large VSD and heart failure may seem to get better over time without any treatment.

1. **Reduction in size of the VSD**: spontaneous partial closure of a perimembranous VSD does occur in some children and can ease symptoms, but it is not the classic mechanism tested here for a child whose defect stays open.
2. **Pulmonary vascular changes**: long standing high flow and high pressure in the lung vessels thickens their walls. This raises pulmonary vascular resistance, which opposes and reduces the left to right shunt through the VSD, so the lungs get less extra blood and the failure symptoms lessen.
3. **Infective endocarditis**: an infection of the heart lining or valves that can complicate a VSD. It makes the child sicker, with fever and new murmurs, it never explains an improvement.
4. **Aortic regurgitation**: a perimembranous VSD can pull an aortic cusp down into the defect over time, causing valve leakage. This adds more work for the heart and worsens failure, it does not cause improvement.

The correct explanation is that the pulmonary arteries thicken and resist flow, quietly cutting down the shunt and easing the symptoms, a sign that is worrying rather than reassuring because it can progress toward irreversible pulmonary hypertension.

Let's summarize:

- Rising pulmonary vascular resistance reduces the left to right shunt through a VSD.
- This can look like clinical improvement, but it marks the start of pulmonary vascular disease.

So the cause of the apparent improvement is pulmonary vascular changes.

Quick Tip: Rising pulmonary vascular resistance from chronic high flow reduces the left to right shunt through the VSD.

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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:

This question asks which cyanotic heart lesion best fits cyanosis appearing right at birth, along with pulmonary oligemia and a normal-sized heart on x-ray.

1. **Tetralogy of Fallot**: gives the textbook normal-sized, boot-shaped heart with oligemia, but its cyanosis more often builds up gradually over the first weeks to months as infundibular narrowing worsens, rather than being obvious in the newborn period.
2. **Pulmonary atresia**: usually shows an enlarged right atrium on x-ray because blood backs up through the tricuspid valve, so the heart tends to look bigger than normal, not normal sized.

3. **Transposition of great vessels**: is the single most common cause of cyanosis appearing within the first day or two of life, since the two circulations run in parallel and only mix through a shunt. Before cardiomegaly develops over the following days, the heart can still look normal, and if there is any associated narrowing of the pulmonary outflow, the lungs show oligemia instead of the usual plethora.
4. **Ebstein's anomaly**: is known for a massively enlarged, wall to wall heart shadow on x-ray, which rules it out here since the heart is described as normal sized.

Putting the timing, the oligemia, and the still-normal heart size together, transposition of the great vessels fits best among these four choices.

Let's summarize:

- Transposition of the great vessels is the top cause of cyanosis presenting right in the neonatal period.
- Ebstein's anomaly and pulmonary atresia usually show an enlarged heart, not a normal one, which rules them out here.

So the likely diagnosis is 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.

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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:

This question is testing which test confirms a suspected inherited haemoglobin disorder in a child with a haemolytic anaemia picture.

1. **Hb electrophoresis**: separates the different types of haemoglobin present in the blood, and it is the direct way to detect an abnormal or reduced haemoglobin type, exactly what target cells and a family history of the same illness point towards.
2. **Bone marrow biopsy**: is used when the marrow itself may be failing or infiltrated, such as in aplastic anaemia or leukaemia, not for a child who already has a raised reticulocyte count showing the marrow is working well.
3. **Osmotic fragility test**: checks how easily red cells burst in a dilute salt solution, and it is the test of choice for hereditary spherocytosis, where the smear shows spherocytes, not the target cells described here.
4. **Chromosomal analysis**: looks for extra, missing, or rearranged chromosomes, useful in syndromes like Down syndrome, but haemoglobin disorders are gene-level problems that this test cannot pick up.

Given the target cells, the high reticulocyte count, and an affected sibling, the picture fits an inherited haemoglobinopathy, and

haemoglobin electrophoresis is the test that will show the abnormal haemoglobin pattern.

Let's summarize:

- Target cells point toward a haemoglobinopathy like thalassemia, not spherocytosis.
- Hb electrophoresis, not osmotic fragility or chromosome studies, confirms this kind of disorder.

So the investigation to perform is haemoglobin electrophoresis.

Quick Tip: Target cells plus a family history point to a haemoglobinopathy; Hb electrophoresis confirms it.

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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:

This question is comparing Hurler and Hunter syndrome, two mucopolysaccharide storage disorders that look alike on the surface but differ in how badly they affect the heart.

1. **Hurler syndrome**: causes severe valve and heart muscle disease early on, giving a clearly abnormal ECG, and untreated children usually die of heart complications by about 6 to 10 years of age. A normal ECG rules this one out.
2. **Hunter syndrome**: shares the coarse face, joint stiffness, and delayed development seen in Hurler syndrome, but its heart involvement is milder and slower to develop, so an ECG can still look normal in infancy. It is also X-linked, mostly affecting boys, and generally runs a gentler course than Hurler syndrome.
3. **Glycogen storage disorder**: for example Pompe disease, causes profound muscle weakness together with a big, failing heart and a grossly abnormal ECG from early infancy, which does not fit a normal ECG.
4. **Phenylketonuria**: leads to developmental delay and a distinct body odour from a build up of phenylalanine, but it does not cause coarse facial features, joint stiffness, or heart changes, so the clinical picture here does not fit.

Since the heart tracing is normal despite the coarse features and weakness, the milder mucopolysaccharidosis, Hunter syndrome, fits better than Hurler syndrome.

Let's summarize:

- Hurler syndrome causes severe early heart disease with an abnormal ECG; Hunter syndrome is milder on the heart.
- A normal ECG in an infant with coarse features favours Hunter syndrome over Hurler syndrome.

So the likely diagnosis is Hunter syndrome.

Quick Tip: Hurler syndrome damages the heart early with an abnormal ECG; Hunter syndrome is milder, so a normal ECG favours Hunter.

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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:

This question links a growth and mental delay together with specific bone x-ray changes to reach a diagnosis.

1. **Hypopituitarism**: growth hormone lack slows physical growth and bone age, but the brain usually still develops close to normally, so it does not explain the mental retardation or the fragmented epiphyses and wormian bones seen here.
2. **Hypothyroidism**: thyroid hormone is needed for both bone growth and brain maturation, so a lack of it in infancy stunts height and slows mental development together. On x-ray, the epiphyses ossify unevenly, giving a stippled, fragmented look, and delayed skull ossification produces extra wormian bones within the sutures.

3. **Hypogonadism**: mainly affects puberty and the growth spurt, it does not cause mental retardation or these particular bone changes in a young boy.
4. **Scurvy**: is caused by a lack of vitamin C and shows up as bone pain, bleeding gums, and a white line with a ground glass look at the metaphysis on x-ray, along with bleeding under the periosteum, a very different picture from fragmented epiphyses and wormian bones, and it does not cause mental delay.

Only hypothyroidism ties together slowed physical growth, slowed mental development, fragmented epiphyses, and wormian bones in one diagnosis.

Let's summarize:

- Thyroid hormone is essential for both bone growth and brain development, so its lack delays both together.
- Fragmented, stippled epiphyses and wormian bones on x-ray are classic hypothyroidism findings.

So the diagnosis is hypothyroidism.

Quick Tip: Combined growth and mental delay with fragmented epiphyses and wormian bones on x-ray points to hypothyroidism.

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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: Think about what the carotid artery is sensing.
Blood in the carotid artery is the first blood to reach the brain, so it is the ideal spot to test how the brain reacts to a change in blood concentration before that blood mixes further.

Step 2: Identify the sensing cells.
Specialised nerve cells called osmoreceptors pick up small shifts in the salt concentration of this blood. These cells lie right around the supraoptic nucleus of the hypothalamus.

Step 3: Follow the reflex.
Once the osmoreceptors near the supraoptic nucleus fire, the supraoptic neurons themselves (which manufacture ADH) change their output, and ADH travels down their axons to the posterior pituitary for release into the blood. This whole reflex is the basis of the body's water balance control, and it is switched on by a change of tonicity delivered right through the carotid artery.

Step 4: Cross off the wrong nuclei.
The medial hypothalamic nucleus governs hunger and satiety. The preoptic nucleus governs body temperature. The paraventricular nucleus also secretes ADH and oxytocin, but the osmoreceptor zone

that first answers a carotid tonicity change belongs to the supraoptic nucleus.

Step 5: State the answer.

Supraoptic nucleus of the hypothalamus

Quick Tip: Osmoreceptors that sense carotid blood tonicity sit next to the ADH-making supraoptic nucleus.

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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: Separate real differences from a look alike difference.
Three of the four choices describe things that are truly different in a newborn compared with an adult. The fourth choice sounds like a difference but is not, once you correct for the newborn's small size.

Step 2: Oxygen demand.
Because a newborn's tissues are metabolically busier per kilogram of body weight than an adult's, its oxygen use rate per kilogram is nearly

double that of an adult. This is a real, true difference.

Step 3: Haemoglobin type.
A newborn still carries mostly fetal haemoglobin, which grabs oxygen more tightly than the adult type. So compared with fetal Hb, adult Hb genuinely does have a lower pull on oxygen. This too is a real difference.

Step 4: Lung size.
A newborn's lungs, alveoli, and airways are all smaller in absolute size than an adult's, simply because the whole newborn body is smaller. Total lung volume is genuinely smaller.

Step 5: FRC, the odd one out.
FRC looks reduced in the newborn only if you compare raw numbers without adjusting for size. Once FRC is expressed per kilogram of body weight, it comes out close to the adult figure. What makes newborns vulnerable is not a truly reduced FRC, but that their oxygen use per kilogram is so high relative to that near normal FRC, so their oxygen reserve runs out fast.

Step 6: Conclude.
The option that is not a genuine difference is the FRC one.

Decreased FRC in the newborn

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: Start with the trigger.
Breathing fast and deep washes CO_2 out of the blood quicker than it is produced.

Step 2: Work out the acid base shift.
Since CO_2 forms carbonic acid in blood, losing CO_2 pushes blood pH up, giving respiratory alkalosis.

Step 3: Follow the calcium.
Blood calcium exists in two pools, one stuck to albumin and one free (ionised). A higher pH changes the charge on albumin so that it grabs more calcium than usual. Total calcium stays the same, but the ionised share drops.

Step 4: Explain the spasm.
Nerves and muscle fibres depend on ionised calcium to keep their membranes calm. With less of it around, the membrane threshold for firing drops, so nerves fire spontaneously and muscles go into spasm, a state called tetany.

Step 5: Eliminate the distractors.
The falling CO_2 is the starting trigger, not the direct cause of spasm. Potassium and sodium are not the ions responsible for this classic hyperventilation tetany.

Step 6: Final answer.

Decreased ionised calcium

Quick Tip: Hyperventilation causes respiratory alkalosis, which lowers ionised (free) calcium.

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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: Set up a two column comparison.
It helps to compare presynaptic inhibition against postsynaptic (direct) inhibition side by side, since most of the statements are really testing that contrast.

Step 2: Anatomy.
Postsynaptic inhibition uses an axo-somatic or axo-dendritic synapse and hyperpolarises the cell body, producing a clear IPSP. Presynaptic inhibition instead uses an axo-axonal synapse, sitting on another axon terminal, so option (a) fits presynaptic inhibition.

Step 3: Electrical signature.
Because presynaptic inhibition works by cutting down transmitter release rather than hyperpolarising the postsynaptic membrane, it does not produce or prolong a postsynaptic IPSP. So option (b) does not belong to presynaptic inhibition.

Step 4: Drug sensitivity.
General anaesthetics boost GABA-A receptor function at the axo-axonal terminal, so they prolong presynaptic inhibition, making option (c) correct. Since GABA-A receptors are the target here, presynaptic inhibition is clearly drug sensitive, so option (d) is wrong.

Step 5: Site of action.
The motor endplate potential belongs to the neuromuscular junction, a site with no axo-axonal GABA synapse, so option (e) does not apply to presynaptic inhibition.

Step 6: Pharmacological block.
Glycine receptor blockers such as strychnine remove postsynaptic inhibition, while GABA-A receptor blockers such as picrotoxin remove presynaptic inhibition. So option (f), strychnine, is wrong for presynaptic inhibition, and option (g), picrotoxin, is right.

Step 7: Collect the correct set.
Putting the correct items together gives (a), (c), and (g).

$$\boxed{a,\ c,\ g}$$

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 WHO guidance on timing.
The recommended gap before giving a semen sample is short, in the range of about 2 to 7 days.

Step 2: See what six weeks does.
Six weeks is well past that window. Holding sperm in the epididymis for so long lets more of them age and die or lose motility, so the sample actually gets worse, not better. That makes the six week claim false, which is the answer we want since the question asks for the one FALSE statement.

Step 3: Confirm motility as a quality marker.
Motility, how well sperm swim forward, is one of the main things checked in a semen analysis because it tracks directly with the chance of natural fertilisation. So this claim is true.

Step 4: Confirm the collection site rule.
Because sperm lose motility quickly once outside the body, the sample should ideally be produced right at the lab, or delivered within a short set time if produced elsewhere. So this claim is true.

Step 5: Confirm the fructose fact.
Fructose is made by the seminal vesicles and is normally present in semen. No fructose points to a blocked ejaculatory duct or absent seminal vesicles. This claim is true too.

Step 6: Conclude.
Only the six week abstinence claim is false.

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: Sort and count.
The seven numbers **2, 5, 7, 10, 10, 15, 20** are already sorted from smallest to largest, and there are **7** of them, an odd count.

Step 2: Trim from both ends.
An easy way to find the median with an odd count is to strike off the smallest and largest values together, one pair at a time, until one value is left.
Remove **2** and **20**: left with **5, 7, 10, 10, 15**.
Remove **5** and **15**: left with **7, 10, 10**.
Remove **7** and the second **10**: left with just **10**.

Step 3: Read the answer.
The one value remaining after trimming is the median.

$$\boxed{10}$$

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 the r scale.
r runs from −1 to +1. The sign shows direction, the size shows strength, and only a truly exact linear relationship gives $r = \pm 1$.

Step 2: Think about the biology first.
As a population's economic status improves, families get better food, cleaner water, and more access to doctors, so infant deaths go down. That is an inverse relationship, so *r* should come out negative.

Step 3: Drop the perfect values.
Since many things besides income affect infant mortality, such as vaccination coverage or disease outbreaks, a perfect straight line fit with $r = +1$ or $r = -1$ is not realistic for this kind of health data.

Step 4: Drop the wrong sign option.
$r = +0.22$ has the wrong sign for this relationship and is also a weak value, so it does not fit the strong known link between poverty and child deaths.

Step 5: Settle on the fitting value.

$r = -0.8$ is negative and strong, matching a real, well documented inverse tie between economic status and infant mortality, without claiming an impossible perfect correlation.

$$\boxed{r = -0.8}$$

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:

This question tests knowledge of epidemiological measures used to compare mortality between two different populations.

1. **Standardised Mortality Rate**: This rate is adjusted for age (or other confounding factors) using a standard population, so it removes the bias caused by different population structures. It is the correct tool for a fair comparison.

2. **Disease Specific Death Rate**: This only reflects deaths from a single disease and gives no correction for age or other structural differences between the two populations.
3. **Proportional Mortality Rate**: This shows the share of total deaths caused by a given condition, but it is influenced by the overall death pattern and is not a true standardised measure.
4. **Age Specific Death Rate**: This is useful for one age band at a time, but comparing many age bands separately is not as practical as a single standardised summary figure.

Because crude rates are distorted by differences in age structure between populations, epidemiologists use standardisation to make a fair comparison. The standardised mortality rate is built exactly for this purpose.

Let's summarize:

- Crude rates cannot be compared directly if population structures differ.
- Standardisation adjusts for these differences using a reference population.

So the best measure for comparing two populations is the 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: What is being measured?
Burden of disease is not just about how many people die. It is about how much healthy life is lost overall, from both death and disability.

Step 2: Recall the tool built for this.
The DALY, short for Disability Adjusted Life Year, was made for this exact job. It adds up years lost to early death (YLL) and years lived with disability (YLD) into a single number for each disease.

Step 3: Check the other choices.
Case fatality rate looks only at people who already have the disease and tells us how deadly it is to them, not the burden on the whole population. Dependence rate is a population structure ratio unrelated to disease. Morbidity data alone just counts sick people without weighing how severe or long lasting the illness is.

Step 4: Conclude.
Since DALY is the only measure that folds both death and disability into a single comparable score, it is the accepted 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:

This question checks how much ethinyl estradiol is present in the newest generation of low dose combined oral contraceptive pills.

1. **20 micrograms**: This is the dose used in the newest ultra low dose pills, developed to cut down side effects like clotting risk while still blocking ovulation reliably.
2. **25 micrograms**: Not the standard dose used in the newest low dose formulations.
3. **30 micrograms**: This dose belongs to an earlier generation of low dose pills, used before the 20 microgram pills came into practice.
4. **35 micrograms**: This was also an earlier low dose option, higher than the newest formulation.

Oral contraceptive pills have moved from high dose formulations of 50 micrograms or more down to low dose and then ultra low dose

pills. The newest ones use just 20 micrograms of ethinyl estradiol.

Let's summarize:

- Pill doses of ethinyl estradiol have fallen over the decades to reduce side effects.
- The newest low dose pills use 20 micrograms, the lowest of the listed options.

So the correct dose for the new low dose pill is 20 micrograms.

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: Know what standard error means.
Standard error tells us how precisely the sample mean estimates the true population mean. A bigger sample gives a smaller standard error.

Step 2: Write the formula.

$$SE = \frac{SD}{\sqrt{n}}$$

Step 3: Plug in the numbers.
We are told $SD = 1$ and $n = 100$ women, so $\sqrt{n} = \sqrt{100} = 10$.

$$SE = \frac{1}{10} = 0.1$$

Step 4: Conclude.
So the standard error works out to 0.1, much smaller than the standard deviation itself, since a sample of 100 gives a fairly precise estimate of the mean.

$$\boxed{0.1}$$

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:

This question is about picking the right statistical test to compare two proportions collected from two separate samples.

1. **Chi square test**: Used to compare proportions (categorical data) between independent groups when sample sizes are adequate. This fits comparing 40 percent versus 60 percent from two samples.
2. **Fischer test**: Used in place of chi square only when the sample is very small and expected cell counts fall below 5. No such small sample is described here.
3. **Paired t test**: Used for comparing two related, quantitative measurements on the same subjects, such as pre and post readings, not for independent proportions.
4. **ANOVA test**: Used to compare means of a quantitative variable across three or more groups, which does not match a two group proportion comparison.

Because the data here are proportions from two independent samples with adequate size, the chi square test is the correct method to check if the difference is statistically significant.

Let's summarize:

- Chi square compares proportions between independent groups.
- Fischer test is reserved for very small samples, and paired t test and ANOVA are for quantitative, not categorical, comparisons.

So the best test to compare these two results is the 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: Recall the standard normal curve landmarks.
For data that follow a normal distribution, fixed shares of the values sit within set distances from the mean, measured in standard deviations (SD).

Step 2: List the key ranges.
About 68% of values lie within ±1 SD, about 95% lie within ±1.96 SD (often rounded to 2 SD), and about 99.7% lie within ±3 SD.

Step 3: Match 1.95 SD to a range.
The figure 1.95 SD in the question is essentially the familiar 1.96 SD cut off, so it marks the boundary that holds about 95% of the sample.

Step 4: Rule out the rest.
68% belongs to 1 SD, not 1.95 SD. 99% needs a wider spread, close to 2.58 SD. 65% is not a standard landmark at all.

Step 5: Conclude.
So the share of the sample within 1.95 SD of the mean is 95%.

$$\boxed{95\%}$$

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: Look at the form of the equation.
The relation given is $y = a + bx$, where y stands for height and x for age.

Step 2: Identify what kind of equation this is.
Here x appears only to the first power, with a constant a added to a constant b times x. This is the exact form of a simple linear equation, the same one used in linear regression, where a is the intercept and b is the slope.

Step 3: Compare with the other shapes listed.
A parabola would need an x^2 term, a hyperbola would need x and y multiplied together or x in a denominator, and a sigmoid curve would need an exponential term. None of these features appear in $y = a + bx$.

Step 4: Conclude.
A plot of y against x for this equation gives a straight line with slope b.

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:

This question checks the basic features of a case control study, one of the two major observational designs used in epidemiology along with the cohort study.

1. **It gives attributable risk**: A case control study samples people by disease status, not by exposure status, so the true incidence of disease among exposed and unexposed people stays unknown. Attributable risk needs that incidence data, so a case control study cannot give it directly. This is the false statement.
2. **It is less expensive**: True. There is no long follow up period, and the sample size stays small, so the running cost stays low.
3. **It involves fewer subjects**: True. Cases of even a rare disease can be pulled from hospital records, and each case needs only one or a few matched controls, so the total subject count stays small.
4. **It provides quick results**: True. Since the disease outcome has already happened by the time the study starts, there is no need to wait years for it to occur, so results are ready sooner than in a cohort study.

The correct answer is that a case control study gives attributable risk, since this is the one claim that does not hold up. A case control study can only estimate relative risk through the odds ratio, never attributable risk.

Let's summarize:

- Case control studies work backward from disease to exposure and rely on an odds ratio, not incidence.
- They are cheap, quick, and need fewer subjects compared with a cohort study.

So the statement that does not hold true for a case control study is that it gives 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: Write down what IMR measures.
IMR tells us how many infants (under one year old) die for every 1000 babies born alive in a year, in one place.

Step 2: Sort the numbers given in the question.
The question gives three numbers: total population 36,000, live births 1200, and infant deaths 60. Only the last two go into the IMR formula. The population figure is a distractor.

Step 3: Set up the ratio.

$$IMR = \frac{60}{1200} \times 1000$$

Step 4: Simplify before multiplying.
60 divided by 1200 is 0.05. Multiply this by 1000 to scale it to the standard "per 1000 live births" base.

$$IMR = 0.05 \times 1000 = 50$$

Step 5: State the answer.

$$\boxed{\text{50 per 1000 live births}}$$

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: Think about why a rate needs a fixed population.
The crude death rate compares deaths in a year to the size of the population that was at risk that year. Since population size shifts every day, one representative figure has to stand in for the whole year.

Step 2: Locate the midpoint of the year.
A calendar year runs from 1^{st} January to 31^{st} December. The date that splits this span into two roughly equal halves is 1^{st} July.

Step 3: See why the midpoint is the right choice.
Using the start of the year (January) would miss growth that happens later. Using the end of the year (December) would overstate the population, since it already reflects a full year of growth. The midpoint balances these two errors and gives the closest estimate to a true year long average.

Step 4: Rule out the other date given.
1^{st} May is close but still falls short of the true halfway point of the year, so it is not the standard reference date used in vital statistics.

Step 5: State the convention.
By convention, 1^{st} July of the year is used as the mid year population for calculating crude rates such as the crude death rate.

$\boxed{1^{st} \text{ July}}$

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:

KFD, or monkey fever, is a tick borne viral fever seen in forested parts of Karnataka. It passes to people through Haemaphysalis tick bites, with monkeys helping to raise the amount of virus circulating in the forest.

1. **Vaccination**: A killed KFD vaccine is offered to people living near or entering the forest, cutting their chance of severe infection. This is a real preventive step.
2. **Deforestation**: Cutting down forest disturbs the natural tick and monkey habitat and pushes ticks toward forest edges where people, cattle, and monkeys mix more closely. This has been tied to more outbreaks, not fewer, so it works against prevention.
3. **Prevention of roaming cattle**: Cattle that graze inside the forest and then return to the village can carry ticks with them. Stopping this movement, or treating cattle against ticks, blocks a real route of spread.
4. **Personal protection**: Repellents, covered clothing, and checking the skin for ticks after a forest visit all cut the chance of being bitten, so this genuinely helps prevention.

Three of the four options, vaccination, cattle control, and personal protection, are established ways to prevent KFD. Deforestation instead disturbs the forest and raises human contact with infected ticks.

Let's summarize:

- KFD spreads through tick bites, with monkeys amplifying the virus in the forest.

- Vaccination, cattle control, and personal protection reduce risk; clearing forest increases it.

So the measure that is NOT useful in preventing KFD is deforestation.

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:

This question is about picking the right statistical test for a before-and-after drug study on two groups of women, obese and non-obese.

1. **Unpaired t test**: This test compares the means of two separate, independent groups of subjects, such as the obese group compared against the non-obese group as two different samples. It does not fit a before-after comparison done on the same women, so it is not the test asked for here.

2. **Paired t test**: Each woman in a limb gives two readings, cholesterol before the drug and cholesterol after the drug. Since both readings come from the same person, they are linked, and the paired t test is built exactly for comparing such related pairs of measurements. This fits the study design.
3. **Chi square test**: This test is meant for categorical data, such as counts sorted into groups, not for continuous numeric readings like cholesterol level in mg/dL. It does not apply here.
4. **Fischer test**: Fischer's exact test is used for small sample categorical data arranged in a 2x2 table, again not suited to continuous before-after cholesterol values.

Since the cholesterol level in each woman is measured twice, once before and once after the drug, the two values are paired within the same subject, and the paired t test is the correct choice to correlate these results.

Let's summarize:

- Same subject measured twice (before-after) needs a paired t test.
- Two separate independent groups measured once each would need an unpaired t test instead.

So the test useful here to correlate the results obtained is the paired t test.

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: Name what is given.
Women with many partners get carcinoma cervix 5 times as often as women with one partner. This ratio of incidences is the relative risk, so $RR = 5$.

Step 2: Understand what attributable risk means.
Out of all the disease seen in the exposed group, some part would have happened anyway due to other causes, and some part is truly caused by the exposure. Attributable risk percent picks out that second part, as a percentage of the total risk in the exposed group.

Step 3: Use the shortcut formula built from RR.

$$AR\% = \frac{RR - 1}{RR} \times 100$$

This works because if incidence in the unexposed is I_0, incidence in the exposed is $RR \times I_0$, and the extra risk due to exposure is $(RR - 1)I_0$. Dividing this extra part by the total exposed incidence $RR \times I_0$ gives the same formula.

Step 4: Plug in $RR = 5$.

$$AR\% = \frac{5-1}{5} \times 100 = \frac{4}{5} \times 100$$

Step 5: Finish the arithmetic.

$$AR\% = 80\%$$

$$\boxed{80\%}$$

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:

This question asks which single figure best sums up how healthy a country's population is overall.

1. **Couple protection rate**: This shows only the share of eligible couples using contraception. It tells us about family planning uptake, not the general health of the population.
2. **IMR**: Infants are highly sensitive to poor maternal care, malnutrition, unsafe water, and gaps in health services. Because of this, changes in infant mortality closely track changes in the overall standard of health and living in a country, making it the most sensitive single indicator available.
3. **MMR**: Maternal mortality rate looks only at deaths linked to pregnancy and childbirth. It matters for judging maternal care, but it is a narrow slice of the population's health, not the whole picture.
4. **Crude birth rate**: This just counts births per 1000 people and reflects fertility patterns, which can stay high or low regardless of how good or bad the health system actually is.

Of these four, IMR is the one that most closely mirrors nutrition, sanitation, maternal care, and access to health services all together, which is why it is used as the best single determinant of a country's health status.

Let's summarize:

- Couple protection rate and crude birth rate reflect fertility and family planning, not overall health.
- MMR is narrow, limited to pregnancy related deaths, while IMR reflects the wider health picture.

So the best determinant of the health status of a country is the infant mortality rate.

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 Concept:
HDI is built on exactly three pillars: health, education, and income. Anything that does not fit under one of these three pillars cannot be part of HDI.

Step 2: Sort the four choices under the three pillars.
Life expectancy fits the health pillar. Educational level fits the education pillar. Per capita income fits the income pillar. All three have a clear home inside HDI.

Step 3: Test the leftover option.
IMR does not fit any of the three pillars. It is instead one of the three

legs of an older measure, the PQLI, alongside life expectancy at age one and literacy rate.

Step 4: Final Answer:
Because IMR has no place in the HDI formula, the correct answer is Infant Mortality Rate.

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 Concept:
PQLI is a three-legged stool. Each leg is a separate indicator, and all three are averaged to give one score for a country or region.

Step 2: Name the three legs.
Leg one is IMR, standing for how many infants survive. Leg two is life expectancy measured from age one, not from birth, so that infant deaths do not skew it twice. Leg three is literacy rate, standing in for

education.

Step 3: Check the odd option out.
MMR looks at maternal deaths during childbirth, a real and important number, but it was never chosen as one of the three PQLI legs.

Step 4: Final Answer:
MMR sits outside the PQLI formula, so it is the correct answer.

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 Concept:
Picture the normal curve as a symmetric hill. The median is the point straight under the peak, splitting the hill into two equal halves by area.

Step 2: Place the median on the hill.
We are told the median is 120 mm Hg, so 120 sits right under the peak of this particular hill.

Step 3: Read off the two halves.
Since the hill is symmetric, the slice to the left of 120 and the slice to the right of 120 carry equal area, each worth half of the total 100 subjects.

Step 4: Final Answer:
The right-hand slice, readings above 120 mm Hg, holds 50 subjects out of 100, so the answer is 50 percent.

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 Concept:
Post-exposure prophylaxis buys time. It only helps when the disease

takes a while to actually appear after exposure, or when ready-made antibodies can be handed to the body straight away.

Step 2: Walk through the three diseases where it works.
Rabies has a long, slow incubation, so vaccine plus immunoglobulin after a bite reliably stops the disease. Hepatitis B also has a long incubation, so HBIG plus vaccine after exposure works well. Measles has an incubation of about 10 to 14 days, long enough for a vaccine given within 72 hours, or immunoglobulin within 6 days, to still help.

Step 3: Now test pertussis.
Pertussis comes on fast, within about a week to ten days of exposure. There simply is not enough time for a vaccine given after exposure to build protection, so contacts are instead covered with antibiotics such as azithromycin, not with a vaccine-based PEP.

Step 4: Final Answer:
Pertussis is the condition where post-exposure prophylaxis in the vaccine or immunoglobulin sense does not work.

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 Concept:
Anemia is not judged by one single Hb number for everyone. WHO sets a separate cut-off for each age and sex group, because what counts as a low Hb depends on the person's stage of life.

Step 2: Place infants under 6 months on the scale.
For children between 6 months and 6 years, the standard WHO cut-off used across most Indian PSM texts is Hb below 110 g/L. Infants younger than 6 months sit just before this group, and their normal Hb range runs a touch higher once the newborn Hb dip has started to recover.

Step 3: Match this to the given options.
Among the four values offered, the reference used for this paper's answer key places the cut-off for the under-6-months group at Hb below 115 g/L, one notch above the 110 g/L used for the older infant and toddler group.

Step 4: Final Answer:
Going by the key used for this question, an Hb below 115 g/L is what defines anemia in an infant under 6 months of age.

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.

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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 Concept:
ICDS runs a supplementary feeding programme through anganwadi centres, and each type of beneficiary, whether a small child, a severely malnourished child, or a pregnant or lactating woman, is given a fixed daily calorie and protein target.

Step 2: Compare beneficiary groups.
A normal preschool child's target sits around 500 calories with 12 to 15 gm protein. A pregnant or lactating woman needs the same order of calories but a clearly higher protein share, since building a baby (or milk) needs extra protein, not just extra energy.

Step 3: Match the numbers to the options.
Out of the choices given, only 500 calories with 25 gm protein reflects that step-up in protein for a pregnant woman, while keeping the

calorie figure in the same range as a child's target.

Step 4: Final Answer:
The correct figure for a pregnant woman under this scheme is 500 calories and 25 gm protein.

Quick Tip: Under ICDS, a pregnant woman gets more protein per day than a young child gets, because pregnancy raises protein need sharply.

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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 Concept:
Whenever one number in a data set is wildly bigger than the rest, as 5000 is here compared to values like 20, 50, or 60, that one number can badly distort a plain average.

Step 2: See what the outlier does to the mean.
The arithmetic mean adds every value and divides by the count, so the huge 5000 gets baked directly into that sum and pulls the mean well

above where most of the data actually clusters. That makes the mean a poor stand-in for typical incidence here.

Step 3: Rule out mode and geometric mean too.
The mode fails outright since no value in the list repeats. The geometric mean is built for growth rates and ratios, not raw incidence counts, and can still be dragged by the same extreme value, so it is not the natural fit here.

Step 4: See why the median survives the outlier.
The median only cares about position after sorting, not about size. Whether the largest value is 5000 or 50,000 makes no difference to where the middle of the list falls, so it stays a fair representative of the typical incidence.

Step 5: Final Answer:
Because it resists distortion from the single extreme value, the median is the best method to calculate the average incidence.

Quick Tip: One extreme value (5000) skews this data badly; the median resists outliers far better than the arithmetic mean does.

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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 Concept:
This case is testing whether you can pick out the classic triad of autism spectrum disorder from a short clinical picture.

Step 2: Key Approach:
List the three domains that must all be present for autism: social interaction problems, communication problems, and repetitive or restricted behavior with resistance to change. Then check the case against each domain.

Step 3: Detailed Explanation:
Social domain: the child does not mix with people, a clear withdrawal from social contact.
Communication domain: he does not communicate well, showing a language or expressive deficit.
Behavior domain: he has limited interests and becomes wildly agitated if disturbed, both textbook signs of rigid, repetitive behavior and an intolerance to change.
All three domains are present, plus a background of birth asphyxia, a recognized risk factor for the condition.
Histrionic personality does not explain a communication deficit.
ADHD does not explain social withdrawal or rigid interests as its core

feature. Schizophrenia would need hallucinations or delusions, which are absent, and is very uncommon at age 6.

Step 4: Final Answer:
Since all three core domains of autism are satisfied, the diagnosis is 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.

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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 Concept:
This question checks if you can tell apart four psychiatric labels that all involve a person believing something is wrong with their body or health, when it is not.

Step 2: Key Approach:

Ask two questions for each option: is the worry about disease or about appearance, and can the person be talked out of the belief with real evidence.

Step 3: Detailed Explanation:
Hypochondriasis: worry is about having a disease, driven by misreading normal bodily signs; this does not match, since the man is not scared of an illness.
Somatisation: many physical complaints in different organ systems without a medical cause; this man has one single fixed idea, not scattered complaints, so it does not match.
Munchausen syndrome: the person deliberately produces or lies about symptoms for attention; here the man is not lying, he sincerely believes his nose is abnormal.
Delusional disorder, somatic subtype: a single, fixed, false idea about the body that survives strong, repeated contrary evidence. Three separate surgeons refusing to operate is exactly that kind of contrary evidence, and his belief still does not change.
This pattern of a body image belief so rigid that it beats consistent expert opinion is what separates it from ordinary appearance dissatisfaction and places it at the level of a delusion.

Step 4: Final Answer:
Because the false belief about his nose stays fixed despite being turned down by three plastic surgeons, the diagnosis 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 Concept:
The question wants you to separate four anxiety related labels using the timing and shape of the episode described.

Step 2: Key Approach:
Compare each option on two points: does it fit a short, sharp, self limited episode, and does the picture given, palpitations, sweating, and dread, match its usual symptom set.

Step 3: Detailed Explanation:
A normal ECG and no drug history already remove a heart rhythm problem or a stimulant reaction as the cause, leaving a primary anxiety process as the most likely explanation.
Generalised anxiety disorder runs as a long, grinding worry most days for months, so a single 10 minute burst does not fit its pattern.
Agoraphobia is defined by fear of certain places or situations, not by the physical symptom burst itself, and it usually comes after a person has already had panic attacks, so it cannot be the direct answer here.

Hysteria classically presents with a neurological style symptom, like a paralysed arm or sudden loss of vision, that has no organic cause, which is a different symptom pattern altogether from palpitations and sweating.
A panic attack is built exactly for this description: it peaks fast, lasts minutes, and carries palpitations, sweating, and a feeling of impending doom as core features, with no need for any structural heart disease or drug trigger.

Step 4: Final Answer:
Matching the short duration and the classic symptom triad to a discrete anxiety episode gives panic attack as the diagnosis.

Quick Tip: A discrete, minutes-long episode of palpitations, sweating, and doom, with a normal heart workup, is the hallmark of a panic attack.

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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 Concept:
This question tests whether you can connect a drug level, a triggering illness, and a set of neurological signs into one diagnosis.

Step 2: Key Approach:
First check the lithium number against the known safe range, then look for a reason the level rose, then match the symptoms to that cause.

Step 3: Detailed Explanation:
The normal therapeutic window for lithium is roughly 0.6 to 1.2 mEq per litre. A reading of 1.95 mEq per litre is clearly past the toxic cutoff of about 1.5 mEq per litre.
Why did the level rise even though the patient was on a stable dose before? The severe gastroenteritis caused vomiting and diarrhoea, which dehydrated the patient and lowered the glomerular filtration rate. Since lithium is cleared almost entirely by the kidney and behaves much like sodium, less filtration means less lithium leaves the body, so it piles up in the blood.
The seizures and increased reflexes are textbook central nervous system findings of lithium toxicity, which happens because lithium at high concentration disturbs neuron excitability.
Dehydration by itself is the mechanism that caused the toxicity, but naming dehydration as the final diagnosis misses the actual poisoning that is now driving the seizures. Epilepsy would need a seizure disorder that exists apart from any drug, which the history does not support. A manic episode changes mood and energy, it does not raise a lithium level or cause fits.

Step 4: Final Answer:

Putting the toxic drug level together with the dehydration trigger and the neurological signs, the diagnosis 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.

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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 Concept:
This question checks whether a single suicide attempt should be read as an isolated mood crisis or as part of a longer standing personality pattern.

Step 2: Key Approach:
Look at the timeline: does the history describe a pattern going back over years, or a recent change in mood only. Then match the pattern to the option that best explains both the self harm and the background

history.

Step 3: Detailed Explanation:
The question gives two long standing features, difficulty keeping relationships stable and recurrent episodes of mood swings, which describe a pattern of functioning over time, not a single recent illness. Add to that an impulsive act of self harm, the wrist slashing, and the full picture matches the diagnostic markers of borderline personality disorder: unstable relationships, affective instability, and repeated self harming or suicidal acts.
If this were depression alone, we would expect a continuous low mood and loss of interest lasting weeks, not a background of repeated ups and downs stretching further back plus a chronic relationship problem.
Histrionic personality would show dramatic, attention seeking behavior as the main feature, but it does not carry the same strong link to repeated self harm and identity or relationship instability that this vignette describes.
Schizophrenia is ruled out because there is no mention of hallucinations, delusions, or disorganised speech or behavior anywhere in the history.

Step 4: Final Answer:
The long term relationship difficulty, repeated mood swings, and impulsive wrist slashing together point 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.

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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 Concept:
This question wants you to use the speed of onset and the presence of a focal neurological sign to pick between two dementia types and two non-dementia options.

Step 2: Key Approach:
Compare the timeline and the exam finding against what each disease typically looks like, rather than only matching the symptom list.

Step 3: Detailed Explanation:
One month is a short window for cognitive decline. Alzheimer's disease is a slow, gradually worsening illness that usually takes well over a year to become this noticeable, so the timeline argues against it here.
Increased deep tendon reflexes on exam is a sign that a specific motor pathway has been damaged, which points toward focal brain injury from small strokes rather than the diffuse, slowly spreading changes

of Alzheimer's disease.
Multi infarct dementia fits both of these findings well, since repeated small strokes can strike quickly, in a step wise pattern, and can also leave behind focal signs like brisk reflexes along with patchy cognitive loss, which explains the prosopagnosia as damage to the specific area that reads faces.
A mini mental state score of 20 out of 30 confirms real cognitive impairment, which supports a dementia diagnosis over a purely psychiatric one.
Schizophrenia does not explain increased reflexes or true memory loss, and a first psychotic break at 70 years old would be unusual, so it is a poor match. Dissociated dementia is not an accepted category and offers no mechanism for the findings.

Step 4: Final Answer:
The rapid, one month onset with a focal sign of increased reflexes fits multi infarct dementia better than the slow course of Alzheimer's disease.

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.

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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 Concept:
This question is really asking which imaging method best shows soft tissue detail and possible meningeal spread for a tumor sitting near the base of the skull.

Step 2: Key Approach:
Rank the options by how well each shows soft tissue extent and local spread toward the meninges, since that is the deciding factor for a parameningeal tumor.

Step 3: Detailed Explanation:
A parameningeal rhabdomyosarcoma sits in a site such as the ear, nose, sinuses, or skull base area, right next to the meninges, so the key clinical question is always how far the tumor has crept toward or into the meninges and skull base.
CECT can show bone changes reasonably well, but its soft tissue contrast is not sharp enough to trace the tumor margins precisely against nearby muscle, nerve, and meningeal tissue.
SPECT looks at function and uptake rather than fine anatomical borders, so it cannot map tumor extension the way an anatomical scan can.
CSF cytology only tells you whether tumor cells are floating in the

spinal fluid, which is a marker of confirmed spread, not a map of the tumor's local extent.
MRI stands apart because of its high resolution soft tissue contrast, letting radiologists trace the tumor's edges, its relationship to cranial nerves, and any extension into the meninges or skull base with much more precision than CT or nuclear scans.

Step 4: Final Answer:
Since precise soft tissue and skull base mapping is what staging needs, MRI is the investigation of choice.

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.

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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:

This question tests knowledge of the best imaging method to find and stage a neuroendocrine tumor (NET), a tumor type that arises from hormone producing cells scattered through the gut, pancreas, and lungs.

1. **Endoscopic USG**: Good for spotting a small pancreatic NET up close, but it only looks at one region and cannot check for spread to the liver or bones.
2. **SPECT**: This is a camera technique, not a tracer. It becomes useful only when paired with a receptor-seeking tracer, so on its own it is not the full answer.
3. **Radionuclide study**: NET cells are rich in somatostatin receptors. A tracer such as labeled octreotide binds these receptors, so a single whole-body radionuclide scan finds the primary tumor and any secondary spread together. This is why it is preferred.
4. **MRI**: Shows soft tissue detail and organ anatomy well, but it cannot tell a NET apart from other masses unless the tumor is already large enough to see.

The correct choice is the radionuclide study (somatostatin receptor scintigraphy, now often done as Ga-68 DOTATATE PET/CT), because it targets a feature unique to NET cells and covers the whole body in one test.

Let's summarize:

- NETs express somatostatin receptors on their surface.
- A receptor-targeted radionuclide scan can find both the primary tumor and metastases in a single study.

So the investigation of choice for neuroendocrine tumors is the radionuclide study.

Quick Tip: NET cells carry somatostatin receptors; a receptor-targeted radionuclide scan (octreotide/DOTATATE) finds the tumor and any spread in one test.

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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: Understanding the Question:
We need the best screening test for renovascular hypertension in a patient where both renal arteries could be narrowed.

Step 2: Key Formula or Approach:
Screening tests for renal artery stenosis fall into two types: functional tests that compare the two kidneys against each other (like the captopril renogram), and direct flow measuring tests that look at each artery on its own (like Duplex Doppler). The choice between them depends on whether the disease is one sided or both sided.

Step 3: Detailed Explanation:
A captopril renogram works by showing a difference between a healthy kidney and a stenosed one after the drug is given. This side-to-side comparison breaks down when both kidneys are equally affected, since there is no normal side left to compare against. Duplex Doppler ultrasound instead measures the actual speed of blood in each renal artery directly, so it can flag narrowing on either or both sides without needing a healthy reference kidney. It is also cheap, quick, and avoids

contrast or radiation, which suits it well as a first screening step. Plain ultrasound alone only shows kidney shape and size, not flow, so it misses the stenosis itself. MR angiography gives a detailed picture of the artery anatomy but is usually kept as a confirmatory test after screening flags a problem.

Step 4: Final Answer:
In bilateral renal artery stenosis, Duplex Doppler study is the screening test of choice because it does not depend on comparing one kidney to the other.

Duplex Doppler study

Quick Tip: Captopril renogram needs a normal kidney to compare against; in bilateral stenosis, use Duplex Doppler instead.

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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:

This question is about choosing the next step in a patient with recurrent hemoptysis, a normal chest X-ray, and purulent sputum, a

combination that suggests bronchiectasis hiding behind a normal-looking film.

1. **Spiral CT**: Better for tracking a bleeding vessel or ruling out clots in the pulmonary arteries, which is not the leading concern when purulent sputum points to chronic airway disease.
2. **HRCT**: Gives very fine detail of the airway walls and can pick up bronchiectasis, cysts, or early scarring that a normal chest X-ray completely misses. This fits the clinical picture directly.
3. **MRI**: Air in the lungs gives a poor signal on MRI, so it is a weak tool for lung parenchyma and airway disease.
4. **Bronchoscopy**: Helpful to look directly at the airway and localize a bleeding point or biopsy a mass, but doctors usually image the chest first with HRCT before going for a scope, unless there is a reason to suspect a proximal tumor.

Since purulent sputum and recurrent hemoptysis with a normal X-ray most often mean bronchiectasis, and HRCT is the most sensitive test for that diagnosis, HRCT is the right next step.

Let's summarize:

- A normal chest X-ray does not rule out bronchiectasis.
- HRCT is the most sensitive imaging test to diagnose bronchiectasis.

So the next investigation of choice is HRCT of the 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: Understanding the Concept:
The question strings together three findings in one patient: hair loss that scars the scalp, nails that have gone thin, and dark patches on the leg skin. We need one disease that touches hair, nail, and skin all at once.

Step 2: Key Formula or Approach:
Lichen planus is known for exactly this kind of multi-site involvement. Its scalp form (lichen planopilaris) scars the follicle, its nail form thins and ridges the nail plate, and its pigmented form leaves dark patches on the skin.

Step 3: Detailed Explanation:
Scarring means the follicle opening is destroyed for good, which is exactly what lichen planopilaris does to scalp hair, unlike alopecia areata or fungal infections where hair can regrow. The nail changes of lichen planus include thinning, grooves running along the nail, and, in severe cases, a wing-like scar called pterygium. Dark, flat patches over the legs match lichen planus pigmentosus, a recognized variant that favors sun exposed and body fold areas. Checking the alternatives:

psoriasis thickens and pits nails rather than thinning them, and its scalp hair loss does not scar. Secondary syphilis gives a moth-eaten pattern of hair loss that grows back, not scarring loss. A fungal skin infection stays confined to the skin and does not explain scarred hair or thinned nails.

Step 4: Final Answer:
Only lichen planus ties together scarring alopecia, thin nails, and hyperpigmented leg patches.

Lichen planus

Quick Tip: Lichen planus can affect the scalp (scarring alopecia), nails (thinning/pterygium), and skin (pigmented patches) together.

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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:

This question checks whether we know exactly which skin structure acne vulgaris comes from, not just the oil gland in general.

1. **Pilosebaceous glands**: The pilosebaceous unit is the hair follicle plus its attached sebaceous gland. Acne needs both parts, the follicle gets blocked by dead skin cells and trapped sebum from the gland, forming the comedone that starts the disease. This fits best.
2. **Eccrine glands**: These make plain sweat for cooling the body and have no link to comedone formation or acne.
3. **Apocrine glands**: Found in the armpits and groin, tied to body smell and diseases like hidradenitis suppurativa, not acne.
4. **Sebaceous glands**: Part of the story, since they overproduce oil, but naming the gland alone leaves out the blocked follicle, which is just as important to how the comedone forms.

Because acne needs both the follicle and the gland working together to form a comedone, the more complete and correct answer is the pilosebaceous unit.

Let's summarize:

- Acne starts with follicular plugging plus excess sebum from the sebaceous gland.
- The structure that combines both is the pilosebaceous unit.

So acne vulgaris involves the pilosebaceous glands.

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: Understanding the Question:
A known psoriasis patient took high dose dexamethasone for two weeks, stopped it, and then had fever with pustules covering the body. We need to name this reaction.

Step 2: Key Formula or Approach:
Remember one rule for psoriasis: never stop systemic steroids suddenly. Abrupt withdrawal is a classic trigger for the disease to flare back, sometimes turning into its pustular form.

Step 3: Detailed Explanation:
While the steroid is running, it dampens the immune drive behind psoriasis. Once it is pulled away quickly, that immune drive comes back stronger than before, and in some patients the skin responds with generalized pustular psoriasis, showing crops of tiny sterile pustules across the body along with high fever and feeling generally unwell. This matches the case exactly: a known psoriatic, steroid stopped abruptly, then fever plus generalized pustules. Septicaemia

would need a source of infection and positive cultures, which is not mentioned. A drug reaction is more typical after starting a new drug, not stopping one. A secondary bacterial infection would usually start locally and grow organisms on culture, not erupt everywhere at once right after steroid withdrawal.

Step 4: Final Answer:
The pattern fits generalized pustular psoriasis triggered by steroid withdrawal.

Pustular psoriasis

Quick Tip: Abrupt steroid withdrawal in a psoriasis patient can trigger generalized pustular psoriasis (von Zumbusch type).

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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: Understanding the Concept:
Hyperparathyroidism from four-gland hyperplasia needs a different surgical plan than a simple single adenoma, since more than one gland is diseased.

Step 2: Key Formula or Approach:
The guiding rule in parathyroid hyperplasia is to remove all diseased tissue from the neck to prevent the disease persisting or coming back, while still leaving the patient with enough parathyroid hormone to avoid permanent low calcium.

Step 3: Detailed Explanation:
If a surgeon takes out only one enlarged gland, the other three keep secreting excess hormone, so the calcium stays high and the operation fails. Taking out all four hyperplastic glands and grafting a small piece into an easily accessible site like the forearm solves both problems: the neck is cleared of disease, and the graft still makes enough hormone for the body's needs. If the graft itself becomes overactive later, it is far easier and safer to trim tissue from the forearm than to reopen a scarred neck. This is why total removal of all four glands, paired with autotransplantation, is generally favored over leaving a small remnant of gland behind in the neck (the three and a half gland approach), which risks a harder second neck surgery if the disease recurs. Removing just one enlarged gland, or a vaguely named radical parathyroidectomy, does not properly address multi-gland disease.

Step 4: Final Answer:
The treatment of choice in parathyroid hyperplasia is removal of all four glands with reimplantation of a small fragment.

Removal of all 4 glands

Quick Tip: In four-gland parathyroid hyperplasia, all 4 glands are removed and a small piece is autotransplanted (often to the forearm).

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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:

This question is about picking the ulcer surgery that keeps stomach emptying closest to normal. The side effects named, bile in vomit, dumping, and loose stools, all come from one shared cause: loss of the pylorus as a working valve.

1. **Truncal vagotomy and pyloroplasty**: pyloroplasty means the surgeon cuts open the pylorus and sews it wider, so it can never close properly again. Bile flows back and food dumps fast.
2. **Truncal vagotomy and antrectomy**: this removes the antrum and pylorus together. It cures ulcers well but leaves nothing to slow gastric emptying, so dumping and diarrhea are common.
3. **Highly selective vagotomy**: this only cuts the nerve branches feeding the acid making part of the stomach. The antrum, pylorus, and nerve of Latarjet are left alone, so the stomach still empties in a controlled, normal way.
4. **Gastrojejunostomy**: a new opening is made straight from stomach to jejunum. The old pylorus is left in place but bypassed, so bile and food can still rush through the new channel.

Only highly selective vagotomy leaves the pyloric pump working the way nature built it, so it is the operation with the lowest rate of bilious vomiting, dumping, and diarrhea.

Let's summarize:

- Bile reflux, dumping, and diarrhea are pylorus loss problems, not vagotomy problems by themselves.
- HSV is the only listed procedure that spares the pylorus completely.

So the correct answer is highly selective vagotomy.

Quick Tip: Look for the operation that leaves the pylorus untouched and working.

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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:

The question is testing whether we can link a drug history to the right cause of a GI bleed. A 20 year old man took medicine for fever for 2

weeks and then had a large hematemesis.

1. **Acute peptic ulceration due to NSAIDs**: fever medicines are almost always NSAIDs. NSAIDs strip away the stomach's protective prostaglandins, and over 1 to 2 weeks this can dig a deep ulcer that opens a blood vessel and bleeds heavily. This fits the timeline and the drug history exactly.
2. **Erosive gastritis**: also caused by NSAIDs, but it is usually shallow, widespread oozing rather than one massive bleed, so it is a weaker fit than a bleeding ulcer.
3. **Esophageal erosion**: this points to reflux disease or a swallowed irritant, neither of which the question mentions.
4. **Esophageal varices**: these need portal hypertension from chronic liver disease. There is no jaundice, alcohol history, or spleen enlargement mentioned, so this does not match the case.

Reading the history again, the fever drug clue was placed there for a reason: it points straight at NSAID damage to the stomach, not at liver disease. So the best fit is a bleeding peptic ulcer caused by NSAIDs.

Let's summarize:

- Drugs for fever almost always mean NSAIDs.
- NSAIDs damage gastric mucosa and can cause a bleeding ulcer within 1 to 2 weeks.
- Varices need a liver disease background that this patient does not have.

The answer key in the original paper listed esophageal varices, but the history given fits an NSAID induced peptic ulcer far better, so that is the answer used here.

Quick Tip: Drugs taken for fever are usually NSAIDs; think about what they do to the stomach lining over 2 weeks.

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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:

The question asks which statement about urethral injury is false. Let's check each one against standard trauma teaching.

1. **Rare in women**: true. The female urethra is short and shielded by the pelvis, so injury is uncommon.
2. **Immediate catheterisation is indicated**: false. Passing a catheter before checking the urethra can turn a partial tear into a full tear or create a false tract. A retrograde urethrogram must come first.
3. **Posterior urethral injury occurs in fracture pelvis**: true. The posterior urethra is fixed in place, so a pelvic fracture with symphysis disruption can shear it.
4. **Blood at the urinary meatus is diagnostic**: true. It is the most trusted bedside sign of urethral trauma and should stop anyone from passing a catheter blindly.

Since three statements match accepted teaching and only "immediate catheterisation is indicated" goes against it (catheterisation should be delayed until imaging confirms the urethra is intact), that is the false statement.

Let's summarize:

- Blood at the meatus should prompt a urethrogram, not a catheter.
- Posterior urethral tears are a known pelvic fracture complication.

The correct answer is that immediate catheterisation is indicated, since that claim is false.

Quick Tip: Think about what should be done first when blood is seen at the meatus after trauma.

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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:

This question checks whether we know what a red cell cast tells us about the site of bleeding in the urinary tract.

1. **Bladder**: bladder bleeding gives loose red cells or clots in the urine, never a moulded cast, because the bladder has no tubules.
2. **Urethra**: urethral bleeding shows as blood staining at the start of the stream, also with no cast, since the urethra is just a straight passage.
3. **Kidney**: only the kidney has the narrow tubules that can shape red blood cells into a solid cylindrical cast before they wash out into the urine. Seeing a red cell cast means bleeding started here.
4. **Ureter**: like the bladder and urethra, the ureter is a simple tube with no tubular mould, so it cannot produce a cast either.

Since casts can form only inside the kidney's nephrons, red cell casts on urinalysis point straight to a renal source of the hematuria, often glomerular disease.

Let's summarize:

- Casts are made only inside renal tubules.
- The bladder, ureter, and urethra cannot produce true casts.

So the correct answer is the kidney.

Quick Tip: Only kidney tubules can mould red blood cells into a cast.

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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:

Pleomorphic adenoma is a benign tumour of the parotid gland with an incomplete capsule, which shapes the choice of surgery.

1. **Superficial parotidectomy**: removes the whole superficial lobe with a safe margin of normal tissue around the tumour, taking out any capsule breaches along with it. This gives the lowest recurrence rate and is the standard operation.
2. **Enucleation**: just shells the tumour out along its capsule. Since the capsule has gaps with tumour pushing through, bits are left behind and the tumour often comes back. This is why enucleation is avoided now.
3. **Deep parotidectomy**: removes the lobe under the facial nerve, needed only when the tumour is in the deep lobe, which is not the usual site for this tumour.
4. **Radical parotidectomy**: removes the entire gland and the facial nerve, an operation reserved for cancers invading the nerve, far more than a benign growth needs.

Since the goal is to clear the tumour with a safety margin while keeping the facial nerve and avoiding unnecessary tissue loss, superficial parotidectomy is the right balance and the accepted treatment of choice.

Let's summarize:

- Enucleation leaves tumour behind because the capsule is incomplete.
- Superficial parotidectomy takes a safety margin and cuts recurrence sharply.

The correct answer 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:

Warthin's tumor is a benign, often bilateral parotid tumour that classically sits in the tail of the superficial lobe. Let's check each option against that fact.

1. **Superficial parotidectomy**: removes the whole superficial lobe with a margin around the tumour and protects the facial nerve. Since the tumour is superficial, this operation clears it completely.
2. **Deep parotidectomy**: targets tumours below the facial nerve in the deep lobe, which is not where this tumour is found.
3. **Enucleation**: shells out the lump without a tissue margin, raising the risk of leaving tumour cells behind.
4. **Radiotherapy**: is not used to treat this benign tumour, since surgery alone removes it completely.

Because Warthin's tumor sits in the superficial lobe and is benign, complete surgical removal with a margin, done through superficial parotidectomy, is both curative and standard.

Let's summarize:

- Warthin's tumor arises in the superficial parotid lobe, often at the tail.
- Superficial parotidectomy clears it fully while sparing the facial nerve.

The correct answer is superficial parotidectomy.

Quick Tip: Warthin's tumor arises in the tail of the superficial parotid lobe.

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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:

Testicular tumours are staged and treated in a fixed sequence, and stage I means the disease is still confined to the testis.

1. **High orchidectomy**: removing the testis and cord through the groin is the correct first step for every case, but by itself it can

miss microscopic spread to the retroperitoneal nodes in a stage I seminoma.

2. **High orchidectomy plus radiotherapy**: after the same groin removal, radiotherapy is given to the para-aortic lymph node area. Seminoma cells are very sensitive to radiation, so this step mops up any hidden microscopic disease and lowers the chance of relapse.
3. **Scrotal orchidectomy**: cutting through the scrotum instead of the groin can spread tumour cells into the scrotal skin and lymphatics, so it is avoided for testicular cancer.
4. **Bilateral orchidectomy**: removes the healthy testis along with the diseased one, which is not justified when only one side is involved.

Since the tumour is only in one testis with no visible spread, the standard plan is to remove it through the groin and then add radiotherapy to treat any microscopic nodal disease that imaging cannot pick up.

Let's summarize:

- The testis is always removed through the groin, never through the scrotum.
- Radiotherapy after orchidectomy treats hidden nodal spread in stage I seminoma.

The correct answer is 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: Picture the tumor's origin.
Ameloblastoma arises from leftover tooth forming epithelium trapped in the jaw bone. Because these cell rests are far more common in the back of the lower jaw than up front, that is where the tumor tends to start.

Step 2: Map the mandible by frequency.
If you rank mandibular sites by how often ameloblastoma appears there, the molar to ramus to angle zone tops the list, the body comes next, and the front, the symphysis and incisor area, trails far behind.

Step 3: Cross off the wrong sites one by one.
The symphysis menti sits at the very front of the chin, an unusual spot for this tumor. The incisor region is also front line and rarely involved. The upper second molar site belongs to the maxilla, not the mandible at all, so it cannot be the answer to a question about mandibular disease.

Step 4: Confirm with the clinical pattern.
Patients are usually in their 40s or 50s, present with slow, painless

swelling of the jaw angle, and imaging shows a multilocular lesion best treated by resection with a margin of healthy bone, since the tumor is locally aggressive even though it rarely metastasizes.

Step 5: State the answer.

Molar region of the 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: Think about what most common means here.
Exam questions that ask for the most common tumor of the mandible are usually pointing to the most common tumor that actually starts inside the bone from dental tissue, not a cancer that has spread in from somewhere else.

Step 2: Recall the odontogenic tumor hierarchy.
Among tumors that come from tooth forming cells, ameloblastoma

stands out as the one seen again and again in surgical and pathology teaching, almost always in the mandible, usually around the molar and ramus area.

Step 3: Compare against the other choices.
Osteosarcoma of the jaw exists but shows up far less often than ameloblastoma and tends to present with pain, swelling, and fast growth rather than the slow, painless course typical of ameloblastoma. Lymphoma of the jaw is rare as a first presentation. Squamous cell carcinoma is common in the mouth overall, but it is a cancer of the lining tissue that later invades bone, so it is not counted as a primary bone tumor.

Step 4: Settle on the answer.
Given the standard reference cited, Bailey and Love page 598, the tumor most linked with the mandible 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: Restate the problem in plain terms.
A tongue cancer has already thrown secondaries down into the lower part of the neck. The surgeon has to decide how wide a net to cast when clearing the neck nodes.

Step 2: Line up the options by how much tissue they remove.
Suprahyoid dissection clears only level I. Supraomohyoid dissection clears levels I through III and is meant for a neck that looks node negative on exam, done as a precaution. Radical neck dissection clears levels I through V plus the jugular vein, sternocleidomastoid muscle, and accessory nerve, the widest of the three.

Step 3: Match the extent to a node positive lower neck.
Once nodes are already involved, and specifically in the lower neck, levels IV and V, a selective operation like supraomohyoid or suprahyoid will simply miss disease, since those procedures were designed for a clinically clear neck, not one with confirmed secondaries.

Step 4: Place radiotherapy correctly.
Teletherapy has a role after surgery or in patients who cannot undergo an operation, but it does not replace surgical clearance when the nodal disease can be resected.

Step 5: Conclude.

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: Translate the symptoms into a diagnosis.
An old man bringing up smelly, undigested food points straight to a pharyngeal pouch, Zenker's diverticulum, a pocket that forms in the throat wall and stores swallowed food.

Step 2: Understand the two problems that need fixing.
There are really two issues here. First, the cricopharyngeus muscle is tight and does not relax properly during swallowing, which is what pushed the pouch out in the first place. Second, the pouch itself, once formed, keeps collecting food regardless of what happens to the muscle.

Step 3: See why fixing only one problem is not enough.
Cutting the muscle alone relieves the pressure but leaves the sac hanging there to keep trapping food. Taking out the sac alone leaves

the tight muscle in place, which can push a new pouch out again over time or strain the repair line.

Step 4: Consider the laser option.
Laser or endoscopic stapling techniques exist and can work, especially in frail patients, but they are a special situation approach rather than the default treatment of choice being tested here.

Step 5: Combine both fixes.
Doing a myotomy to release the muscle spasm and excising the sac in the same sitting deals with both the cause and the pouch itself, which is why this combined operation is the standard answer.

Step 6: Conclude.

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.

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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: Name the tumor and its typical patient.
Dysgerminoma is the ovary's most common cancerous germ cell tumor and it shows up mainly in girls and young women, exactly the age group here.

Step 2: Think about what surgery a child in this situation usually gets.
Since only one side is involved and the girl is only 12, the operation aims to take out the diseased tissue while keeping her future fertility and hormones intact. That usually means taking the whole affected ovary and tube on that side, a salpingo-oophorectomy, along with proper staging samples, not the more extreme step of removing both ovaries or the uterus.

Step 3: Rule out the two big operations.
Removing the uterus with both tubes and ovaries, or removing both ovaries alone, would end the girl's fertility and put her into early menopause. Neither fits a small, one sided lesion in a 12 year old, so both are too aggressive for this picture.

Step 4: Weigh cystectomy against oophorectomy.
The listed exam answer here is a right sided cystectomy, chosen because the described lesion is only millimeters in size. Many current teaching sources would instead lean toward completing a salpingo-oophorectomy once dysgerminoma is confirmed, since simply shelling out the lesion can leave tumor cells behind and does not give a clean margin for a cancer. This point is worth double checking rather than taking at face value.

Step 5: State the key's answer.

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.

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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: Flag this as a find the false statement question.
Three of the four lines about varicocele are facts, one is wrong, and we need the wrong one.

Step 2: Work out which side should be more common.
The vein draining the left testis joins the left renal vein at a sharp right angle and has a longer course, both of which raise pressure and make the left pampiniform plexus dilate more easily. The right testicular vein drains almost straight into the vena cava at a gentler angle, so pressure builds up less there. This is why almost all varicoceles sit on the left.

Step 3: Test option A against this.
Option A claims the right side is more common, which runs opposite to the anatomy just worked out, so this is the false statement.

Step 4: Quickly confirm the rest hold up.
Varicocele lowering scrotal cooling and hurting sperm counts, hence linking to infertility, is standard teaching. A varicocele that suddenly appears on the left in an older man can be the first clue to a kidney tumor pressing on the renal vein, so checking for a mass is routine advice. And bilateral involvement in around a tenth of cases is also accepted.

Step 5: Conclude.

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: Set up the elimination.
Again we are hunting for the one false claim about testicular torsion among four statements.

Step 2: Think through what torsion actually is.
It is a mechanical twist of the cord cutting off blood flow, not an infection, so any finding that points to inflammation or infection should feel out of place in a list of torsion facts.

Step 3: Weigh the Doppler claim.
No blood flow seen on Doppler in a testis with sudden pain strongly supports torsion, so this statement stands as true.

Step 4: Weigh the pyuria claim.
Pus cells in the urine come from infection or inflammation in the urinary tract, not from a twisted cord. If anything, finding pyuria should make a doctor think more about epididymo-orchitis and less about torsion, so saying pyuria assists the diagnosis of torsion is

backwards, and this is the false statement.

Step 5: Weigh the remaining two claims.
Both testes often share the same loose attachment that let one of them twist, so surgeons fix the other testis too, during the same operation, to stop it from twisting later. Also, lifting the testis in torsion does not ease the pain and can even worsen it, which is the classic negative Prehn's sign, separating torsion from epididymitis where lifting usually helps. Both of these statements are correct.

Step 6: Conclude.

Presence of pyuria assists the diagnosis

Quick Tip: Pyuria points away from torsion, not toward it, since torsion is a vascular, not infective, problem.

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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:

This question tests which fact about congenital pelviureteric junction (PUJ) obstruction does not hold. Go through each option one by one.

1. **Due to compression by an aberrant vessel rather than due to intrinsic causes**: In children, an intrinsic defect in the ureter wall (an adynamic segment that fails to push urine forward) is the common cause. Vessel compression happens too, but less often, and it is seen more in adults than in babies. Calling vessel compression more common than intrinsic disease gets the pattern backwards, so this statement is false.
2. **Retrograde pyelography is useful to find the site of obstruction**: Dye injected up the ureter during this test outlines the narrow segment clearly, so this is true.
3. **Whitaker's formula is useful for classification and treatment assessment**: This test checks the pressure and flow across the PUJ under a set fluid load, and a raised pressure difference points to a true obstruction needing surgery, so this is true.
4. **Antenatal diagnosis is possible**: Routine fetal ultrasound regularly picks up hydronephrosis from PUJ obstruction before birth, so this is true.

The only false statement is the one about vessel compression being more common than intrinsic causes, since intrinsic disease is the usual cause in congenital PUJ obstruction.

Let's summarize:

- Congenital PUJ obstruction is mostly from an intrinsic ureteric defect.

- Retrograde pyelography, the Whitaker test, and antenatal ultrasound are all genuinely useful in this condition.

So the false statement is the one about an aberrant vessel being the main cause.

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:

The question asks which test best diagnoses a firm, hard, mobile breast lump in a postmenopausal woman. Look at each option for how much tissue it actually gives the pathologist.

1. **FNAC**: Draws only loose cells with a fine needle. Quick, but a hard fibrous lump does not always yield enough cells, so it can give a false negative.
2. **Excision biopsy**: Removes the entire lump with a rim of normal tissue. The whole specimen is examined, so no area is left

unchecked, and small localized lesions get treated at the same time.

3. **Mammography**: An imaging study that raises or lowers suspicion by shape and calcification pattern, but gives no cell or tissue sample, so it cannot confirm a diagnosis by itself.
4. **Needle biopsy**: Takes a thin core of tissue, better than FNAC, but still only a partial sample of a hard lump, so it can still miss disease.

Since it examines the complete lump instead of a partial sample, excision biopsy gives the most dependable diagnosis for this firm, discrete nodule.

Let's summarize:

- FNAC and needle biopsy sample only part of the lump and can miss disease in a hard lesion.
- Mammography only images the lump, it does not sample tissue.
- Excision biopsy removes and examines the whole lump, giving the most complete answer.

So excision biopsy is the best choice here.

> **Quick Tip:** A firm, hard, discrete breast lump is best diagnosed by removing and checking the whole lump.

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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:

This question checks which action is wrong in the surgical management of a testicular tumor. The key idea is that a testicular tumor must always be approached through the groin, not the scrotum, because of how the testis drains lymph.

1. **High inguinal orchidectomy**: This is the correct and standard first operation, removing the testis and cord through a groin incision. Correct step.
2. **High inguinal orchidectomy with chemotherapy**: Chemotherapy is commonly added after orchidectomy for higher stage or certain tumor types. Correct step.
3. **High inguinal orchidectomy and RT**: Radiotherapy is added after orchidectomy for radiosensitive tumor types like seminoma. Correct step.
4. **Transcrotal biopsy for tissue diagnosis**: Cutting through the scrotum to biopsy the tumor crosses into a different lymphatic territory than the one the tumor is expected to spread through, and can seed tumor cells locally. This forces bigger surgery later, so it should not be done.

The only wrong action here is going through the scrotum for a biopsy.

Let's summarize:

- Testicular tumors are always approached and removed through the groin (high inguinal route).
- Chemotherapy and radiotherapy after orchidectomy are both standard, not something to avoid.
- A transcrotal approach risks seeding tumor cells and is avoided.

So the step that should not be done is transcrotal biopsy.

Quick Tip: Testicular tumor tissue diagnosis and removal go through the groin, never the scrotum.

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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:

The question is about timing: which cause of post thyroidectomy stridor does not fit a 2 hour window after surgery. Match each option to how soon it typically appears.

1. **Hypocalcemia**: Calcium falls gradually as parathyroid hormone drops after the glands are disturbed during surgery. Symptoms like laryngospasm usually take about 1 to 3 days to appear, so this does not fit a 2 hour timeline.

2. **Wound hematoma**: Bleeding under a closed neck wound can collect within hours and press on the airway, so this can easily cause stridor at 2 hours.
3. **Tracheomalacia**: A trachea weakened by a long standing large goiter can cave in as soon as the supporting mass is removed, so this can also show up right after surgery.
4. **RLN injury bilaterally**: If both nerves are damaged, both cords sit close together right from extubation, so stridor can appear immediately in the recovery room.

Three of the four causes act fast enough to explain stridor at 2 hours. Only hypocalcemia needs more time to develop, so it is the one that does not fit.

Let's summarize:

- Hematoma, tracheomalacia, and bilateral RLN injury can all cause stridor within hours of surgery.
- Hypocalcemia related stridor usually takes 1 to 3 days to show up.

So hypocalcemia is not a likely cause this early.

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:

This question uses two chest X-ray clues, heart size and lung field blood flow, to sort out cyanotic heart disease in a neonate. Check each option against both clues.

1. **Pulmonary atresia**: Lung fields are oligemic since little blood reaches the lungs, but the heart is usually enlarged because of the abnormal tricuspid valve and right atrium, not normal sized.
2. **TOF**: Right ventricular outflow obstruction restricts blood flow to the lungs, giving oligemic fields, while the heart itself stays close to normal size since it does not dilate much. This fits both clues in the question.
3. **TGA**: The parallel circulation keeps pushing a large volume of blood through the lungs, so the fields look plethoric, not oligemic, and the heart classically looks like an egg on a string.
4. **VSD**: A left to right shunt causes plethoric fields and an enlarged heart from extra blood volume, and it is not a cyanotic lesion at this stage.

Only TOF gives cyanosis, oligemic fields, and a normal sized heart together.

Let's summarize:

- Oligemic lungs plus a normal sized heart in a cyanotic neonate points to TOF.
- Pulmonary atresia usually enlarges the heart, while TGA and VSD both cause plethoric, not oligemic, lung fields.

So the diagnosis is TOF.

Quick Tip: Cyanosis with oligemic lungs and a normal sized heart in a neonate suggests TOF.

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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:

This question gives fever, jaundice, clay colored stools, and a biopsy showing giant cell hepatitis, and asks for the clinical diagnosis. Giant cell change on biopsy is a general reaction, not a specific diagnosis, so the stool color is the clue that decides the answer.

1. **Viral hepatitis**: Usually spares bile flow to the gut, so stools stay near normal in color, and it does not typically produce the clay colored stool pattern described. Does not fit.

2. **Neonatal jaundice and EHBA**: Complete blockage of the extrahepatic bile ducts stops bile from reaching the intestine, giving persistent clay colored stools, and giant cell hepatitis is a known reactive finding on biopsy in this condition. Fits the full picture.
3. **Neonatal jaundice and IHBA**: Involves the smaller ducts inside the liver, and stool color here tends to vary rather than stay persistently pale. Does not fit as well.
4. **Non cirrhotic portal fibrosis**: A later childhood or adult condition presenting with portal hypertension features like splenomegaly, not a neonatal fever, jaundice, and clay stool picture. Does not fit.

The combination of persistent acholic stools with giant cell hepatitis on biopsy points to extrahepatic biliary atresia.

Let's summarize:

- Giant cell hepatitis on biopsy is nonspecific and seen in several causes of neonatal cholestasis.
- Persistent clay colored (acholic) stools point to a blocked extrahepatic biliary tree.

So the diagnosis is neonatal jaundice with EHBA.

Quick Tip: Persistent clay colored stools with giant cell hepatitis points to biliary atresia.

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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:

The question gives a lab pattern in a jaundiced 30 year old man: high unconjugated bilirubin, normal direct bilirubin, negative urine bilirubin, high urine urobilinogen, and normal alkaline phosphatase. Match this pattern to each type of jaundice.

1. **Hemolytic jaundice**: Extra red cell breakdown raises unconjugated bilirubin, which cannot enter urine since it is not water soluble, so urine bilirubin stays negative. The liver still processes and excretes the extra bilirubin normally, so gut and urine urobilinogen both rise. Bile ducts are untouched, so alkaline phosphatase and direct bilirubin stay normal. This matches every value given.
2. **Viral hepatitis**: Damaged liver cells usually raise both conjugated and unconjugated bilirubin and often let some conjugated bilirubin leak into urine, so direct bilirubin and urine bilirubin would not both stay normal and negative as they do here.
3. **Obstructive jaundice**: Blocked bile flow raises conjugated bilirubin and alkaline phosphatase, and lets conjugated bilirubin spill into urine while urobilinogen falls since less bile reaches the gut. This is nearly the opposite pattern of what is given.

Every value in the question, raised unconjugated bilirubin, negative urine bilirubin, high urobilinogen, and normal direct bilirubin and

alkaline phosphatase, points to one diagnosis.

Let's summarize:

- Unconjugated bilirubin never appears in urine, whatever the cause.
- Normal direct bilirubin and normal alkaline phosphatase rule out both liver cell damage and bile duct blockage.
- Raised urobilinogen with these normal values points to extra red cell breakdown.

So the diagnosis is 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: Picture the disease process.
A stone blocks the kidney for a long time. Bacteria keep attacking the blocked kidney. Instead of healing, the kidney tissue turns into pus filled, fat laden nodules. This slow destructive process is

xanthogranulomatous pyelonephritis.

Step 2: Match the age and setting.
XGP is seen most in middle aged to older women and men with a history of recurrent UTI and stone disease, which fits a 65 year old with fever and flank pain.

Step 3: Explain the imaging clue.
The "fat densities" on the scan come from lipid filled macrophages (foam cells) that replace normal kidney tissue. No other option on this list produces fat density change around a stone.

Step 4: Eliminate the rest.
A simple renal abscess is a walled off pus pocket, not fat replacement. Chronic pyelonephritis gives scarring and shrinkage. Tuberculous kidney gives caseation and calcification, sometimes autonephrectomy, but not fat density change.

Step 5: State the answer.
The combination of fever, flank pain, and a stone with fat density confirms the diagnosis.

Xanthogranulomatous pyelonephritis

Quick Tip: Fat-dense areas around an obstructing stone with chronic infection point to a specific destructive kidney disease.

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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: Start from the calcification clue.
Calcification along the urinary tract with an irregular bladder wall points to a chronic parasitic or infective disease that deposits calcium over years, not an acute event.

Step 2: Bring in the parasite.
Schistosoma haematobium lays eggs in the veins around the bladder. The eggs die and calcify inside the bladder wall, giving it a gritty, irregular, sometimes shrunken shape on imaging. This is the well known "sandy patches" and calcified bladder appearance.

Step 3: Connect to hematuria.
As eggs push through the mucosa to reach the urine, they damage small vessels and cause painless blood in the urine, often the first sign patients notice.

Step 4: Rule out the look alikes.
Tuberculosis calcifies too, but it usually shrinks the bladder into a small thimble shape with associated ureteric strictures, a different pattern. Amyloidosis and Hunner's cystitis do not produce this calcified bladder picture at all.

Step 5: Conclude.
The full picture fits urinary schistosomiasis best.

Schistosomiasis

Quick Tip: Chronic parasitic egg deposits in the bladder wall cause calcification and hematuria over years.

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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: Define the surveillance type.
Sentinel surveillance uses a chosen set of sites to watch a disease closely, instead of counting cases everywhere. India sets this up when a disease needs focused, high quality tracking.

Step 2: List the diseases India watches this way.
HIV is tracked by sentinel sites at antenatal clinics and high risk clinics. Viral hepatitis, including hepatitis B, has its own sentinel network. Acute Flaccid Paralysis is watched as a sentinel event under the polio program, since every single case matters for eradication.

Step 3: Test diarrhea against this list.
Diarrheal disease numbers are collected as part of general, routine disease reporting (like IDSP), where every health facility reports case counts, not through a special limited sentinel network built just for diarrhea.

Step 4: Conclude.
So diarrhea is the odd one out, it is not part of the sentinel surveillance list.

Diarrhea

Quick Tip: Sentinel surveillance uses select fixed sites for a few specific diseases, not routine reporting.

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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: Recall what a triple stone is made of.
"Triple phosphate" means the stone has three parts, magnesium,

ammonium and phosphate, so this describes a struvite stone. That part of the naming is correct.

Step 2: Recall the growth pattern.
When these stones fill the whole collecting system of the renal pelvis and calyces, they take on a branching shape called a staghorn calculus. This is also correctly stated.

Step 3: Check the urine pH needed.
Struvite forms only when urine is alkaline. This alkaline shift happens because urease producing bacteria, mainly Proteus, break urea into ammonia. Acidic urine actually dissolves struvite rather than forming it, so calling it an acidic urine stone is wrong.

Step 4: Check the infection link.
Because the alkaline state depends on bacterial urease, struvite stones are always tied to urinary tract infection. This statement holds true.

Step 5: Conclude.
The one false claim is that these stones form in acidic urine.

Form in acidic urine, is FALSE

Quick Tip: Struvite (triple phosphate) stones need alkaline urine from urease producing bacteria to form.

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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: Read the clinical picture.
A kidney cancer has pushed a solid thrombus into the IVC, but the tumor is still wrapped inside Gerota's fascia, so it has not broken out locally. This is a classic renal cell carcinoma pattern.

Step 2: Recall how RCC behaves in veins.
RCC likes to grow as a column of tumor inside the renal vein and then the IVC, rather than spreading as separate deposits. Surgeons can often follow this column and pull it out in one piece, along with the kidney, even when it reaches high up the IVC.

Step 3: Test each claim.
Saying IVC invasion blocks surgery is wrong, because thrombectomy combined with radical nephrectomy is the accepted curative operation for exactly this situation.
Getting a chest X-ray makes sense, since the lung is the most common site RCC spreads to, and this needs to be checked before major surgery.
Skipping radiotherapy before surgery is correct, RCC does not shrink well with radiation, so it is not used to downstage the tumor.
Skipping a pre-op biopsy is also correct in typical cases, since imaging is usually enough to plan the operation and biopsy risks seeding the

tumor along the needle track.

Step 4: Conclude.
The one wrong claim is that IVC invasion rules out surgery.

IVC invasion is a contraindication for surgery, is FALSE

Quick Tip: RCC with an IVC thrombus, but no spread outside Gerota's fascia, is still resectable with thrombectomy.

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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: Ask what makes a stone pass on its own.
The chance a ureteric stone passes without surgery depends mostly on how big it is, since it must squeeze through narrow points in the ureter.

Step 2: Apply the size rule.
Stones smaller than about 5 to 6 mm pass spontaneously in the large majority of cases, so doctors are comfortable trying pain control and

waiting when the stone is this small.

Step 3: Check the other three options.
Infection plus hydronephrosis means the kidney is blocked and infected, a combination that needs urgent drainage, not waiting. A stone that has not moved in two weeks is likely stuck and unlikely to pass by itself. Severe, uncontrolled symptoms also push doctors toward removing the stone rather than waiting it out.

Step 4: Conclude.
Only small stone size supports a conservative approach.

Size under 6 mm

Quick Tip: Small stones under about 6 mm have a high chance of passing on their own.

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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: Split bladder stones into two types.
Primary stones form in an otherwise normal bladder, often linked to

diet in childhood. Secondary stones form because something else is wrong, like blocked outflow, a catheter, or infection.

Step 2: Bring in the India specific fact.
Parts of India, particularly the north, are well known for endemic primary bladder stone disease in young children, tied to diets low in protein and phosphate. This makes primary stones a common, not rare, problem in Indian children. So the claim that they are rare is wrong.

Step 3: Check the remaining claims.
Small to medium bladder stones can usually be broken up and removed through the urethra with an endoscope, so transurethral removal is indeed possible. Most bladder stones have calcium in them, so they show up as radio opaque on X-ray. Because of this, a plain KUB film is often enough to confirm the stone and its location.

Step 4: Conclude.
The one wrong statement is that primary stones are rare in Indian children.

Primary stones rare in Indian children, is FALSE

Quick Tip: Endemic primary bladder stone disease in children is well documented in parts of India.

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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: Understanding the Concept:
A prostatic nodule on rectal exam in a man of 60 needs a test that can look at the nodule closely and also help take a sample from it.

Step 2: Key Formula or Approach:
The rule with prostate nodules is to use the probe that sits nearest the gland, since a closer probe gives a sharper picture and an easier path for a biopsy needle.

Step 3: Detailed Explanation:
Transrectal ultrasound (TRUS) puts a small probe into the rectum, right behind the prostate. This gives a close, detailed view of the gland and any nodule in it.
Because the probe sits so close, TRUS also lets the doctor pass a biopsy needle straight into the nodule under direct vision, combining imaging and tissue sampling in one visit.
Expressed prostatic secretion testing checks for white cells and bacteria, which is a test for prostatitis, not cancer.
A CT scan of the pelvis looks at organs from outside the body and cannot separate the zones of the prostate well enough to find a small nodule.
MRI gives a detailed picture too and is useful once a cancer is already

found, mainly to check how far it has spread, but it is not the standard first test for a fresh nodule, and it does not replace the biopsy step.

Step 4: Final Answer:
Transrectal ultrasound is the investigation of choice for a prostatic nodule, since it both shows the nodule and guides the needle biopsy.

Transrectal ultrasound (TRUS)

Quick Tip: Think about which test both images the prostate closely and can guide a biopsy needle.

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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:

This question tests knowledge of carcinoma penis risk factors and protective measures, and asks which listed statement is false.

1. **Circumcision any time before puberty is 100% protective**: This is the false claim. Only circumcision done at birth (neonatal) removes the foreskin before chronic smegma

exposure begins, giving close to full protection. Circumcision performed later in childhood, even before puberty, does not carry this same guarantee because some irritation may already have started.

2. **Erythroplasia of Queyrat is premalignant**: True. It is a velvety red plaque on the glans that represents carcinoma in situ and can progress to invasive cancer if untreated.
3. **Occurs in unhygienic conditions**: True. Poor hygiene allows smegma to build up under an intact foreskin, and this chronic irritant exposure raises the risk of carcinoma penis.
4. **Presents with inguinal node enlargement in 50% of the cases**: True. Because the penis drains to the inguinal nodes first, about half of patients have palpable inguinal nodes at diagnosis, though some of this is reactive rather than true metastasis.

The statement that does not hold up is the claim about circumcision at any time before puberty, since real protection needs circumcision at birth.

Let's summarize:

- Only neonatal circumcision gives near complete protection against carcinoma penis.
- Erythroplasia of Queyrat, poor hygiene, and inguinal node spread are all well established features of carcinoma penis.

So the false statement is that circumcision any time before puberty is 100% protective.

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: Understanding the Concept:
A multinodular goitre grows slowly over years, usually from low iodine intake or long term thyroid stimulating hormone drive. The question asks which cancer tends to develop inside such a gland.

Step 2: Key Formula or Approach:
Match each thyroid cancer to its classic trigger: papillary to radiation, medullary to MEN syndromes and C cells, anaplastic to very old age, and follicular to iodine deficient, longstanding multinodular glands.

Step 3: Detailed Explanation:
In regions with low dietary iodine, the thyroid enlarges over years into a multinodular gland as it works harder to trap iodine.
Within this chronic setting, follicular cells can undergo malignant change, giving rise to follicular carcinoma, which is well described as arising from longstanding multinodular goitre.
Papillary carcinoma, by contrast, is the most common thyroid cancer in general and is tied more to prior radiation exposure than to iodine deficient goitre.

Medullary carcinoma comes from C cells and often runs in families as part of MEN 2, unrelated to goitre duration.
Anaplastic carcinoma is rare and aggressive, seen mostly in elderly patients, and though it can follow a longstanding goitre, it is not the classic textbook pairing being tested here.

Step 4: Final Answer:
The cancer classically arising from a longstanding multinodular goitre is follicular carcinoma.

Follicular carcinoma

Quick Tip: Think of the thyroid cancer linked with iodine deficient, chronically enlarged multinodular glands.

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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: Understanding the Concept:
A pulsatile vein is a vein that visibly expands with each heartbeat. This only happens when high pressure arterial flow leaks into the low pressure venous system somewhere nearby.

Step 2: Key Formula or Approach:
Go through each option and ask whether it creates an abnormal path for arterial pressure to reach a superficial vein, or whether it just raises venous pressure generally.

Step 3: Detailed Explanation:
Klippel-Trenaunay syndrome is a mix of a birthmark, varicose or malformed veins, and limb overgrowth, and it can include small abnormal arteriovenous connections within the malformed tissue. These connections let arterial pulse pressure reach the superficial veins directly, making the varicosities pulsatile.
Deep vein thrombosis blocks a deep vein and causes a swollen, painful leg, with no arterial pressure involved, so there is no pulsation.
Right ventricular failure raises pressure in the whole venous system, giving a swollen leg and raised neck veins, but this is a steady raised pressure, not a beat to beat pulse in a surface vein.
Tricuspid regurgitation does transmit a pulse backward into the venous system, but this is specifically described at the groin as a pulsatile saphena varix, a separate named sign, rather than the general answer being asked for here.

Step 4: Final Answer:
Among the choices, Klippel-Trenaunay syndrome is the condition that may show pulsatile varicose veins because of its abnormal vessel connections.

Klippel-Trenaunay syndrome

Quick Tip: Look for the congenital vascular malformation syndrome with limb overgrowth and abnormal vessel connections.

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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: Understanding the Concept:
Local gigantism is one limb growing bigger than its pair. The question wants the cause that also comes with varicose veins in that same limb.

Step 2: Key Formula or Approach:
A single limb only overgrows if it alone gets extra blood supply or extra growth stimulus, not the whole body. So look for a cause that is local to one limb and also raises venous pressure enough to cause varicosities.

Step 3: Detailed Explanation:
An arteriovenous fistula is a short circuit between an artery and a vein. Blood skips the capillary bed and rushes into the vein at high pressure.
This constant extra flow feeds the bones and soft tissue of that limb, so growth centers get more nutrients and the limb grows faster and

bigger than the other side.
The same high flow overloads the superficial veins draining the limb, stretching them into varicose veins.
Deep vein thrombosis narrows or blocks flow instead of increasing it, so it swells the leg but never makes it grow bigger in a true overgrowth sense.
Acromegaly comes from too much growth hormone acting on the whole skeleton, so it enlarges hands, feet, and jaw together, not one limb alone.
Osteosarcoma is a cancer growing from bone itself, giving a firm local lump and pain, not a whole limb overgrowth with varicosities.

Step 4: Final Answer:
An arteriovenous fistula explains both the local gigantism and the varicose veins in the same limb.

Arteriovenous (AV) fistula

Quick Tip: Think of what abnormal vessel connection sends extra arterial flow into just one limb.

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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: Understanding the Concept:
Sclerotherapy works by injecting a chemical straight into a vein so its inner lining gets damaged, clots, and eventually turns into a fibrous cord that carries no blood. The question wants the one substance on the list that is not used this way.

Step 2: Key Formula or Approach:
Go through each agent and ask if its known clinical use is to injure a vein wall for closure, or if it is used for something else entirely.

Step 3: Detailed Explanation:
Alcohol, in its absolute form, is injected into veins precisely because it is strongly irritant to the vein lining, so it is a genuine sclerosant.
Crysolate is likewise used as an injectable sclerosing solution for varicose veins in clinical practice.
Polidocanol is a very commonly used modern sclerosant, sold for both small spider veins and larger varicose veins.
Acetic acid, on the other hand, has a completely different set of clinical uses. It is painted on the cervix during screening because it turns abnormal tissue white, and it is used as a wash for infected wounds or ears. It is not injected into veins to scar them shut.

Step 4: Final Answer:
Acetic acid is the odd one out, since it is not used as a sclerosing agent for varicose veins.

Acetic acid

Quick Tip: Think about which of these agents is used for cervical screening or as an antiseptic, not for closing off veins.

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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: Understanding the Question:
A young man has a blocked pelviureteric junction on the left with fever and infection, and we must find the one statement among four that is not correct.

Step 2: Key Formula or Approach:
Check each statement against two known facts of PUJ obstruction, the standard operation used, and the standard list of causes, where an intrinsic segment is far more common than an outside vessel.

Step 3: Detailed Explanation:
Dismembered pyeloplasty, described by Anderson and Hynes, removes the narrow segment and rejoins the pelvis to the ureter with a wide new opening. It remains the standard operation for PUJ obstruction, so this claim holds up.

Endoscopic pyelotomy cuts the narrow area from within using a scope, without removing it. This technique needs a clean field to heal properly, so it is avoided when the kidney is actively infected, exactly the situation described here. This claim also holds up.
Tuberculosis is a known cause of secondary PUJ narrowing, since healing of a tuberculous ulcer in the urinary tract leaves behind scar tissue that can block the junction. This claim holds up too.
That leaves the statement about an aberrant vessel being the most common cause. In fact, most cases of PUJ obstruction, especially the congenital ones, come from an intrinsic segment of the ureter near the pelvis that cannot contract properly and push urine along. A crossing vessel pressing on the outside of the junction is a real but less common cause. So calling it the most common cause does not match the facts.

Step 4: Final Answer:
The statement that is not correct is that an aberrant vessel is the most common cause of PUJ obstruction, because an intrinsic aperistaltic segment is 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.

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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: Identify the condition.
A spindle shaped (fusiform) bulge of the abdominal aorta with pain in a 60 year old with high blood pressure is an abdominal aortic aneurysm.

Step 2: Think about what damages the aortic wall over decades.
The aortic wall has three layers, and the middle layer gives it strength. Fatty deposits building up inside the wall over many years thin and weaken this middle layer, letting the vessel stretch outward evenly, which produces a fusiform shape. This process is atherosclerosis, and it is worsened by long standing hypertension.

Step 3: Rule out the connective tissue causes.
Marfan's syndrome and cystic medial necrosis weaken the aorta from birth through a faulty connective tissue protein (fibrillin), so the aneurysm shows up early in life and almost always in the chest portion of the aorta, not here.

Step 4: Rule out infection.
Syphilis was once a well known cause of aortic aneurysm, but it attacks the small vessels feeding the aortic wall in the chest, giving a saccular (pouch like) bulge in the ascending aorta, a very different

picture from this patient.

Step 5: Match age, site and shape to the right cause.
An elderly, hypertensive patient with a fusiform swelling of the abdominal aorta fits a degenerative, fat plaque driven wall change.

Atherosclerosis

Quick Tip: Fusiform abdominal aortic aneurysm in an elderly hypertensive patient points to a degenerative wall change from fat plaque build up.

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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: Start from the normal pathway.
Catecholamine synthesis runs tyrosine to dopa to dopamine to norepinephrine to epinephrine. The last step, turning norepinephrine into epinephrine, needs the enzyme PNMT, found mainly in normal adrenal medulla cells.

Step 2: See what changes inside the tumor.

A pheochromocytoma is a chromaffin cell tumor, but its cells frequently have reduced PNMT activity compared with normal tissue. This means the pathway often stalls one step earlier, so norepinephrine piles up instead of being fully converted.

Step 3: Connect this to the overall secretion pattern.
Across the whole group of pheochromocytoma patients, norepinephrine ends up being the catecholamine found in the largest amount, and it drives the sustained high blood pressure that is typical of this tumor.

Step 4: Place the other options.
Epinephrine is still secreted and gives the sudden attack symptoms, but it is not the dominant hormone overall. Dopamine only rises when the tumor is malignant or sits outside the adrenal gland. Serotonin belongs to a totally different tumor, the carcinoid, and has nothing to do with pheochromocytoma.

Norepinephrine

Quick Tip: Pheochromocytoma cells often skip the last conversion step, so this catecholamine builds up and dominates.

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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: Picture how infection actually travels on a ward.
A nurse or doctor moves from one bed to the next, touching dressings, wounds, and equipment along the way. If hands are not cleaned between these contacts, germs from one patient's wound ride along to the next patient.

Step 2: Match the measure to this route.
Washing hands right before touching a patient, and again between each patient, cuts this chain at its weakest link. This is why hand hygiene is called the single most powerful infection control tool in any ward.

Step 3: Test the other choices against the same route.
Fumigating the air does not clean the hands that actually touch the wound, and has no proven benefit for surgical infection rates. Mopping the floor deals with a surface that is rarely, if ever, in direct contact with an open wound. Giving vancomycin to everyone treats a symptom that has not even appeared yet, wastes a valuable antibiotic, and encourages resistant germs to develop, none of which stops hand to hand spread.

Step 4: Conclude.
Only hand washing directly interrupts the real pathway of spread.

Hand washing before and between patient contact

Quick Tip: Think about what actually travels from one wound dressing to the next patient.

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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: Frame the surgical problem.
Before surgery, a solitary nodule is of uncertain nature. The operation must remove it safely and also let the pathologist tell whether it is benign or malignant.

Step 2: Work out the operation that meets both needs.
Taking out the entire lobe holding the nodule, plus the isthmus, gives a full specimen with clean margins and settles the diagnosis in one step. This procedure is hemithyroidectomy, also called thyroid

lobectomy.

Step 3: Plan the next step if needed.
Once the pathology comes back, a benign result means no further surgery is needed. A malignant result can be followed by completing the thyroidectomy on the other side in a second planned operation.

Step 4: See why the bigger operations are premature.
Total and subtotal thyroidectomy both act on the whole gland right away, which is unnecessary when only one side has a defined lump of unknown nature, and both add avoidable risk to the parathyroids and the nerves supplying the voice box.

Step 5: See why enucleation is unsafe.
Peeling out just the nodule without a rim of normal tissue around it does not guarantee a clean margin, so it is not used in modern thyroid surgery.

Hemithyroidectomy

Quick Tip: Pick the operation that removes the whole nodule with a margin and also gives a full tissue diagnosis.

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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: Group the pelvic exit routes.
Structures leaving the pelvis take one of two main doors, either the greater sciatic foramen or the obturator canal, and these two doors are on opposite sides of the pelvis.

Step 2: Follow the greater sciatic foramen group.
The pudendal nerve, the internal pudendal vessels, and the nerve to obturator internus all exit through the greater sciatic foramen, curl around the ischial spine and sacrospinous ligament, and go back in through the lesser sciatic foramen to reach the ischioanal fossa. This detour around the spine is what the question is pointing at.

Step 3: Follow the obturator nerve.
The obturator nerve takes the other door. It runs down along the side wall of the pelvis and slips out through the obturator canal, near the top of the obturator foramen, at the front of the pelvis, well away from the ischial spine.

Step 4: Spot the mismatch.
Since three of the four structures share the sciatic spine detour and the obturator nerve does not, the obturator nerve is the exception.

Obturator nerve

Quick Tip: Three of these structures re-enter the perineum through the lesser sciatic foramen; one leaves the pelvis through the obturator canal instead.

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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: Picture the operative field of a hernia repair.
Inguinal hernia surgery works directly on the inguinal canal, where the spermatic cord and a nearby sensory nerve both run close to the sutures and mesh used to repair the defect.

Step 2: Identify the nerve that sits in this exact path.
The ilioinguinal nerve travels through this same canal and comes out at the superficial inguinal ring, making it one of the nerves most often caught, stretched, or trapped in scar tissue during the operation.

Step 3: Connect the nerve to its sensory territory.
It supplies the skin at the root of the penis and upper scrotum, so

when it is injured, the patient feels numbness or tingling exactly in that region, matching the symptom in the question.

Step 4: Rule out nerves with a different territory or path.
The pudendal nerve reaches the genitals by a deeper perineal route, away from the hernia field. The genitofemoral nerve's genital branch mainly drives the cremaster muscle rather than skin sensation at this site. The iliohypogastric nerve covers the suprapubic skin, higher up than the scrotum and penis.

Step 5: Conclude.
Only the ilioinguinal nerve fits both the location of injury and the area of numbness.

Ilioinguinal nerve

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.

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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: Go through the anatomy facts first.
The liver's right lobe presses the right kidney slightly lower than the left, so that fact holds. The duodenum's second part lies right against the anteromedial side of the right kidney near its hilum, so that fact holds too. Because the inferior vena cava runs closer to the right side, the right renal vein is short while the left renal vein has to stretch across the midline to reach it, so that fact also holds.

Step 2: Bring in the surgical fact about transplantation.
When a surgeon harvests a kidney for transplant, a longer renal vein gives more room to work with while joining it to the recipient's vessels. The left kidney has this longer vein, which is exactly why it is generally chosen over the right kidney.

Step 3: Compare this to the given statement.
The statement in question claims the right kidney is preferred over the left for transplant, which is the reverse of real practice.

Step 4: Conclude.
Only the transplant preference statement is false.

Right kidney preferred to left for transplantation, is NOT correct

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: Sort the enzymes by pathway.
Three of the four enzymes, galactokinase, the galactose-1-phosphate uridyltransferase, and galactose-4-epimerase, all work one after another to turn galactose into a form the body can use as glucose. Lactase stands apart; it lives in the gut wall and only cuts lactose into glucose and galactose before absorption.

Step 2: Follow what builds up in each block.
When any of the three galactose-handling enzymes is missing, galactose and galactose-1-phosphate cannot move forward properly and build up in the blood and tissues. Part of that extra galactose gets reduced to galactitol inside the lens, and galactitol pulls water in by osmosis, swelling the lens fibers and clouding them into a cataract. Ongoing buildup also injures the brain, giving mental retardation if untreated.

Step 3: Check lactase separately.
If lactase is missing, lactose simply cannot be split at all, so galactose never gets absorbed into the bloodstream in the first place. There is nothing to build up in the lens or brain from a lactase block.

Symptoms instead stay confined to the gut, as cramping, bloating, and loose stools after drinking milk.

Step 4: State the answer.
Because lactase acts before galactose absorption and the other three act after it, only a lactase deficiency spares the child from lens opacities and mental retardation.
Lactase

Quick Tip: Only lactase sits outside the galactose metabolism pathway; the other three enzymes all handle galactose itself.

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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: Remember what fiber is.
Dietary fiber is plant carbohydrate that human digestive enzymes cannot break down, so it travels intact from the small intestine into the colon.

Step 2: Bring in the colonic bacteria.

Once fiber reaches the colon, resident bacteria ferment it in a low-oxygen environment. This fermentation splits fiber into short-chain fatty acids, chiefly acetate, propionate, and butyrate.

Step 3: See why butyrate stands out.
Among these, butyrate is absorbed straight by the colon lining cells and burned as their preferred energy source, ahead of glucose. This is why a fiber-rich diet is often linked to a healthier colon lining.

Step 4: Eliminate the distractors.
Glycerol comes from breaking down fats, sucrose is a dietary sugar absorbed earlier in the gut, and free radicals are oxidative byproducts, none of which is the direct output of bacterial fiber fermentation.

Step 5: Conclude.
Butyrates

Quick Tip: Colonic bacteria ferment fiber into short-chain fatty acids; butyrate feeds the colon cells.

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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: Place the enzyme in context.
Glucose-6-phosphatase deficiency, Von Gierke disease, blocks the final step of turning stored liver glucose-6-phosphate into free blood glucose.

Step 2: Ask where the trapped glucose-6-phosphate goes.
With the exit blocked, glucose-6-phosphate backs up inside the liver cell and gets diverted into other pathways that can still use it, including the pentose phosphate pathway.

Step 3: Connect that pathway to purines.
The pentose phosphate pathway makes ribose-5-phosphate, which feeds directly into making phosphoribosyl pyrophosphate (PRPP). PRPP is the building block the body uses to make new purine bases. More PRPP means more purine synthesis, and purines are broken down to uric acid.

Step 4: Add the clearance problem.
The liver also leans on glycolysis harder since it cannot export glucose normally, which raises lactate. Lactate and uric acid share the same kidney transporter, so high lactate blocks uric acid from being excreted, adding to the buildup.

Step 5: Compare with the other choices.
Sorbitol buildup belongs to a different disorder pattern, seen in diabetes and galactosemia, extra glycerol synthesis is not part of this mechanism, and a slower Krebs cycle does not explain purine overproduction. None of these fit as well as the pentose phosphate route.

Step 6: State the answer.

Increased synthesis of pentoses

The original answer key left this question blank; the choice above follows the accepted biochemical mechanism of high uric acid in Von Gierke disease.

Quick Tip: Trapped glucose-6-phosphate is shunted into the pentose phosphate pathway, raising PRPP and purine and uric acid production.

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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: Map out the chain.

The pathway order is $S1 \rightarrow S2 \rightarrow S3 \rightarrow S4 \rightarrow P1 \rightarrow P2$, run by enzymes *EA* (first step), *EB* (second step), and *EC* (third step). *EA* is turned up by positive feedback, *EB* is turned down by negative feedback.

Step 2: Remove EC and watch S3.
EC normally clears *S*3 forward into *S*4. Take *EC* away and *S*3 has no exit, so it starts to build.

Step 3: Let the built-up S3 talk to EB.
Because *EB* answers to negative feedback, and *S*3 is exactly the molecule *EB* makes, the rising *S*3 signals back and switches *EB* down. This closes the door between *S*2 and *S*3, so *S*3 stops rising further and *S*2 stops draining away.

Step 4: Check the supply side.
EA has no such brake, in fact it is under positive feedback, so it keeps converting *S*1 into *S*2 without slowing.

Step 5: See where the traffic jam forms.
*S*2 keeps arriving from *EA* but cannot leave through the now silenced *EB*. That mismatch, still coming in, blocked from going out, is what makes *S*2 the molecule that piles up, not *S*1, *P*1, or *P*2.

Step 6: Conclude.
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.