

General Instructions

- (i) This booklet contains 200 questions, each provided with a complete, step-by-step solution.
- (ii) It comprises 199 single-correct multiple-choice questions and 1 multiple-correct question.
- (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. Which activity will be difficult to perform for a patient with an anterior cruciate deficient joint?

- (A) Walk downhill
- (B) Walk uphill
- (C) Sit cross leg
- (D) Getting up from sitting

Correct Answer: (A) Walk downhill

SOLUTION:

Step 1: Recall the job of the ACL.

The ACL keeps the shin bone from sliding forward on the thigh bone. Without it, that forward slide is unchecked.

Step 2: Think about the quadriceps and the tibia together.

Any time the quadriceps fires hard while the knee is bent, it pulls the tibia forward through the kneecap tendon. Walking downhill is exactly

this situation, the knee stays bent and the quadriceps works eccentrically to slow the body down each step.

Step 3: See why that forward pull matters here.

In a normal knee the ACL absorbs this pull and keeps the joint steady. In an ACL deficient knee there is no brake, so the tibia jogs forward and the patient feels the knee buckle or give way.

Step 4: Compare with the other choices.

Uphill walking loads the hip more than the quadriceps. Sitting cross legged is a slow, low force movement. Standing up from sitting is controlled and does not carry the same sudden shear. None of these stress the ACL the way downhill walking does.

Step 5: State the answer.

Walk downhill is the activity that becomes difficult after an ACL tear.

Walk downhill

Quick Tip: The ACL resists forward shift of the tibia; activities needing a strong quadriceps pull in a bent knee, like walking downhill, are hardest.

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2. The treatment of choice for atticointral variety of CSOM is

- (A) Mastoidectomy
- (B) Medical management
- (C) Underlay myringoplasty
- (D) Insertion of ventilation tube

Correct Answer: (A) Mastoidectomy

SOLUTION:

Step 1: Split CSOM into two patterns.

One pattern, tubotympanic disease, is a safe type with a central hole in the eardrum. The other, atticotympanic disease, is unsafe and almost always carries cholesteatoma, skin growing where it should not, eating into bone.

Step 2: Ask what cholesteatoma needs.

Cholesteatoma keeps enlarging and eroding whatever bone is nearby, the ossicles, the facial nerve canal, even the inner ear. No drop or tablet dissolves this skin mass.

Step 3: Match the disease to the fix.

Since the problem is a physical, expanding mass, the fix has to be physical too, opening the mastoid and clearing every trace of the disease. That operation is mastoidectomy.

Step 4: Cross off the rest.

Medical treatment only quiets infection for a while. Underlay myringoplasty fixes a plain central perforation, which belongs to the safe type, not this one. A ventilation tube treats fluid behind an intact eardrum, a different problem altogether.

Step 5: Conclude.

Mastoidectomy clears the cholesteatoma and stops the bone destruction.

Mastoidectomy

Quick Tip: Atticoantral CSOM usually means cholesteatoma, and only surgery (mastoidectomy) can clear it.

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3. The most appropriate management of antrochoanal polyp in children is

- (A) Caldwell Luc operation
- (B) Intranasal polypectomy
- (C) Corticosteroids
- (D) Wait and watch

Correct Answer: (B) Intranasal polypectomy

SOLUTION:

Step 1: Picture the polyp.

An antrochoanal polyp begins inside the maxillary sinus and sends a long stalk out through the sinus opening, across the nose, sometimes down to the throat opening.

Step 2: Note the trap in treating it.

If a surgeon removes only the part seen inside the nose and leaves the base sitting in the sinus, the polyp regrows quickly. Full removal, base and all, is what stops recurrence.

Step 3: Pick the route that suits a child's face.

Going in through the cheek bone, as in the Caldwell-Luc operation, risks the tooth buds and growth centres of a child's skull. Going in through the nostril with an endoscope avoids all of that while still reaching the sinus base of the polyp.

Step 4: Dismiss the weaker options.

Steroids shrink swelling but cannot remove this fleshy, stalked mass for good. Simply watching lets the polyp keep growing and block the airway further. The Caldwell-Luc route works but is not the preferred first step in a child.

Step 5: State the answer.

Intranasal polypectomy, done endoscopically with removal of the antral base, is the right choice in children.

Intranasal polypectomy

Quick Tip: In children, endoscopic intranasal removal avoids the facial growth risks of the Caldwell-Luc approach.

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4. The cough response caused while cleaning the ear canal is mediated by stimulation of

- (A) The V cranial nerve
- (B) Innervation of external ear canal by C1, C2
- (C) The X cranial nerve
- (D) Branches of VII cranial nerve

Correct Answer: (C) The X cranial nerve

SOLUTION:

Step 1: List who supplies the ear canal.

Several nerves share the skin of the ear canal: the trigeminal nerve up front, the vagus nerve mostly at the back and floor, and small patches

from the facial and glossopharyngeal nerves.

Step 2: Trace where the vagus nerve goes next.

The same vagus nerve carries sensation from the throat, voice box, and airway, the exact spots that set off a cough when irritated. Its ear branch, called Arnold's nerve, taps into this same wiring.

Step 3: Put the two facts together.

So when a cotton bud or water touches the vagus-supplied skin in the canal, the brain reads it as if the airway were being irritated, and a cough follows. This is Arnold's reflex, sometimes called the ear-cough reflex.

Step 4: Check the distractors.

The trigeminal nerve and the small facial nerve twigs feel the canal too but do not connect to the cough pathway. C1 to C2 fibres via the great auricular nerve serve the outer ear skin, not the canal's cough trigger.

Step 5: Give the answer.

The X cranial nerve (vagus), through Arnold's nerve, is behind this cough response.

The X cranial nerve

Quick Tip: The ear canal floor is supplied by the vagus nerve (Arnold's nerve), linking ear stimulation to the cough reflex.



5. A 38 year old gentleman reports of decreased hearing in the right ear for the past 2 years. On testing with a 512 Hz tuning fork, the Rinne's test (without masking) is negative on the right ear and positive on the left ear, with Weber's test perceived as louder in the left ear. The patient most likely has

- (A) Right conductive hearing loss
- (B) Right sensorineural hearing loss
- (C) Left sensorineural hearing loss
- (D) Left conductive hearing loss

Correct Answer: (B) Right sensorineural hearing loss

SOLUTION:

Step 1: Read the Rinne result at face value.

On the right side, bone conduction beats air conduction, a negative Rinne. Taken alone, this usually points to a blocked middle ear on that side.

Step 2: Read the Weber result.

The sound is louder in the left ear during Weber's test. In a purely conductive problem, the ear with the block should sound louder in Weber, not the other ear. Here the louder side, left, is the opposite of the negative-Rinne side, right, so the two tests seem to disagree.

Step 3: Recall why a very damaged cochlea can trick the Rinne test.

If the right cochlea is badly damaged, a fork placed on the right mastoid still sends vibration through the skull bone to the left cochlea, which is healthy and picks it up well. The patient reports hearing the bone-conducted sound in that right test, but it is really the left ear doing the hearing. This makes the right Rinne look falsely negative, as

though bone beats air, even with no true conductive block on the right.

Step 4: Resolve the disagreement.

Once we know the Rinne can be fooled this way, the Weber test becomes the deciding clue. Weber lateralizing to the left, the ear with normal hearing, is exactly what happens in sensorineural loss on the right, since the poorer cochlea cannot compete for the sound.

Step 5: Match against each choice.

A true right conductive loss needs Weber to go right, not left, so that is excluded. A left-sided problem, conductive or sensorineural, would need an abnormal left Rinne, but the left Rinne here is normal (positive), so the left ear is fine.

Step 6: Conclude.

Right sensorineural hearing loss explains a falsely negative right Rinne together with a Weber that lateralizes to the healthy left ear.

Right sensorineural hearing loss

Quick Tip: A falsely negative Rinne from severe unilateral sensorineural loss still shows a Weber that lateralizes to the healthy ear.

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6. Which of the following is not correct for ethmoidal polyp?

- (A) Allergy is an etiological factor
- (B) Occur in first decade of life
- (C) Are bilateral
- (D) Are often associated with bronchial asthma

Correct Answer: (B) Occur in first decade of life

SOLUTION:

Step 1: Set the usual picture of ethmoidal polyps.

These polyps grow from the ethmoid sinus lining, usually driven by allergy or ongoing inflammation, and are mostly a disease of adults.

Step 2: Test the allergy claim.

Allergy is a well known trigger for this kind of polyp, so that statement holds up and is not the odd one out.

Step 3: Test the age claim.

Seeing this kind of polyp in a child under ten years old is unusual, so much so that doctors think of cystic fibrosis first in that age group. So saying they occur in the first decade of life does not hold up.

Step 4: Test the laterality claim.

Unlike the single, one-sided antrochoanal polyp, ethmoidal polyps usually appear on both sides at once, so calling them bilateral is correct.

Step 5: Test the asthma claim.

They often go along with bronchial asthma and aspirin sensitivity as part of a well known triad, so that statement is also correct.

Step 6: Pick the false one.

Only the age statement fails to match reality.

Occur in first decade of life

Quick Tip: Ethmoidal polyps are usually bilateral, allergy related, asthma associated, and seen in adults, not young children.

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7. Foetal hydronephrosis is diagnosed in the mother at 34 weeks of gestation. The amniotic fluid is normal. Which of the following is the most appropriate management?

- (A) Fetal intervention to decompress hydronephrotic kidney
- (B) Premature termination of pregnancy followed by pyeloplasty
- (C) Delivery at term followed by radiological examination
- (D) Delivery at term followed by pyeloplasty

Correct Answer: (C) Delivery at term followed by radiological examination

SOLUTION:

Step 1: Read the clue given by the amniotic fluid.

Amniotic fluid late in pregnancy comes mostly from fetal urine. Normal fluid at 34 weeks means both kidneys, working together, are still producing urine well, so there is no severe obstruction putting the baby at immediate risk.

Step 2: Ask if the baby needs to be treated before birth.

Procedures done inside the womb to relieve a blocked kidney are kept

for babies with low fluid and a failing kidney, since that combination threatens lung development and kidney survival. That danger sign is missing here, so there is no case for fetal surgery or for delivering the baby early.

Step 3: Plan care after this point.

Let the pregnancy run its full course to term, avoiding the extra risks of prematurity. Once the baby is born, image the kidneys properly, an ultrasound scan first, sometimes repeated after a week or so because a newborn's mild dehydration can hide the true degree of swelling, and add further studies such as a voiding study or a renogram if the ultrasound looks worrying. Surgery is only planned once these tests show a real, obstructing problem.

Step 4: Weigh the other choices.

Decompressing the kidney before birth is overtreatment when the fluid is normal. Ending the pregnancy early just to operate sooner trades a mild, unconfirmed kidney finding for the real complications of prematurity. Operating right after term birth, without imaging first, risks operating on a kidney that may not even need surgery.

Step 5: Give the answer.

Delivery at term followed by radiological examination balances safety for the baby with proper diagnosis before any operation.

Delivery at term followed by radiological examination

Quick Tip: Normal amniotic fluid means the kidneys are still functioning; no need for early delivery or fetal intervention.



8. A 2 year old boy has Vitamin D refractory Rickets. Investigations show serum calcium 9 mg/dl, phosphate 2.4 mg/dl, alkaline phosphatase 1040 IU. Parathyroid hormone level and bicarbonate levels are normal. The most probable diagnosis is

- (A) Distal Renal Tubular Acidosis
- (B) Hypophosphatemic Rickets
- (C) Vitamin D Dependent Rickets
- (D) Proximal Renal Tubular Acidosis

Correct Answer: (B) Hypophosphatemic Rickets

SOLUTION:

Step 1: List what each rachitic cause should do to the labs.

Distal RTA: low bicarbonate (acidosis), variable calcium and phosphate.

Proximal RTA: low bicarbonate (acidosis), often with glucosuria and other Fanconi features.

Vitamin D dependent rickets: low calcium, high PTH (secondary hyperparathyroidism), variable phosphate.

Hypophosphatemic rickets: normal calcium, low phosphate, normal PTH, normal bicarbonate.

Step 2: Compare this list against the given child.

The child has calcium 9 mg/dl (normal), phosphate 2.4 mg/dl (low), alkaline phosphatase 1040 IU (high), PTH normal, bicarbonate normal.

Step 3: Match line by line.

Bicarbonate being normal removes both RTA types, since acidosis is their hallmark. Calcium being normal, not low, and PTH being

normal, not raised, removes vitamin D dependent rickets, since that condition runs on hypocalcemia driving up PTH.

Step 4: Confirm the fit.

Only the hypophosphatemic rickets row matches every value: normal calcium, low phosphate, normal PTH, normal bicarbonate, with a raised alkaline phosphatase from active bone disease.

Step 5: State the answer.

The most probable diagnosis is Hypophosphatemic Rickets.

Hypophosphatemic Rickets

Quick Tip: Normal bicarbonate rules out RTA; normal calcium and PTH rule out vitamin D dependent rickets; low phosphate with normal calcium points to hypophosphatemic rickets.

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9. Neurological complications of meningitis include all of the following except

- (A) Seizures
- (B) Increased Intracranial Pressure
- (C) Cerebral Hematoma
- (D) Subdural Effusions

Correct Answer: (C) Cerebral Hematoma

SOLUTION:

Step 1: Think about what meningitis physically does.

Meningitis inflames the coverings of the brain and the fluid around it. This inflammation can irritate the cortex, block CSF drainage, and injure small bridging veins.

Step 2: Trace each of those effects to an outcome.

Cortical irritation gives seizures. Blocked CSF drainage plus swelling gives raised intracranial pressure. Injury to bridging veins in infants gives subdural effusions. All three of these follow directly from how meningitis behaves.

Step 3: Ask whether bleeding into the brain fits this picture.

A hematoma inside brain tissue usually needs a torn vessel from trauma or an aneurysm, not simple meningeal inflammation. Meningitis does not typically tear a vessel to cause a hematoma.

Step 4: Pick the exception.

Since seizures, raised ICP, and subdural effusions are all genuine complications, and cerebral hematoma is not, cerebral hematoma is the odd one out.

Cerebral Hematoma

Quick Tip: Seizures, raised ICP and subdural effusions are classic meningitis complications; a brain parenchymal hematoma is not.

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10. The gold standard for definitive diagnosis of extrahepatic biliary atresia is

- (A) Per-operative Cholangiography
- (B) Hepatobiliary Scintigraphy
- (C) Alkaline Phosphatase Level
- (D) Liver Biopsy

Correct Answer: (A) Per-operative Cholangiography

SOLUTION:

Step 1: Rank the four tests by how directly they answer the question.

Alkaline phosphatase only tells us that some kind of cholestasis is present, nothing more, so it is the weakest test here.

Step 2: Place scintigraphy next.

The HIDA scan is a functional test. It picks up the tracer well in the liver but fails to show it in the gut when the duct is blocked. The catch is that severe hepatocellular disease can also block tracer excretion without any true duct blockage, so this test screens rather than confirms.

Step 3: Place liver biopsy next.

A biopsy examines the tissue directly and picks up features like bile duct proliferation and portal tract fibrosis. It is highly accurate, close to 90 to 95 percent, but it is still an indirect, pattern based read, not a direct look at the duct itself.

Step 4: Place per-operative cholangiography at the top.

This test injects dye straight into the biliary system during surgery and takes a live X-ray picture. If dye reaches the duodenum, the ducts are open. If it does not, the diagnosis of biliary atresia is confirmed on the spot. This is a direct anatomical answer, not an indirect inference, so

it is the gold standard.

Step 5: State the answer.

Even though the original key names scintigraphy, direct visualization by per-operative cholangiography is the accepted gold standard in current practice.

Per-operative Cholangiography

Quick Tip: Per-operative cholangiography directly visualizes the biliary tree and confirms the block; scintigraphy only screens.

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11. The length of feeding tube to be inserted for transpyloric feeding is measured from the tip of the

- (A) Nose to Umbilicus
- (B) Ear Lobe to Umbilicus
- (C) Nose to Knee Joint
- (D) Ear Lobe to Knee Joint

Correct Answer: (A) Nose to Umbilicus

SOLUTION:

Step 1: Compare the two common tube-sizing rules.

NEX (nose to ear to xiphoid) sizes a tube meant to stop in the stomach. NEMU (nose to ear to mid-umbilicus) sizes a tube meant to pass beyond the stomach into the small bowel, which is what transpyloric feeding needs.

Step 2: Match the question to the right rule.

Since the question asks about transpyloric feeding, meaning the tube tip sits past the pylorus, the longer NEMU measurement applies, not the shorter gastric one.

Step 3: Read off the landmarks in NEMU.

The measurement starts at the nose, runs to the ear, then down to the umbilicus. In short form this is described as nose to umbilicus.

Step 4: Eliminate the knee-based options.

The knee joint is never used as a landmark in feeding tube sizing in infants, so any option built around it can be dropped right away.

Step 5: Conclude.

Nose to Umbilicus

Quick Tip: Transpyloric tubes use the longer NEMU rule: nose to ear to mid-umbilicus.

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12. Which layer of the epidermis is underdeveloped in Very Low Birth Weight (VLBW) infants in the initial 7 days?

- (A) Stratum Germinativum
- (B) Stratum Granulosum
- (C) Stratum Lucidum
- (D) Stratum Corneum

Correct Answer: (D) Stratum Corneum

SOLUTION:

Step 1: Picture the epidermis as a stack of layers.

The stack, from the bottom up, runs basal layer, spinous layer, granular layer, and finally the tough outer corneum. This outer layer is what actually meets the outside world.

Step 2: Ask which layer forms last before birth.

Skin barrier development is one of the last things to finish in fetal life, concentrated in the final weeks before term. A baby born early misses much of this final step.

Step 3: Connect this to VLBW infants.

VLBW infants are almost always premature, so they are born before the outer corneum layer has thickened and keratinized properly. In the first 7 days this layer is still catching up.

Step 4: Use the clinical clue to confirm.

Preterm babies are known for high fluid loss through the skin and a greater chance of skin injury and infection in the first week. Both problems trace back to a weak outer barrier, which is the stratum corneum, not the deeper layers.

Step 5: Conclude.

Stratum Corneum

Quick Tip: The barrier stratum corneum matures late in fetal life, so it is thin in VLBW infants right after birth.



13. In pediatric advanced life support, intraosseous access for drug and fluid administration is recommended for a pediatric age of

- (A) Less than 1 year
- (B) Less than 5 years
- (C) Less than 6 years
- (D) Any age

Correct Answer: (D) Any age

SOLUTION:

Step 1: Start from why IO access exists.

IO access is a backup plan. When a sick or injured child needs urgent drugs or fluids and a vein cannot be found fast, a needle goes into the bone marrow instead, since the marrow connects to the venous system.

Step 2: Ask what decides whether IO can be used, age or urgency.

Modern teaching frames the decision around time and difficulty of finding a vein, not the child's age. If two tries or about 90 seconds pass without IV access in an emergency, IO access should be used.

Step 3: Check the age question directly.

Because the decision is time based rather than age based, IO access is considered appropriate across the whole age range, from infants to older children and even adults, using an insertion site suited to the size of the patient.

Step 4: Compare with the age-limited choices.

Options that cap IO use at under 1, under 5, or under 6 years all impose a ceiling that current resuscitation science does not support.

Step 5: Conclude.

Present-day PALS teaching supports IO access at any age when IV access is not quick to get.

Any age

Quick Tip: Current PALS guidance drops the old age ceiling; IO access is used at any age when IV access is not quick.

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14. A 3 year old boy is brought to the casualty by his mother with progressive shortness of breath for 1 day. The child has a history of bronchial asthma. On examination, the child is gasping and unresponsive. What will you do first?

- (A) Intubate
- (B) Administer 100% Oxygen by Mask
- (C) Ventilate with Bag and Mask
- (D) Administer Nebulized Salbutamol

Correct Answer: (C) Ventilate with Bag and Mask

SOLUTION:

Step 1: Translate the exam findings into a working diagnosis.

Shortness of breath worsening over a day in a known asthmatic, now gasping and unresponsive, describes a child sliding from severe asthma into respiratory failure and arrest.

Step 2: Use the standard airway, breathing, circulation approach.

After confirming the airway is open, the very next step for a child who is not breathing well enough is to support breathing directly. This means giving breaths for the child, not waiting to see if extra oxygen alone will be inhaled.

Step 3: Test each option against "is the child breathing well enough on their own".

The child is gasping, so the answer is no. Oxygen by mask assumes the child pulls in the gas, which will not happen reliably here. Bag and mask ventilation forces air in regardless of the child's own effort, so it directly answers the problem. Nebulized salbutamol treats the airway spasm but needs the child to breathe it in, which will not happen well during gasping. Intubation is the next step in sequence, done once the child is being oxygenated and ventilated and the team is ready.

Step 4: Rank the actions by urgency.

Bag and mask ventilation buys time and supports the child right away with equipment already at the bedside. Intubation and nebulized therapy follow once ventilation is underway.

Step 5: Conclude.

The first action for a gasping, unresponsive asthmatic child is to ventilate with bag and mask.

Ventilate with Bag and Mask

Quick Tip: A gasping, unresponsive child cannot breathe in oxygen on their own, so assisted ventilation with bag and mask comes first.



15. A 5 year old child presents with confusion, increased salivation, lacrimation, fasciculations, miosis, tachycardia and hypertension. Which one of the following poisons can cause these manifestations?

- (A) Opium
- (B) Organophosphate insecticide
- (C) Dhatura
- (D) Organochlorine pesticide

Correct Answer: (B) Organophosphate insecticide

SOLUTION:

The child shows small pupils, heavy salivation and tearing, muscle twitching, a fast heart rate and high blood pressure, along with confusion. This is a cholinergic overload picture, so acetylcholine is not being cleared properly at the nerve endings. Let's go through each poison.

1. **Opium:** Opium and other opioids cause sleepiness, slow breathing and pinpoint pupils, but they do not cause salivation, sweating or muscle fasciculations, so this does not explain the picture.
2. **Organophosphate insecticide:** These compounds stop the enzyme that normally breaks down acetylcholine. Acetylcholine then piles up everywhere, giving both the wet muscarinic signs (salivation, lacrimation, miosis) and the nicotinic signs (fasciculations, fast heart rate, high blood pressure), plus confusion from the brain effect. Every symptom in the question fits.
3. **Dhatura:** Dhatura blocks acetylcholine receptors instead of raising acetylcholine, so it produces a dry mouth, hot flushed

skin, dilated pupils and a fast heart rate without sweating or salivation, the reverse pattern of what is described.

4. **Organochlorine pesticide:** These act on nerve sodium channels and mainly cause tremor, irritability and seizures, without the salivation, miosis and fasciculation combination seen here.

Only the organophosphate insecticide produces this exact combined muscarinic and nicotinic overload with confusion, so it is the poison responsible.

Let's summarize:

- Muscarinic signs: salivation, lacrimation, miosis, slow gut and slow heart rate.
- Nicotinic signs: fasciculations, muscle weakness, and a fast heart rate with high blood pressure.

The correct answer is organophosphate insecticide poisoning.

Quick Tip: Think of the mixed muscarinic (SLUD, miosis) plus nicotinic (fasciculations, tachycardia) picture seen with cholinesterase inhibitor poisoning.

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16. The drug of choice for thoracic actinomycosis is

- (A) Amphotericin B
- (B) Penicillin
- (C) Co trimoxazole
- (D) Itraconazole

Correct Answer: (B) Penicillin

SOLUTION:

Actinomycosis sounds fungal because of its name, but *Actinomyces israelii* is actually a branching, filament forming bacterium. Treatment choice depends on which drug reliably kills this organism and can reach deep, scarred chest tissue.

1. **Amphotericin B:** This is a strong antifungal drug used for infections like aspergillosis or candidiasis. Since actinomycosis is bacterial, this drug has no target here.
2. **Penicillin:** *Actinomyces* species remain highly sensitive to penicillin, and high dose intravenous penicillin continued for weeks to months is what actually clears the deep sinus tracts and fibrotic lung or chest wall disease seen in thoracic actinomycosis.
3. **Co trimoxazole:** This is kept as a backup choice for patients who cannot tolerate penicillin, not as the primary drug.
4. **Itraconazole:** Like amphotericin B, this is an antifungal agent and does not act on this bacterial infection.

Because *Actinomyces* is a bacterium and stays sensitive to it, penicillin remains the drug of choice, usually given in high doses for a long duration to penetrate the dense, fibrous thoracic lesions.

Let's summarize:

- Actinomycosis is bacterial, so antifungal drugs like amphotericin B and itraconazole do not work.
- High dose, prolonged penicillin is the standard first line treatment.

The correct answer is penicillin.

Quick Tip: *Actinomyces israelii* is a bacterium, not a fungus, and it is highly sensitive to penicillin given in high dose for a long course.

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17. All of the following statements regarding malignant potential of colorectal polyps are true EXCEPT

- (A) Polyps of familial polyposis coli could undergo malignant change
- (B) Pseudopolyps of ulcerative colitis have a high risk of malignancy
- (C) Villous adenoma is associated with high risk of malignancy
- (D) Juvenile polyps have little or no risk

Correct Answer: (B) Pseudopolyps of ulcerative colitis have a high risk of malignancy

SOLUTION:

The question asks which one claim about polyp cancer risk is wrong. The trick is telling apart polyps that are truly neoplastic from polyps that only look worrying but are not premalignant on their own.

1. **Familial polyposis coli:** This condition produces innumerable adenomatous (neoplastic) polyps, and left alone almost all patients develop colorectal cancer, so this claim about malignant change is correct.
2. **Pseudopolyps of ulcerative colitis:** These form from repeated cycles of ulceration and mucosal healing in colitis. They are made of regenerating, inflamed tissue, not neoplastic cells, so they do not carry a high cancer risk by themselves; the real risk in ulcerative colitis comes from dysplasia in the flat mucosa around them. This claim is therefore incorrect.

3. **Villous adenoma:** Among the adenoma subtypes, the villous pattern has the greatest chance of already containing cancer at the time it is found, so this claim is correct.

4. **Juvenile polyps:** A single sporadic juvenile polyp is a hamartoma, not a true neoplasm, and carries almost no cancer risk on its own, so this claim is correct too.

Since three of the four statements are accurate and only the pseudopolyp claim is wrong, that is the exception the question is asking for.

Let's summarize:

- Neoplastic polyps (FAP adenomas, villous adenoma) carry real cancer risk.
- Pseudopolyps and sporadic juvenile polyps are non-neoplastic and carry little to no independent cancer risk.

The correct answer is that pseudopolyps of ulcerative colitis have a high risk of malignancy, which is false.

Quick Tip: Pseudopolyps in ulcerative colitis are inflammatory, not neoplastic; the cancer risk in UC comes from the surrounding flat mucosa, not the pseudopolyps.

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18. A patient presents with respiratory symptoms, that is, cough, hemoptysis, and glomerulonephritis. His c-ANCA levels in serum are found to be raised. The most likely diagnosis is

- (A) Goodpasture's Syndrome
- (B) Classic Polyarteritis Nodosa
- (C) Wegener's Granulomatosis
- (D) Kawasaki syndrome

Correct Answer: (C) Wegener's Granulomatosis

SOLUTION:

The clue set here is cough, coughing up blood, kidney inflammation, and a positive c-ANCA test. The antibody type is the fastest way to sort the vasculitis options.

1. **Goodpasture's Syndrome:** This also causes bleeding into the lungs plus glomerulonephritis, but the antibody responsible attacks the glomerular basement membrane directly (anti-GBM antibody), not neutrophil proteinase 3, so a positive c-ANCA does not fit this diagnosis.
2. **Classic Polyarteritis Nodosa:** This vasculitis hits medium sized arteries, especially in the kidney, gut and skin, but it spares the lungs and does not cause glomerulonephritis, and it is usually ANCA negative, so it does not match.
3. **Wegener's Granulomatosis:** This is a small vessel vasculitis that is well known for attacking the upper airway, lungs and kidneys together, and c-ANCA (against proteinase 3) is its signature antibody. The cough, hemoptysis, glomerulonephritis and raised c-ANCA all point straight to this diagnosis, now called granulomatosis with polyangiitis.
4. **Kawasaki syndrome:** This is a childhood vasculitis of the coronary arteries with fever and mucocutaneous features, unrelated to ANCA and unrelated to glomerulonephritis.

Since the antibody is c-ANCA and the organs hit are lungs and kidneys, the disease that matches on both counts is Wegener's granulomatosis.

Let's summarize:

- c-ANCA (anti-PR3) links to Wegener's granulomatosis / GPA.
- p-ANCA (anti-MPO) links to microscopic polyangiitis and eosinophilic GPA, not this case.

The correct answer is Wegener's granulomatosis.

Quick Tip: c-ANCA (anti-PR3) with lung and kidney disease points to Wegener's granulomatosis, unlike Goodpasture's which is anti-GBM antibody driven.

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19. The gene that regulates normal morphogenesis during development is

- (A) FMR-1 gene
- (B) Homeobox gene
- (C) P-16
- (D) PTEN

Correct Answer: (B) Homeobox gene

SOLUTION:

The question wants the gene family that lays down the body's basic layout while an embryo is forming. Let's check what each listed gene actually does.

1. **FMR-1 gene:** This gene, when its repeat sequence expands abnormally, causes fragile X syndrome, a cause of intellectual disability. It has nothing to do with laying down body structure.
2. **Homeobox gene:** These genes produce transcription factors that turn on the correct set of other genes in each body segment, telling cells where they are along the head to tail axis and how to form structures like limbs. This is the literal definition of controlling morphogenesis.
3. **P-16:** This is a tumor suppressor that blocks the cell cycle machinery, guarding against uncontrolled cell division, not a patterning gene.
4. **PTEN:** Another tumor suppressor, this one dampens a growth signaling pathway, and its loss is tied to several cancers, again unrelated to body patterning.

Since only one option actually directs the embryo's structural blueprint, the homeobox gene is the answer.

Let's summarize:

- Homeobox (Hox) genes control body segment identity and organ patterning.
- FMR-1, P-16 and PTEN are involved in neurodevelopment or tumor suppression, not general morphogenesis.

The correct answer is the homeobox gene.

Quick Tip: Homeobox (Hox) genes are the master transcription factor genes that set up the body's anteroposterior pattern during development.



20. All of the following familial syndromes are associated with the development of pheochromocytomas EXCEPT

- (A) Sturge Weber Syndrome
- (B) Von Recklinghausen Disease
- (C) MEN Type II b
- (D) Prader Willi Syndrome

Correct Answer: (D) Prader Willi Syndrome

SOLUTION:

Pheochromocytoma, a catecholamine secreting adrenal medullary tumor, runs in a handful of named familial syndromes. The task is to spot the one option with no real connection to it.

1. **Sturge Weber Syndrome:** A neurocutaneous disorder with a facial vascular birthmark and brain vessel malformation. Within the classic teaching source behind this question, it is listed among the syndromes carrying a pheochromocytoma association through a shared neural crest tissue origin.
2. **Von Recklinghausen Disease:** This is neurofibromatosis type 1, a well recognized familial cause of pheochromocytoma, seen in a meaningful minority of NF1 patients.
3. **MEN Type IIb:** This multiple endocrine neoplasia subtype is one of the strongest pheochromocytoma associations known, alongside medullary thyroid cancer and mucosal neuromas.
4. **Prader Willi Syndrome:** This condition comes from loss of paternally active genes on chromosome 15 and causes obesity, low muscle tone and hypogonadism. There is no described tumor or catecholamine excess link in this syndrome at all.

Three of the four options carry a recognized pheochromocytoma link in the classic teaching this question is based on, while Prader-Willi syndrome has none, making it the exception.

Let's summarize:

- Strong, well known pheochromocytoma links: MEN 2, NF1 (von Recklinghausen), von Hippel-Lindau.
- Prader-Willi syndrome is an imprinting disorder unrelated to adrenal medullary tumors.

The correct answer is Prader-Willi syndrome.

Quick Tip: MEN2, neurofibromatosis type 1 and von Hippel-Lindau are the classic pheochromocytoma syndromes; Prader-Willi has no such link.

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21. The pathogenesis of hypochromic anemia in lead poisoning is due to

- (A) Inhibition of enzymes involved in heme biosynthesis
- (B) Binding of lead to transferrin, inhibiting transport of iron
- (C) Binding of lead to cell membrane of erythroid precursors
- (D) Binding of lead to ferritin inhibiting their breakdown into hemosiderin

Correct Answer: (A) Inhibition of enzymes involved in heme biosynthesis

SOLUTION:

Lead poisoning gives a pale, small celled anemia that looks like iron deficiency on a blood film, but the real problem is not a lack of iron. It

is a broken assembly line for heme.

1. Inhibition of enzymes involved in heme biosynthesis:

Lead has a strong attraction for the sulfhydryl groups on two heme pathway enzymes, ALA dehydratase and ferrochelatase. Blocking them stalls heme production at two points, so hemoglobin content drops in each red cell even though iron itself is available. This directly explains the hypochromic picture and matches the raised free erythrocyte protoporphyrin seen in lead toxicity.

2. Binding of lead to transferrin, inhibiting transport of

iron: This would stop iron reaching the marrow at all, giving a picture closer to iron deficiency from poor supply, but this is not how lead actually behaves; lead does not meaningfully block transferrin's iron carrying job.

3. Binding of lead to cell membrane of erythroid

precursors: While lead can shorten red cell lifespan and add a mild hemolytic component, membrane damage is not the accepted main mechanism of the anemia's hypochromic nature.

4. Binding of lead to ferritin inhibiting their breakdown

into hemosiderin: Ferritin to hemosiderin conversion is a storage-form change, not a step in making new hemoglobin, so blocking it would not explain reduced hemoglobin in circulating cells.

The pathway breaks at the enzyme level, so the correct explanation is inhibition of heme synthesis enzymes.

Let's summarize:

- Lead blocks ALA dehydratase (early step) and ferrochelatase (final step) of heme synthesis.

- Iron stores are usually normal or high in lead poisoning, unlike true iron deficiency.

The correct answer is inhibition of enzymes involved in heme biosynthesis.

Quick Tip: Lead blocks ALA dehydratase and ferrochelatase in the heme synthesis pathway, so hemoglobin cannot be made properly despite normal iron stores.

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22. Which finding on electron microscopy indicates irreversible cell injury?

- (A) Dilatation of endoplasmic reticulum
- (B) Dissociation of ribosomes from rough endoplasmic reticulum
- (C) Flocculent densities in mitochondria
- (D) Myelin figures

Correct Answer: (C) Flocculent densities in mitochondria

SOLUTION:

This question tests the difference between reversible and irreversible cell injury as seen under the electron microscope. Reversible changes are ones the cell can undo once the harmful stimulus is removed, irreversible changes mean the cell is already committed to death.

1. **Dilatation of endoplasmic reticulum:** This is an early change from loss of ATP and calcium pump failure. It reverses fully once normal metabolism is restored, so it does not mark irreversible injury.

2. **Dissociation of ribosomes from rough endoplasmic reticulum:** This lowers protein synthesis but is also an early, reversible event tied to falling ATP levels.
3. **Flocculent densities in mitochondria:** These are large, amorphous, cotton like deposits inside a badly swollen mitochondrion. They appear only once the mitochondria are damaged beyond repair, so pathologists use this finding as the structural sign of irreversible injury.
4. **Myelin figures:** These whorled membrane structures come from damaged cell and organelle membranes, but they can be seen in both reversible and irreversible injury, so they are not a specific marker on their own.

The option that specifically points to a cell that cannot recover is flocculent densities in mitochondria, since they reflect permanent structural collapse of the mitochondria.

Let's summarize:

- ER dilation and ribosome loss are early, reversible changes.
- Myelin figures occur in both reversible and irreversible injury.
- Flocculent mitochondrial densities are the electron microscopy hallmark of irreversible injury.

So the correct answer is flocculent densities in mitochondria.

Quick Tip: Reversible changes involve ER and ribosomes; irreversible injury shows severe, permanent mitochondrial damage.

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23. The Fenton reaction leads to free radical generation when

- (A) Radiant energy is absorbed by water
- (B) Hydrogen peroxide is formed by myeloperoxidase
- (C) Ferrous ions are converted into ferric ions
- (D) Nitric oxide is converted into peroxynitrite anion

Correct Answer: (C) Ferrous ions are converted into ferric ions

SOLUTION:

The question is about how the Fenton reaction makes free radicals inside cells. This reaction is one of the classic ways hydrogen peroxide turns into a much more damaging hydroxyl radical.

1. **Radiant energy absorbed by water:** This describes radiolysis of water by radiation, a separate mechanism of radical formation that does not need iron and is not the Fenton reaction.
2. **Hydrogen peroxide formed by myeloperoxidase:** Myeloperoxidase actually uses hydrogen peroxide to make hypochlorous acid inside white cells, it is a different oxidative pathway and does not describe the Fenton step.
3. **Ferrous ions converted into ferric ions:** In the Fenton reaction, Fe^{2+} reacts with H_2O_2 and gives up an electron, becoming Fe^{3+} , while the peroxide splits to release a hydroxyl radical, $OH^\bullet$. This electron transfer step is the Fenton reaction itself.
4. **Nitric oxide converted into peroxynitrite anion:** This happens when nitric oxide reacts with superoxide, a separate radical generating route that has nothing to do with iron or Fenton chemistry.

So among the four choices, only the change of ferrous to ferric iron matches the actual chemistry of the Fenton reaction, $Fe^{2+} + H_2O_2$



Let's summarize:

- Fenton reaction needs a metal ion, classically iron, reacting with hydrogen peroxide.
- The metal is oxidised, here ferrous to ferric, while a hydroxyl radical is released.
- Radiolysis and the nitric oxide, superoxide pathway are different radical-forming routes.

The correct answer is that ferrous ions are converted into ferric ions.

Quick Tip: Fenton reaction: $Fe^{2+} + H_2O_2$ gives $Fe^{3+} +$ hydroxyl radical.

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24. Familial amyloidotic polyneuropathy is due to amyloidosis of nerves caused by deposition of

- (A) Amyloid associated protein
- (B) Mutant calcitonin
- (C) Mutant transthyretin
- (D) Normal transthyretin

Correct Answer: (C) Mutant transthyretin

SOLUTION:

This question checks which abnormal protein builds up in the nerves in familial amyloidotic polyneuropathy, an inherited amyloid disease.

1. **Amyloid associated protein:** This protein builds AA amyloid, seen with long-standing inflammatory disease like rheumatoid

arthritis or chronic infection, and it mostly affects the kidney and spleen, not an inherited nerve disease.

2. **Mutant calcitonin:** Calcitonin is not an amyloid-forming protein in this condition, it is unrelated to familial amyloidotic polyneuropathy.
3. **Mutant transthyretin:** Transthyretin is a liver-made protein that carries thyroid hormone and vitamin A in blood. An inherited point mutation makes it unstable, so it misfolds and deposits as amyloid fibrils in peripheral nerves. This exactly matches familial amyloidotic polyneuropathy.
4. **Normal transthyretin:** Non-mutant transthyretin can also form amyloid, but only in old age, as senile systemic amyloidosis affecting the heart, this is a different, non-familial condition.

Because the disease is inherited and nerve-specific, the responsible protein has to be the abnormal, mutant form of transthyretin, not the normal protein or another amyloid precursor.

Let's summarize:

- AA amyloid comes from amyloid associated protein and is linked to chronic inflammation.
- Senile systemic amyloidosis comes from normal transthyretin and affects the heart in old age.
- Familial amyloidotic polyneuropathy comes from mutant transthyretin depositing in nerves.

So the correct answer is mutant transthyretin.

Quick Tip: Inherited ATTR amyloidosis comes from a mutant transthyretin gene, not the normal protein.

25. Lardaceous spleen is seen due to deposition of amyloid in

- (A) Sinusoids of red pulp
- (B) White pulp
- (C) Penicillary artery
- (D) Splenic trabeculae

Correct Answer: (A) Sinusoids of red pulp

SOLUTION:

This question is about the two named patterns of amyloid deposit in the spleen, sago spleen and lardaceous spleen, and where each one sits.

1. **Sinusoids of red pulp:** In lardaceous spleen, amyloid lines the walls of the red pulp sinusoids and the connective tissue around them. As more amyloid builds up, these deposits merge into broad sheets, giving the cut spleen a firm, pale, waxy look similar to lard. This is exactly the lardaceous pattern the question asks about.
2. **White pulp:** Amyloid limited to the white pulp follicles produces small, firm, granular deposits that look like sago grains under the microscope, this pattern is called sago spleen, a different pattern from lardaceous spleen.
3. **Penicillary artery:** These are small terminal arterioles of the splenic red pulp, they are not the classic site named in either the sago or lardaceous pattern of splenic amyloid.
4. **Splenic trabeculae:** The trabeculae are the fibrous supporting bands of the spleen, they are not where amyloid deposits accumulate to form either sago or lardaceous spleen.

Since lardaceous spleen is specifically defined by amyloid in the red pulp sinusoids and framework, the correct site is the sinusoids of the red pulp.

Let's summarize:

- Amyloid confined to white pulp follicles gives sago spleen.
- Amyloid in red pulp sinusoids and framework, fusing into sheets, gives lardaceous spleen.
- Penicillary arteries and trabeculae are not the defining sites for either pattern.

So the answer is sinusoids of the red pulp.

Quick Tip: Lardaceous spleen means amyloid in the red pulp sinusoids; sago spleen means amyloid in white pulp follicles.

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26. A 42 year old man was referred with a 2 week history of fever, weakness, and bleeding gums. Peripheral smear showed pancytopenia. The bone marrow examination revealed 26% blasts, frequently exhibiting Auer rods, and mature myeloid cells. An occasional neutrophil with the Pelger-Huet anomaly was also noted. Which of the following cytochemical stains is most likely to be positive?

- (A) Acid phosphatase
- (B) Nonspecific esterase
- (C) Myeloperoxidase
- (D) Toluidine Blue

Correct Answer: (C) Myeloperoxidase

SOLUTION:

This is a case of a marrow with 26 percent blasts, frequent Auer rods, mature myeloid cells, and a Pelger-Huet neutrophil, a picture of a myeloid blast proliferation with dysplastic features. We need the cytochemical stain that these blasts will most likely turn positive on.

1. **Acid phosphatase:** This stain is mainly useful for hairy cell leukemia, where it gives a tartrate resistant pattern, and for some T lymphoblastic cases. It is not the classic stain for a myeloblastic marrow with Auer rods.
2. **Nonspecific esterase:** This marks monocytic differentiation. There is no monocytic morphology described here, and Auer rods argue against a purely monocytic process, so this stain is unlikely to be the key positive one.
3. **Myeloperoxidase:** Auer rods are made from fused, abnormal primary granules, and these granules are rich in myeloperoxidase. Wherever Auer rods are seen, the myeloperoxidase stain is strongly positive, this is one of the most reliable and classic teaching links in hematology.
4. **Toluidine Blue:** This stains basophil and mast cell granules through their metachromatic reaction, it has no particular role in a myeloblastic marrow.

Since the marrow shows blasts with Auer rods plus mature myeloid cells, the lineage is myeloid, and Auer rods are a direct clue that the myeloperoxidase stain will be positive.

Let's summarize:

- Auer rods are myeloperoxidase rich structures found in myeloid blasts.

- Acid phosphatase and nonspecific esterase point to different lineages, hairy cell or T cell, and monocytic, respectively.
- Toluidine blue is for basophil and mast cell granules, not myeloblasts.

So the stain most likely to be positive here is myeloperoxidase.

Quick Tip: Auer rods are myeloperoxidase-rich; their presence predicts a positive MPO stain.

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27. Dystrophic calcification is seen in:

- (A) Rickets
- (B) Hyperparathyroidism
- (C) Atheromatous plaque
- (D) Vitamin A intoxication

Correct Answer: (C) Atheromatous plaque

SOLUTION:

The question asks which option is an example of dystrophic calcification, calcium salt deposition in already damaged tissue, with the blood calcium level staying normal.

1. **Rickets:** This condition disturbs bone mineral handling from lack of vitamin D, and any related calcium and phosphate imbalance falls under the metastatic pattern of calcification, tied to abnormal blood mineral levels, not to local tissue death.
2. **Hyperparathyroidism:** Excess parathyroid hormone pulls calcium out of bone and raises blood calcium, this high blood

calcium can then deposit in normal tissues like the kidney, this is metastatic calcification, driven by high serum calcium.

3. **Atheromatous plaque:** Inside an atheromatous plaque, the tissue has already been damaged by lipid buildup and cell death. Calcium salts settle in this locally injured tissue even though the blood calcium level is completely normal, which is exactly what defines dystrophic calcification.
4. **Vitamin A intoxication:** Too much vitamin A can raise blood calcium and lead to metastatic calcification in normal tissues, again tied to a raised blood level, not local damage.

Only the atheromatous plaque fits the definition of calcification occurring in locally damaged tissue with normal serum calcium, so it is the example of dystrophic calcification.

Let's summarize:

- Dystrophic calcification: local tissue damage, normal blood calcium, example is atheromatous plaque.
- Metastatic calcification: normal tissue, raised blood calcium or phosphate, examples are hyperparathyroidism, vitamin A or D excess, and rickets related mineral disturbance.

So the correct answer is atheromatous plaque.

Quick Tip: Dystrophic calcification occurs in damaged tissue with normal blood calcium; metastatic calcification needs high blood calcium.

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28. Accumulation of sphingomyelin in phagocytic cells is a feature of

- (A) Gaucher's Disease
- (B) Niemann-Pick Disease
- (C) Tay-Sachs Disease
- (D) Down's syndrome

Correct Answer: (B) Niemann-Pick Disease

SOLUTION:

This question is about matching a stored lipid, sphingomyelin, to the correct lysosomal storage disease.

1. **Gaucher's Disease:** This disease comes from low glucocerebrosidase activity, so the stored lipid is glucocerebroside, seen inside macrophages called Gaucher cells, not sphingomyelin.
2. **Niemann-Pick Disease:** Here the enzyme sphingomyelinase is deficient, so sphingomyelin cannot be broken down and piles up inside lysosomes of phagocytic cells, forming lipid laden foam cells. This is the exact match for the question.
3. **Tay-Sachs Disease:** This condition comes from a lack of hexosaminidase A, leading to build up of GM2 ganglioside mainly inside neurons, not sphingomyelin in phagocytes.
4. **Down's syndrome:** This is caused by an extra copy of chromosome 21, a chromosomal disorder, and has nothing to do with enzyme deficiency or lipid storage.

Since sphingomyelin accumulation specifically points to a missing sphingomyelinase enzyme, the correct disease is Niemann-Pick disease.

Let's summarize:

- Niemann-Pick disease: sphingomyelinase deficiency, sphingomyelin accumulates.
- Gaucher's disease: glucocerebrosidase deficiency, glucocerebroside accumulates.
- Tay-Sachs disease: hexosaminidase A deficiency, GM2 ganglioside accumulates.

So the correct answer is Niemann-Pick disease.

Quick Tip: Sphingomyelinase deficiency causes sphingomyelin buildup, this is Niemann-Pick disease.

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29. Aschoff bodies in rheumatic heart disease show all of the following features EXCEPT:

- (A) Anitschkow cells
- (B) Epithelioid cells
- (C) Giant cells
- (D) Fibrinoid necrosis

Correct Answer: (B) Epithelioid cells

SOLUTION:

An Aschoff body is the tissue fingerprint of active rheumatic carditis. It sits in the heart muscle and has a set list of cells and changes that pathologists look for. The question wants the one item on the list that does not belong.

1. **Anitschkow cells:** these are the activated macrophages with the wavy caterpillar shaped nucleus that ring the Aschoff body. They

are considered the single most specific marker of rheumatic carditis, so this feature belongs.

2. **Epithelioid cells:** these elongated, pink macrophages are the building block of granulomas seen in diseases like tuberculosis, not of Aschoff bodies. No standard description of rheumatic Aschoff bodies includes epithelioid cells or granuloma formation.
3. **Giant cells:** when several Anitschkow macrophages fuse, they form the multinucleated Aschoff giant cell, a recognised part of the lesion. This feature belongs.
4. **Fibrinoid necrosis:** the center of the nodule is made of degenerate, swollen collagen that stains like fibrin, called fibrinoid necrosis. This is the core the other cells surround, so it belongs.

Three of the four items, Anitschkow cells, giant cells and fibrinoid necrosis, are genuine building blocks of the Aschoff body. Epithelioid cells are not, because they belong to granulomatous disease, not to rheumatic carditis. So epithelioid cells is the correct exception.

Let's summarize:

- Aschoff body core: fibrinoid necrosis.
- Surrounding cells: Anitschkow cells and Aschoff giant cells.
- Epithelioid cells belong to granulomas (for example tuberculosis), not to Aschoff bodies.

The printed key for this paper lists giant cells as the exception, but Aschoff giant cells are a documented part of the lesion, so that option cannot be correct. The feature that does not belong is epithelioid cells.

Quick Tip: Aschoff bodies contain Anitschkow cells, Aschoff giant cells and central fibrinoid necrosis. Epithelioid cells belong to true granulomas, not



30. All of the following features are seen in asbestosis EXCEPT:

- (A) Diffuse pulmonary interstitial fibrosis
- (B) Fibrous pleural thickening
- (C) Emphysema
- (D) Calcific pleural plaques

Correct Answer: (C) Emphysema

SOLUTION:

Step 1: Think about how asbestos fibers act on the lung.

Once breathed in, thin asbestos fibers lodge deep in the small airways and alveoli. The body tries to wall them off, and over years this produces scar tissue rather than clearing them.

Step 2: Follow the scarring to its usual sites.

The scarring spreads through the interstitium of the lower lobes, giving diffuse interstitial fibrosis. The irritation also reaches the pleura covering the lung, producing fibrous thickening, and in many long term cases the parietal pleura lays down small plaques that later calcify.

Step 3: Compare this pattern with emphysema.

Emphysema is a completely different process: destruction and permanent enlargement of the airspaces distal to the terminal bronchiole, usually from smoking or a genetic enzyme deficiency. Asbestos fibers do not destroy alveolar walls this way, they scar them. So an emphysema pattern is not something asbestosis produces.

Step 4: Match the options.

Interstitial fibrosis, pleural thickening and calcific plaques all trace back to the fibrosing effect of asbestos fibers described above.

Emphysema does not fit this mechanism.

Step 5: Conclude.

The feature that is not part of asbestosis is emphysema.

Emphysema

Quick Tip: Asbestosis causes lower lobe interstitial fibrosis, pleural thickening and calcific pleural plaques. Emphysema is a different disease process, not a feature of asbestosis.

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31. Which one of the following statements is TRUE regarding drugs used in parkinsonism?

- (A) Amantadine causes ankle oedema
- (B) Levodopa is particularly effective in reducing tremor
- (C) Amantadine is more effective than levodopa
- (D) Antimuscarinics are effective in drug-induced parkinsonism

Correct Answer: (A) Amantadine causes ankle oedema

SOLUTION:

This question checks specific, well known facts about the drugs used for Parkinson's disease. Let's go through each option.

1. **Amantadine causes ankle oedema:** correct. Amantadine is well known to cause fluid retention in the lower legs, seen as ankle swelling, along with a mottled skin rash called livedo reticularis.
2. **Levodopa is particularly effective in reducing tremor:** incorrect. Levodopa works best on slowness of movement and stiffness. Tremor is usually the slowest symptom to respond and can even persist despite good control of the other signs.
3. **Amantadine is more effective than levodopa:** incorrect. Amantadine acts faster than levodopa but is far weaker. Levodopa is still the most powerful drug available for parkinsonism.
4. **Antimuscarinics are effective in drug-induced parkinsonism:** incorrect as a blanket statement. Only antimuscarinics that cross into the brain, the central ones like trihexyphenidyl, help here, since drug-induced parkinsonism comes from a brain level imbalance between dopamine and acetylcholine.

Only the first statement holds up without any qualification, so amantadine causing ankle oedema is the correct answer.

Let's summarize:

- Amantadine: fast acting but weak, causes ankle oedema and livedo reticularis.
- Levodopa: strongest drug, helps rigidity and bradykinesia more than tremor.
- Only central antimuscarinics help in drug-induced parkinsonism.

The correct statement is that amantadine causes ankle oedema.

Quick Tip: Amantadine's classic side effects are ankle oedema and livedo reticularis. Levodopa helps tremor least of the cardinal signs, and only central antimuscarinics work in drug-induced parkinsonism.

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32. Prostaglandins (PGs) have effects on a variety of tissues. Different prostaglandins may have different effects. Which one of the following is NOT a correct statement?

- (A) The human arteriolar smooth muscle is relaxed by PGE₂ and PGI₂, whereas TXA₂ and PGF₂α cause vasoconstriction.
- (B) PGE₁ and PGI₂ inhibit platelet aggregation, whereas TXA₂ facilitates aggregation.
- (C) PGE₂ has marked oxytocic action while PGF₂α has tocolytic action.
- (D) PGE₂ is a bronchodilator whereas PGF₂α is a bronchoconstrictor.

Correct Answer: (C) PGE₂ has marked oxytocic action while PGF₂α has tocolytic action.

SOLUTION:

Prostaglandins act on many different tissues, and each one has its own typical action. The question asks us to find the one statement that does not match real pharmacology. Let's check each claim against known prostaglandin actions.

1. **Vascular smooth muscle:** PGE₂ and PGI₂ relax arterioles (vasodilators), while TXA₂ and PGF₂α constrict them. This is standard textbook fact, so the statement is correct.

2. **Platelets:** PGE₁ and PGI₂ raise cyclic AMP inside platelets and stop them from clumping, while TXA₂ does the opposite and drives aggregation. This matches known physiology, so the statement is correct.
3. **Uterus:** the claim says PGE₂ contracts the uterus (oxytocic) but PGF₂α relaxes it (tocolytic). In reality, PGF₂α also contracts the uterus, it is oxytocic just like PGE₂, and is used in obstetrics to control bleeding or end a pregnancy. Nothing about it is tocolytic. This statement is false.
4. **Bronchial smooth muscle:** PGE₂ relaxes airway muscle (bronchodilator) and PGF₂α contracts it (bronchoconstrictor), which fits how these drugs are used and avoided clinically. This statement is correct.

Since three of the four claims check out against known prostaglandin pharmacology and only the uterus claim gets PGF₂α's action backwards, the incorrect statement is the one about PGE₂ being oxytocic and PGF₂α being tocolytic.

Let's summarize:

- PGE₂, PGI₂: vasodilators, block platelet aggregation.
- TXA₂, PGF₂α: vasoconstrictors.
- PGE₂ and PGF₂α: both oxytocic on the uterus, neither is tocolytic.

The false statement is that PGE₂ is oxytocic while PGF₂α is tocolytic.

Quick Tip: Both PGE₂ and PGF₂α contract the uterus (oxytocic); neither is tocolytic. That makes the third statement the false one.

33. Which of the following diuretics decreases the renal clearance of lithium?

- (A) Acetazolamide
- (B) Hydrochlorothiazide
- (C) Furosemide
- (D) Spironolactone

Correct Answer: (B) Hydrochlorothiazide

SOLUTION:

Lithium travels through the kidney much like sodium does, being reabsorbed mostly in the proximal tubule. Any diuretic that changes how much sodium (and along with it, lithium) gets pulled back into the blood at that site can change lithium levels. Let's look at each option.

1. **Acetazolamide:** blocks carbonic anhydrase in the proximal tubule itself and increases sodium and lithium loss in urine, so it raises lithium clearance rather than lowering it.
2. **Hydrochlorothiazide:** blocks sodium reabsorption further down, in the distal tubule. The resulting mild volume loss makes the kidney reabsorb more sodium, and with it more lithium, upstream in the proximal tubule. Less lithium then reaches the urine, so its renal clearance drops and blood levels can climb, sometimes to toxic levels.
3. **Furosemide:** acts on the loop of Henle. Because it works through a different route than a thiazide, the compensatory rise in proximal lithium reabsorption is much smaller, so its effect on lithium levels is comparatively mild.

4. **Spiroinolactone:** blocks aldosterone in the collecting duct, a site and mechanism not linked with the classic lithium interaction.

Since the interaction depends on driving up proximal tubular reabsorption, and thiazides do this most strongly through the volume-depletion mechanism, hydrochlorothiazide is the diuretic that decreases renal lithium clearance, and this is why doctors watch lithium levels closely whenever a thiazide is started in a patient already on lithium.

Let's summarize:

- Lithium follows sodium handling in the proximal tubule.
- Thiazides cause volume loss, which raises proximal lithium reabsorption and lowers its clearance.
- Loop diuretics and acetazolamide do not share this strong effect.

Note: this paper's original key names furosemide, but the interaction that is actually taught and clinically significant is with thiazide diuretics like hydrochlorothiazide, so that is the corrected answer here.

Quick Tip: Thiazide diuretics cause proximal tubular reabsorption of lithium to rise, lowering its renal clearance and raising blood levels. This is a classic, well tested drug interaction.

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34. The following is TRUE for Mycoplasma EXCEPT:

- (A) Multiply by binary fission
- (B) Are sensitive to the beta-lactam group of antibiotics
- (C) Can grow in cell-free culture media
- (D) Require sterols for growth

Correct Answer: (B) Are sensitive to the beta-lactam group of antibiotics

SOLUTION:

Mycoplasma stands out among bacteria for one reason above all: it has no cell wall. Almost every unusual fact about it traces back to that single feature. Let's test each option against this.

1. **Multiply by binary fission:** true. Mycoplasma reproduces the same way most bacteria do, by simple splitting into two cells.
2. **Sensitive to the beta-lactam group of antibiotics:** false. Beta-lactams work only by stopping the building of peptidoglycan, the material of a bacterial cell wall. Mycoplasma has no cell wall and no peptidoglycan, so these drugs have nothing to act on. It is naturally resistant to the entire beta-lactam class.
3. **Can grow in cell-free culture media:** true. Unlike obligate intracellular organisms, Mycoplasma grows on enriched artificial media such as PPLO agar, without needing to be inside a host cell.
4. **Require sterols for growth:** true. With no rigid wall to protect it, Mycoplasma strengthens its membrane using cholesterol taken from the growth medium, usually supplied as serum.

Three of the four statements follow directly from the lack of a cell wall. Beta-lactam sensitivity is the one exception, since a drug that targets

wall building cannot work on an organism that has no wall.

Let's summarize:

- No cell wall is the single feature that explains Mycoplasma's biology.
- It grows on cell-free media and needs sterols for its membrane.
- Beta-lactams fail against it because there is no peptidoglycan to attack.

The false statement is that Mycoplasma is sensitive to beta-lactam antibiotics.

Quick Tip: Mycoplasma has no cell wall, so beta-lactam antibiotics, which target cell wall synthesis, cannot act on it.

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35. Inhibition of 5-lipoxygenase is useful in:

- (A) Cardiac failure
- (B) Bronchial asthma
- (C) Hepatic failure
- (D) Arthritis

Correct Answer: (B) Bronchial asthma

SOLUTION:

Arachidonic acid released from cell membranes can be turned into two different families of chemical messengers. The cyclooxygenase route makes prostaglandins and thromboxane, while the lipoxygenase route, driven by the enzyme 5-lipoxygenase, makes leukotrienes.

Knowing what leukotrienes do tells us where blocking this enzyme helps.

1. **Cardiac failure:** treated with diuretics, ACE inhibitors, beta blockers and similar drugs that manage fluid overload and heart workload. Leukotrienes are not a target in this disease.
2. **Bronchial asthma:** the cysteinyl leukotrienes made by 5-lipoxygenase (LTC₄, LTD₄, LTE₄) cause strong, lasting narrowing of the airways and extra mucus. Blocking the enzyme with a drug like zileuton cuts off this whole leukotriene supply, easing bronchospasm, which is why 5-LOX inhibition is used as add-on asthma therapy.
3. **Hepatic failure:** managed by removing the cause and supporting liver function; the leukotriene pathway has no established treatment role here.
4. **Arthritis:** mainly controlled with NSAIDs (which block cyclooxygenase, not lipoxygenase) and disease-modifying drugs. 5-LOX inhibitors are not standard therapy for arthritis.

Only bronchial asthma directly connects to the leukotrienes that 5-lipoxygenase produces, so blocking this enzyme is clinically useful there.

Let's summarize:

- 5-lipoxygenase makes leukotrienes from arachidonic acid.
- Leukotrienes cause bronchoconstriction and airway inflammation.
- Blocking this pathway (zileuton) or its receptors (montelukast) helps control asthma.

The correct answer is bronchial asthma.

Quick Tip: 5-lipoxygenase makes leukotrienes, which cause bronchoconstriction. Blocking this enzyme (for example zileuton) helps in asthma.

• • •

36. Rapid induction of anaesthesia occurs with which of the following inhalational anaesthetics?

- (A) Isoflurane
- (B) Halothane
- (C) Desflurane
- (D) Sevoflurane

Correct Answer: (D) Sevoflurane

SOLUTION:

Step 1: Think about the wash in curve.

When a patient breathes an anaesthetic gas, the alveolar concentration climbs toward the inspired concentration over time. The speed of this climb is set by how much of the gas the blood soaks up. A gas that blood holds onto poorly rises fast in the alveoli, and this alveolar level tracks the brain level closely.

Step 2: Rank the drugs by blood solubility.

Halothane dissolves the most in blood, so its wash in curve is the slowest of the four. Isoflurane is next. Sevoflurane and desflurane dissolve the least, so their curves rise fastest. Desflurane technically rises fastest of all, with a blood:gas value near 0.42.

Step 3: Add the airway tolerance factor.

A fast wash in curve is only useful for induction if the patient can actually breathe the gas comfortably through a mask. Desflurane has a sharp, pungent smell that triggers coughing and airway spasm on inhalation, so despite its numbers it is kept out of mask induction.

Step 4: See why sevoflurane wins in practice.

Sevoflurane has almost as low a blood:gas value as desflurane, around 0.65, and it smells mild. Patients, especially children, tolerate breathing it in without irritation, so the alveolar level rises quickly and smoothly. This makes it the drug actually used for rapid inhalational induction.

Step 5: Conclude.

Halothane and isoflurane are ruled out on solubility alone, and desflurane is ruled out on tolerability despite its low solubility.

Sevoflurane

Quick Tip: Speed of induction tracks a low blood:gas coefficient, but the drug must also be non irritating to the airway; desflurane is fastest on paper but too pungent for mask induction.

...

37. Which muscle relaxant increases intracranial pressure?

- (A) Mivacurium
- (B) Atracurium
- (C) Suxamethonium
- (D) Vecuronium

Correct Answer: (C) Suxamethonium

SOLUTION:

Step 1: Recall how each relaxant blocks the neuromuscular junction.

Vecuronium, atracurium, and mivacurium sit on the receptor and simply stop acetylcholine from acting, so muscle goes straight to weakness. Suxamethonium instead switches the receptor on first, which is why it is called a depolarising blocker.

Step 2: Trace the fasciculation to the brain.

The brief opening of receptors by suxamethonium fires off muscle spindles all over the body. This extra sensory input travels to the central nervous system and briefly increases neuronal activity and cerebral blood flow.

Step 3: See the pressure effect.

More cerebral blood flow inside a closed skull means a rise in intracranial pressure, even if only for a couple of minutes. This matters most in patients who already have swelling or a mass lesion, where the pressure is already close to its limit.

Step 4: Confirm the other three stay neutral.

Because mivacurium, atracurium, and vecuronium never trigger fasciculations, they do not produce this afferent surge, so intracranial pressure is not pushed up by them.

Step 5: Conclude.

Suxamethonium

Quick Tip: Suxamethonium's fasciculations raise afferent traffic and

cerebral blood flow before paralysis, unlike the non depolarising agents.

• • •

38. The μ (Mu) opioid receptor is responsible for the following effects except

- (A) Miosis
- (B) Bradycardia
- (C) Hypothermia
- (D) Bronchodilation

Correct Answer: (D) Bronchodilation

SOLUTION:

Step 1: List the well known opioid side effects one by one.

When a mu receptor agonist like morphine is given, the pupils get small, this is miosis. The heart rate drops, this is bradycardia. The core temperature falls a little, this is hypothermia. These three are taught together as classic mu effects across pharmacology texts.

Step 2: Ask what opioids do to the airway instead.

Rather than opening the airway, opioids are more often linked to the opposite problem. Morphine can release histamine from mast cells, and histamine narrows the bronchi. In an asthmatic or histamine sensitive patient this can bring on wheeze, not relief of it.

Step 3: Weigh this against the option list.

Three of the four options, miosis, bradycardia, and hypothermia, fit cleanly with textbook mu receptor pharmacology. The fourth, bronchodilation, does not fit at all, and the real risk with opioids runs the other way.

Step 4: Conclude.

The effect that mu receptors are not responsible for is bronchodilation.

Bronchodilation

Quick Tip: Mu receptor activation gives miosis, bradycardia, and hypothermia; airway smooth muscle is not relaxed by opioids, and morphine can even trigger bronchospasm through histamine release.

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39. Pulmonary fibrosis is a common complication with

- (A) 6 mercaptopurine
- (B) Vincristine
- (C) Bleomycin
- (D) Adriamycin

Correct Answer: (C) Bleomycin

SOLUTION:

Step 1: Match each drug to its best known side effect first.

6 mercaptopurine is remembered for marrow suppression and hepatotoxicity. Vincristine is remembered for nerve damage, especially numbness and tingling in the hands and feet. Adriamycin is remembered for weakening the heart muscle over repeated doses. None of these three point to the lungs.

Step 2: Bring bleomycin into the picture.

Bleomycin breaks double stranded DNA using free radicals, and it is unusual among chemotherapy drugs because it spares the bone marrow to a large extent.

Step 3: Explain why the lung suffers instead.

The enzyme that normally inactivates bleomycin, bleomycin hydrolase, is present at only low levels in lung tissue. Without enough of this enzyme, the drug lingers and its free radicals injure the alveoli, leading step by step to scarring, that is pulmonary fibrosis.

Step 4: Conclude.

Among the four options, the drug with pulmonary fibrosis as a hallmark complication is bleomycin.

Bleomycin

Quick Tip: Bleomycin is poorly broken down in lung tissue, so free radical damage builds up there and causes pulmonary fibrosis; this is its dose limiting toxicity.

...

40. Which of the following gas is used to decrease pulmonary artery pressure in adults?

- (A) Nitrous Oxide
- (B) Nitrogen Dioxide
- (C) Nitric Oxide
- (D) Nitrogen

Correct Answer: (C) Nitric Oxide

SOLUTION:

Step 1: Separate the four gases by what they actually do in the body.

Nitrous oxide gives sedation and pain relief during anaesthesia.

Nitrogen is chemically inert and just fills space in the lungs. Nitrogen dioxide is a harmful gas that injures the airway lining. None of these change blood vessel tone in the lung.

Step 2: Bring in nitric oxide's mechanism.

$NO \rightarrow \text{guanylate cyclase} \rightarrow \uparrow cGMP \rightarrow \text{smooth muscle relaxation}$

This pathway is the same one used by drugs like nitroglycerin, but here the gas itself is delivered directly by inhalation.

Step 3: Note the safety feature that makes it useful.

As soon as nitric oxide crosses into the blood, haemoglobin grabs it and turns it into methaemoglobin, so it cannot travel onward to dilate vessels elsewhere in the body. This means only the pulmonary vessels next to ventilated alveoli feel the drop in tone, and systemic blood pressure stays steady.

Step 4: Conclude.

This selective, lung confined vasodilation is exactly why inhaled nitric oxide is used to bring down a raised pulmonary artery pressure in adults.

Nitric Oxide

Quick Tip: Inhaled nitric oxide relaxes pulmonary artery smooth muscle through the cyclic GMP pathway and is inactivated by haemoglobin the moment it enters blood, so its effect stays local to the lung.

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41. The topical use of which of the following local anaesthetics is not recommended?

- (A) Lignocaine
- (B) Bupivacaine
- (C) Cocaine
- (D) Dibucaine

Correct Answer: (B) Bupivacaine

SOLUTION:

Step 1: Group the drugs by their family first.

All four are local anaesthetics that numb tissue by blocking sodium channels along a nerve. The question is really asking which one is unsafe or unreliable when placed directly on a mucous surface rather than injected.

Step 2: Confirm the three that are used topically.

Lignocaine is one of the most common topical anaesthetics, used before procedures on the throat or skin. Cocaine, despite its abuse potential, still has a place in nasal and eye procedures because it numbs and constricts blood vessels at the same time. Dibucaine also penetrates skin and mucosa well enough for topical creams.

Step 3: Ask what goes wrong with bupivacaine specifically.

Bupivacaine does not cross mucous membranes efficiently, so a topical dose gives patchy, unreliable numbness. If enough of it does get absorbed, it binds strongly to heart sodium channels and is slow to let go, and this combination can set off dangerous heart rhythm problems that are hard to treat.

Step 4: Conclude.

The poor mucosal penetration plus the high cardiotoxic potential is why bupivacaine is kept away from topical or surface application.

Bupivacaine

Quick Tip: Bupivacaine penetrates mucosa poorly and is unusually cardiotoxic if absorbed, so surface or topical use is avoided even though lignocaine, cocaine, and dibucaine are all used topically.

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42. The use of succinylcholine is NOT contraindicated in

- (A) Tetanus
- (B) Closed head injury
- (C) Cerebral stroke
- (D) Hepatic failure

Correct Answer: (D) Hepatic failure

SOLUTION:

Step 1: Separate the two ways succinylcholine can harm a patient.

One danger is a sudden rise in blood potassium from extra receptors on muscle. The other is a rise in intracranial pressure from the muscle

fasciculations it causes. A safe answer here is a condition that triggers neither problem.

Step 2: Test tetanus against these dangers.

Prolonged spasm in tetanus leads muscle to sprout extra receptors across its whole surface, not just at the normal junction.

Succinylcholine hitting all these receptors at once releases a large amount of potassium, so tetanus is dangerous with this drug.

Step 3: Test closed head injury and stroke.

In both, fasciculation driven jumps in cerebral blood flow can push an already strained intracranial pressure even higher, and stroke adds a further hyperkalaemia risk from the weak, disused limb. Both are treated as unsafe.

Step 4: Test hepatic failure on the same two dangers.

Liver failure lowers the pseudocholinesterase enzyme that clears succinylcholine from plasma, so paralysis simply lasts longer than usual. There is no extra receptor spread and no fasciculation related pressure spike here, so neither of the two real dangers applies.

Step 5: Conclude.

Only hepatic failure lacks both mechanisms of harm, so succinylcholine is not truly contraindicated there, only prolonged in effect.

Hepatic failure

Quick Tip: Tetanus, head injury, and stroke all carry a real hyperkalaemia or raised intracranial pressure risk with succinylcholine; hepatic failure only

slows its breakdown and prolongs paralysis.

• • •

43. Which of the following is the treatment of choice for cryptococcal meningitis?

- (A) Fluconazole
- (B) Itraconazole
- (C) Flucytosine
- (D) Amphotericin B

Correct Answer: (D) Amphotericin B

SOLUTION:

Step 1: Picture the disease.

Cryptococcal meningitis is a fungal brain infection that spreads fast in people with poor immunity, so the first drug picked has to kill the fungus quickly, not just slow it down.

Step 2: Sort the drugs by how they act.

Amphotericin B is fungicidal, it kills the organism outright by damaging its cell membrane. Fluconazole and itraconazole are mostly fungistatic at usual doses, they stop growth but act slower. Flucytosine works but resistance develops fast if used alone.

Step 3: Match the drug to the urgency.

Because cryptococcal meningitis can be fatal within days, guidelines call for starting Amphotericin B, paired with flucytosine for the first two weeks, as induction therapy, then stepping down to fluconazole for consolidation and maintenance once the patient stabilizes.

Step 4: Rule out the rest.

Itraconazole has poor penetration into the brain and is not used here. Fluconazole alone and flucytosine alone are both too weak or too resistance prone to be first line by themselves.

Step 5: Settle on the answer.

The induction drug that treats this infection is Amphotericin B.

Amphotericin B

Quick Tip: Cryptococcal meningitis needs a fast fungicidal drug first; amphotericin B is that drug, usually combined with flucytosine, then followed by fluconazole.

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44. The lymphocytopenia seen a few hours after administration of a large dose of prednisolone to a patient with lymphocytic leukemia is due to

- (A) Massive lymphocyte apoptosis
- (B) Bone marrow depression
- (C) Activation of cytotoxic cells
- (D) Stimulation of Natural killer cell activity

Correct Answer: (A) Massive lymphocyte apoptosis

SOLUTION:

Step 1: Note the timing.

The lymphocyte count drops within a few hours of the steroid dose, which is too fast to be explained by any change in bone marrow

production.

Step 2: Recall what steroids do to lymphocytes.

Glucocorticoids act directly on lymphocytes through their glucocorticoid receptors and switch on a death program inside the cell, known as apoptosis. This happens fast, within hours, and does not need new cells to be made or destroyed in the marrow.

Step 3: Apply this to leukemic cells.

In lymphocytic leukemia the circulating lymphocytes are themselves the malignant clone, and these cells are unusually rich in glucocorticoid receptors. So a big steroid dose kills large numbers of them at once, this is actually the basis for using steroids as part of leukemia chemotherapy protocols.

Step 4: Eliminate the other choices.

Bone marrow depression is a slow process taking days, not hours, so it does not fit. Steroids suppress, rather than activate, cytotoxic T cells and natural killer cells, so those two options describe the opposite of what steroids actually do.

Step 5: Conclude.

The fast fall in lymphocyte count comes from apoptosis of the leukemic lymphocytes.

Massive lymphocyte apoptosis

Quick Tip: Steroids trigger apoptosis in lymphocytes, and leukemic lymphocytes are highly sensitive to this effect.

45. Prolactin secretion will be inhibited by

- (A) Haloperidol
- (B) GABA
- (C) Neurophysin
- (D) Dopamine

Correct Answer: (D) Dopamine

SOLUTION:

Step 1: Recall the unusual control of prolactin.

Most anterior pituitary hormones are switched on by a hypothalamic releasing hormone, but prolactin is different, it is mainly kept switched off by an inhibiting factor.

Step 2: Name the inhibiting factor.

This prolactin inhibiting factor is dopamine, made by neurons in the hypothalamus and carried to the pituitary through the hypophyseal portal vessels.

Step 3: See how it works.

Dopamine attaches to D2 receptors on the lactotroph cells of the anterior pituitary and keeps prolactin release turned down. If dopamine signaling drops, for example when a drug blocks its receptor, prolactin rises instead.

Step 4: Check the other options against this logic.

Haloperidol blocks dopamine receptors, so it raises prolactin, the opposite of what the question asks. GABA is not the recognized prolactin inhibiting factor. Neurophysin only carries oxytocin and

vasopressin, it has nothing to do with prolactin.

Step 5: Conclude.

The hormone that inhibits prolactin secretion is dopamine.

Dopamine

Quick Tip: Dopamine is the hypothalamic prolactin inhibiting factor, acting on D2 receptors of lactotrophs.

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46. The production of cervical mucus is stimulated by

- (A) Progesterone
- (B) Estradiol
- (C) Estriol
- (D) Pregnenolone

Correct Answer: (B) Estradiol

SOLUTION:

Step 1: Think about what changes across the cycle.

Cervical mucus is watery and stretchy near ovulation and thick and sticky after it, so two different hormones must be pulling it in opposite directions.

Step 2: Assign the fertile phase hormone.

Just before ovulation, the growing follicle pours out estradiol. This hormone makes the cervical glands secrete more mucus and makes

that mucus thin, clear and stretchy, ideal for sperm to travel through.

Step 3: Assign the post ovulation hormone.

After ovulation the corpus luteum secretes progesterone, which thickens the mucus into a scanty plug that blocks the cervical canal.

Step 4: Place the other two hormones.

Estriol is a weak estrogen important mostly in pregnancy, not in the cyclical mucus pattern. Pregnenolone is just a precursor molecule used to build other steroid hormones, it has no direct glandular action.

Step 5: Conclude.

The hormone that stimulates cervical mucus production is estradiol.

Estradiol

Quick Tip: Estradiol makes cervical mucus abundant and thin around ovulation; progesterone does the opposite.

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47. A travelling nerve impulse does not depolarize the area behind it, because

- (A) It is hyperpolarized
- (B) It is refractory
- (C) It is not self propagating
- (D) The condition is always orthodromic

Correct Answer: (B) It is refractory

SOLUTION:

Step 1: Picture the moving impulse.

An action potential travels along the axon like a wave, and behind that wave the membrane has just finished firing.

Step 2: Look at the channels behind the wave.

Sodium channels that just opened and closed enter an inactivated state. In this state they will not open again, even with a strong stimulus, until the membrane potential returns close to resting level.

Step 3: Name this state.

This period where the channels refuse to reopen is called the refractory period, split into an absolute part where no stimulus works and a relative part where only an unusually strong stimulus works.

Step 4: Rule out the distractors.

The membrane is briefly hyperpolarized after firing, but that only makes it harder to reach threshold, it is not the direct reason the sodium channels refuse to reopen. Saying the impulse is not self propagating is simply false, since propagation is the defining feature of a nerve impulse. Calling the direction orthodromic just names the forward direction, it does not explain the blockade.

Step 5: Conclude.

The impulse cannot go backward because the membrane just behind it is refractory.

Refractory state

Quick Tip: The membrane behind an advancing impulse is refractory, its sodium channels cannot reopen right away.

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48. A 10 Degree Centigrade decrease in temperature causes a decrease in cerebral metabolic rate by

- (A) 10%
- (B) 30%
- (C) 50%
- (D) 70%

Correct Answer: (C) 50%

SOLUTION:

Step 1: Recall the temperature and metabolism link.

Cooling any tissue slows its chemical reactions, and the brain is no exception, its oxygen use falls as its temperature falls.

Step 2: Use the per degree rule.

Clinical teaching gives a working number, about 6 to 7 percent fall in cerebral oxygen consumption for every 1 degree Centigrade the brain cools.

Step 3: Scale this up to 10 degrees.

Compounding a 6 to 7 percent fall over 10 separate degree steps does not simply add to 60 to 70 percent, because each step acts on an already reduced value. Working through the compounding gives a

final metabolic rate close to half the starting value, so the drop over the full 10 degrees works out to about 50 percent.

Step 4: Connect this to practice.

This is exactly why cooling the brain by around 10 degree Centigrade during neurosurgery or after a cardiac arrest is described as roughly halving its oxygen needs, giving it better tolerance to a temporary drop in blood supply.

Step 5: Conclude, with a note on the source key.

The physiologically correct fall for a 10 degree Centigrade drop is about 50 percent. The scanned original marks 30 percent with an uncertain symbol next to it, which does not match the standard 6 to 7 percent per degree teaching, so the answer here is corrected to 50 percent.

50%

Quick Tip: Cerebral metabolic rate falls about 6-7% per degree Centigrade, so a 10 degree Centigrade drop nearly halves it.

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49. The laboratory report shows values of gonadotropin and ovarian hormone levels of the blood sample taken on day 20 of the menstrual cycle of a young woman. Whether her cycle was ovulatory or not may be validly assessed by the reported serum levels of

- (A) FSH
- (B) LH
- (C) Oestradiol
- (D) Progesterone

Correct Answer: (D) Progesterone

SOLUTION:

Step 1: Place day 20 in the cycle.

In a textbook 28 day cycle, day 20 sits about a week after ovulation, squarely in the luteal phase.

Step 2: Ask what is unique to this phase.

Once an egg is released, the empty follicle turns into the corpus luteum, and this structure is the only source of a large, sustained rise in progesterone during the cycle.

Step 3: Connect this to proof of ovulation.

Because progesterone only rises this way if a corpus luteum has formed, and a corpus luteum only forms after an egg has actually been released, a high day 20 progesterone is direct, reliable proof of ovulation. This is why clinicians order a day 21 progesterone test to check for ovulation.

Step 4: Compare with the other hormones.

FSH and LH both have their important peaks earlier in the cycle, around the time of ovulation itself, and are back to baseline by day 20, so they cannot confirm what already happened a week earlier.

Oestradiol rises with follicle growth in general and is not specific to a functioning corpus luteum, so it is a weaker marker than progesterone

for this purpose.

Step 5: Conclude.

The valid marker of ovulation at day 20 is serum progesterone.

Progesterone

Quick Tip: Mid luteal serum progesterone, around day 20 to 22, is the standard test to confirm ovulation.

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50. Which of the following is NOT a usual feature of right middle cerebral artery territory infarct?

- (A) Aphasia
- (B) Hemiparesis
- (C) Facial weakness
- (D) Dysarthria

Correct Answer: (A) Aphasia

SOLUTION:

This question tests whether you know which brain functions live in which hemisphere. The right middle cerebral artery feeds the right hemisphere, and in nearly everyone the right hemisphere does not run language.

1. **Aphasia:** This is a language problem. Language sits in the dominant hemisphere, the left side in almost all people. A right

sided stroke does not usually touch this area, so aphasia is not expected here. This is the odd one out.

2. **Hemiparesis:** Motor fibers from the right hemisphere cross over and control the left limbs. A right MCA infarct causes left sided weakness, so this is expected.
3. **Facial weakness:** The same crossed pathway controls the lower face. A right sided stroke gives left lower facial droop, so this is expected too.
4. **Dysarthria:** This is slurred speech from weak articulation muscles, not a language problem. Both hemispheres feed the muscles of speech, so dysarthria can follow a stroke on either side, including the right.

Three of the four, hemiparesis, facial weakness and dysarthria, are standard findings after a right MCA infarct. Aphasia stands apart because it needs damage to the language dominant hemisphere, which for a right sided stroke is usually spared.

Let's summarize:

- Right hemisphere strokes give left sided weakness and left facial droop through crossed motor pathways.
- Language lives in the left hemisphere in almost everyone, so right sided strokes typically spare speech content, even though articulation (dysarthria) can still be affected.

So the feature that is NOT usual in a right MCA territory infarct is aphasia.

Quick Tip: Language (aphasia) is a dominant, usually left hemisphere, function, not a right hemisphere one.

51. A 5 year old child is scheduled for strabismus (squint) correction. Induction of anesthesia is uneventful. After conjunctival incision, as the surgeon grasps the medial rectus, the anesthesiologist looks at the cardiac monitor. Why do you think he did that?

- (A) He wanted to check the depth of anaesthesia.
- (B) He wanted to be sure that the blood pressure did not fall.
- (C) He wanted to see if there was an oculocardiac reflex.
- (D) He wanted to make sure there were no ventricular dysrhythmias which normally accompany incision.

Correct Answer: (C) He wanted to see if there was an oculocardiac reflex.

SOLUTION:

This question checks whether you know what happens when the eye muscles are pulled during strabismus surgery, a classic trigger for a specific heart reflex.

1. **Check the depth of anaesthesia:** Depth is judged from several signs together, movement, blood pressure trend, and pupil size, not by watching the monitor at the exact moment a muscle is grasped. This does not fit the timing described.
2. **Make sure blood pressure did not fall:** A generic blood pressure drop is not specifically linked to grasping an eye muscle. It does not explain why the anesthesiologist reacted right at that moment.
3. **See if there was an oculocardiac reflex:** Pulling the medial rectus is the classic trigger for this vagal reflex, which can cause

the heart rate to drop suddenly. Watching the monitor exactly when the muscle is grasped fits this perfectly.

4. **Watch for ventricular dysrhythmias from the incision:** A simple conjunctival incision does not normally trigger ventricular arrhythmias. This option invents a link that does not exist in practice.

The oculocardiac reflex is well documented in eye muscle surgery, especially in children, and can appear within seconds of traction, which matches the scenario exactly.

Let's summarize:

- Traction on extraocular muscles, most strongly the medial rectus, sends a signal via the trigeminal nerve that triggers a vagal slowing of the heart.
- This oculocardiac reflex is common in pediatric strabismus surgery and needs quick recognition so traction can be released.

So the anesthesiologist checked the monitor to look for signs of the oculocardiac reflex.

Quick Tip: Traction on the medial rectus can trigger the oculocardiac (Aschner) reflex, causing sudden bradycardia.

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52. A 55 year old male accident victim in casualty urgently needs blood. The blood bank is unable to determine his ABO group, as his red cell group and plasma group do not match. Emergency transfusion of the patient should be with

- (A) RBC corresponding to his red cell group and colloids/crystalloid.
- (B) Whole blood corresponding to his plasma group.
- (C) O positive RBC and colloids/crystalloid.
- (D) AB negative whole blood.

Correct Answer: (C) O positive RBC and colloids/crystalloid.

SOLUTION:

This question is about what to transfuse when the blood bank cannot pin down a patient's ABO group because his forward and reverse typing disagree, yet he needs blood urgently.

1. **RBC matching his red cell group, plus fluids:** This assumes we already trust the red cell typing result, but that is the very result under doubt here. Using it could still trigger a reaction if the true group is different.
2. **Whole blood matching his plasma group:** Whole blood brings donor plasma and donor cells together. If the plasma typing itself is unreliable, matching to it is risky, and whole blood is not the modern emergency standard anyway.
3. **O positive RBC and colloids/crystalloid:** Group O red cells have no A or B antigen on their surface, so no anti-A or anti-B antibody, in a recipient of any group, can attack them. Volume is made up separately with fluids that carry no blood group antigens at all. This is the accepted emergency approach.
4. **AB negative whole blood:** AB plasma is the universal plasma donor because it has no anti-A or anti-B antibodies, but whole blood also carries AB red cells, which do carry both antigens, so this is not the safe universal red cell choice.

Because the red cell group is genuinely uncertain, the only component that is safe no matter what his true group turns out to be is O group red cells, backed up with plain fluids for volume.

Let's summarize:

- O group red cells carry no ABO antigens, so they are compatible with any recipient plasma.
- Volume replacement in this emergency comes from colloids or crystalloids, not from unmatched type specific blood.

So the correct emergency transfusion here is O positive red cells with colloids or crystalloids.

Quick Tip: When ABO grouping is unclear, give O group RBCs, which carry no A or B antigen, with fluids for volume.

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53. Although more than 400 blood groups have been identified, the ABO group system remains the most important in clinical medicine because

- (A) It was the first blood group system to be discovered.
- (B) It has four different blood groups: A, B, AB, O(h).
- (C) ABO(h) antigens are present in most body tissues and fluids.
- (D) ABO(h) antibodies are invariably present in plasma when a person's RBCs lack the corresponding antigen.

Correct Answer: (D) ABO(h) antibodies are invariably present in plasma when a person's RBCs lack the corresponding antigen.

SOLUTION:

The question asks why ABO, out of more than 400 known blood group systems, remains the one doctors worry about most in daily practice.

1. **It was discovered first:** Being discovered first, by Karl Landsteiner, is a historical fact, but history alone does not make a system clinically dangerous today.
2. **It has four groups, A, B, AB, O(h):** This just describes the system's structure. Many other blood group systems also have multiple types, so this does not explain its special clinical weight.
3. **ABO(h) antigens are on most tissues:** True, and useful for organ transplant matching, but tissue distribution alone is not why a mismatched transfusion causes an immediate reaction.
4. **ABO(h) antibodies are always present when the matching antigen is missing:** This is the real reason. Almost everyone, from early childhood, already carries anti-A and/or anti-B antibody against whichever antigen their own red cells lack, with no prior transfusion needed to develop it.

Because these antibodies exist naturally and in high amounts, a first ever ABO incompatible transfusion can trigger a rapid, severe hemolytic reaction, which is not true in the same way for most of the other 400 systems, where antibodies usually need a prior exposure to form.

Let's summarize:

- ABO antibodies are naturally occurring and present from early life, unlike antibodies in most other blood group systems.
- This is why every single transfusion is ABO checked, making it the clinically dominant system.

So the ABO system stays most important because its antibodies are reliably present whenever the matching antigen is absent from the red cells.

Quick Tip: Anti-A and anti-B antibodies occur naturally, without prior exposure, which makes ABO mismatch dangerous immediately.

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54. The neurons may get irreversibly damaged if exposed to significant hypoxia for

- (A) 8 minutes
- (B) 2 minutes
- (C) 30 seconds
- (D) 15 seconds

Correct Answer: (A) 8 minutes

SOLUTION:

This question is about the time limit after which oxygen starved neurons cannot recover, a number that different textbooks state slightly differently.

1. **8 minutes:** This is the option marked in the original answer key. It falls in the same broad range, several minutes, as commonly quoted teaching values, though it runs a bit longer than the tightest textbook figures.
2. **2 minutes:** Some sources treat this as the point where reversible stress starts to build, but most standard teaching still allows a little more time than this before damage becomes permanent.

3. **30 seconds:** Far too short. This length of oxygen loss usually causes only brief, reversible symptoms such as light headedness, not lasting neuron death.
4. **15 seconds:** Also far too short for permanent injury, this is closer to the time it takes to feel dizzy from a sudden drop in brain blood flow, not to cause fixed damage.

The commonly quoted textbook range for irreversible cortical neuron injury is about 4 to 6 minutes of complete anoxia at normal body temperature. None of the four options match this exactly, and the source paper itself marks its own chosen answer with a question mark, noting the 3 to 5 minute range as the more standard figure.

Let's summarize:

- Neurons have almost no energy reserve and depend on constant oxygen delivery, so anoxia damages them faster than most other tissues.
- Most texts place the irreversible injury point at about 4 to 6 minutes, though this exact paper's key and annotation disagree slightly on the number.

Following the original key, the marked answer here is 8 minutes, with the caveat that standard teaching favors a shorter window of about 4 to 6 minutes.

Quick Tip: Standard teaching puts irreversible neuron damage at about 4 to 6 minutes of complete anoxia; this paper's key is self flagged as uncertain.



55. A 41 year old woman presented with a history of aches and pains all over the body and cannot sleep because of the illness, and has lost her appetite as well. She has lack of interest in work and doesn't like to meet friends and relatives. She denies feelings of sadness. Her most likely diagnosis is

- (A) Somatoform pain disorder.
- (B) Major depression.
- (C) Somatization disorder.
- (D) Dissociative disorder.

Correct Answer: (A) Somatoform pain disorder.

SOLUTION:

This vignette gives a woman with body wide pain, poor sleep, poor appetite, loss of interest, and social withdrawal, but she says she does not feel sad. The question wants the diagnosis that fits this picture best.

1. **Somatoform pain disorder:** Here pain, without a clear physical cause, is the main and persistent complaint, and the other symptoms are read as accompanying it. This is the answer given in the original key.
2. **Major depression:** Poor sleep, poor appetite, and loss of interest are core depressive symptoms, and denying sadness while having these features is sometimes called masked depression. Many exam takers argue for this option instead, which is why this particular question is a known point of debate.
3. **Somatization disorder:** This needs a long standing pattern of many physical complaints spread across different body systems, usually starting before age 30. The vignette gives one dominant

complaint, body pain, without that broader multi system, multi year history, so it fits less well.

4. **Dissociative disorder:** This involves a break in memory, identity, or awareness of surroundings. Nothing in the vignette describes memory loss or altered identity, so this does not fit.

The case sits at the overlap between pain focused somatoform presentations and depression hiding behind physical symptoms, which is exactly why answer choices differ across sources for this question.

Let's summarize:

- Pain as the chief, persistent complaint without adequate physical explanation points toward somatoform pain disorder.
- The same vegetative symptoms could also support masked depression, making this a debated question in practice.

Following the original paper's answer key, the diagnosis here is somatoform pain disorder.

Quick Tip: Widespread unexplained pain as the chief complaint points to somatoform pain disorder; note this vignette is a classically debated one.

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56. Lack of insight is not a feature of

- (A) Panic disorder.
- (B) Schizophrenia.
- (C) Mania.
- (D) Reactive psychosis.

Correct Answer: (A) Panic disorder.

SOLUTION:

Insight is the patient's own recognition that they are ill. This question asks which of the four listed conditions usually leaves that recognition intact.

1. **Panic disorder:** This is an anxiety condition, not a psychotic one. During an attack the person is terrified, but they still know they are having a panic attack and are not losing touch with reality. Insight stays intact, making this the exception.
2. **Schizophrenia:** A classic psychotic disorder. Delusions and hallucinations feel completely real to the patient, so they commonly do not believe they are ill. Insight is usually poor.
3. **Mania:** Especially in severe episodes, patients often see their racing thoughts, grand plans, or reduced need for sleep as normal or even a sign of being unusually capable, not as illness. Insight is often reduced.
4. **Reactive psychosis:** By definition a psychotic state triggered by severe stress. Like other psychoses, it usually comes with a loss of accurate self awareness about being unwell.

Three of the four options are psychotic conditions, and psychosis as a category tends to blur a patient's own sense that something is wrong. Panic disorder does not carry that reality distortion.

Let's summarize:

- Loss of insight is a hallmark of psychotic conditions: schizophrenia, mania, and reactive psychosis.
- Panic disorder keeps reality testing intact, so patients usually stay aware they are anxious, not psychotic.

So the condition where lack of insight is not a usual feature is panic disorder.

Quick Tip: Psychotic disorders, schizophrenia, mania, reactive psychosis, impair insight; panic disorder, a neurotic condition, does not.

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57. A 25 year old man with a psychotic illness was treated with haloperidol 30 mg/day. On the third day he developed pacing and an inability to sit still at one place. Which medication is likely to help?

- (A) Phenytoin
- (B) Propranolol
- (C) Methylphenidate
- (D) Trihexyphenidyl

Correct Answer: (B) Propranolol

SOLUTION:

The question describes a man started on a high dose of haloperidol who, within three days, starts pacing and cannot sit still. This is a well-known adverse effect pattern, and the job here is to name it correctly before picking a drug to treat it.

1. **Phenytoin:** This is used for seizures. It has no place in treating a movement side effect from an antipsychotic, so it does not fit.
2. **Propranolol:** A beta blocker. It is the treatment most consistently shown to relieve the inner restlessness of akathisia, so this is the right choice.

3. **Methylphenidate**: A stimulant drug. Since the patient is already agitated and unable to keep still, adding a stimulant would only add fuel to the fire.
4. **Trihexyphenidyl**: An anticholinergic. It is very useful for acute dystonia and for drug-induced parkinsonism, but it does not treat akathisia reliably, since akathisia is not primarily a cholinergic problem.

The picture of pacing and restlessness starting soon after a high dose of a potent antipsychotic like haloperidol is classic akathisia. Among the four choices, propranolol is the one with proven benefit for this specific side effect.

Let's summarize:

- Akathisia is an extrapyramidal side effect distinct from dystonia and parkinsonism.
- Beta blockers such as propranolol are the preferred treatment for akathisia.
- Anticholinergics like trihexyphenidyl are better suited to dystonia and parkinsonism, not akathisia.

So the correct answer is propranolol.

Quick Tip: Pacing and restlessness on haloperidol is akathisia, not dystonia or parkinsonism, and beta blockers work best for it.



58. A 65 year old male is brought to the outpatient clinic with a one year illness marked by forgetfulness, visual hallucinations, suspiciousness, personality decline, poor self care and progressive deterioration in his condition. His Mini Mental Status Examination (MMSE) score is 10. What is his most likely diagnosis?

- (A) Dementia
- (B) Schizophrenia
- (C) Mania
- (D) Depression

Correct Answer: (A) Dementia

SOLUTION:

This question gives an elderly man with a year-long, worsening illness that includes forgetfulness, visual hallucinations, suspicion of others, a change in personality, and loss of self-care skills, along with a very low MMSE score of 10. The task is to pick the diagnosis that best fits this pattern.

1. **Dementia:** A chronic, progressive loss of memory and thinking skills in an older adult, often with hallucinations and personality change added in. This matches the case closely, including the very low MMSE.
2. **Schizophrenia:** Typically begins in the teens or twenties, and its hallucinations are not paired with a year of steady memory decline and a crashing MMSE score in a 65 year old.
3. **Mania:** Presents with high energy, elevated or irritable mood, and less need for sleep, none of which are described here.
4. **Depression:** Can slow thinking and cause forgetfulness in older people, but visual hallucinations and a full year of progressive

deterioration fit dementia far better than a mood disorder.

Putting the age, the one year progressive course, the low MMSE, and the mix of memory loss with hallucinations and personality change together, dementia is the best fit.

Let's summarize:

- A slow, one year cognitive decline in an elderly patient with a low MMSE score signals dementia.
- Visual hallucinations and personality change can occur within dementia, especially dementia with Lewy bodies.
- Schizophrenia, mania, and depression do not explain this steady cognitive fall.

So the correct answer is dementia.

Quick Tip: Progressive memory loss over a year with a low MMSE score in an elderly patient points to dementia.

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59. A 16 year old girl was brought to the psychiatric emergency after she slashed her wrists in a suicide attempt. On enquiry, her father revealed that she had made several such wrist-slashing attempts in the past, mostly in response to trivial fights at home. She also has marked mood fluctuations with a pervasive pattern of unstable interpersonal relationships. What is the most probable diagnosis?

- (A) Borderline personality disorder
- (B) Major depression
- (C) Histrionic personality disorder
- (D) Adjustment disorder

Correct Answer: (A) Borderline personality disorder

SOLUTION:

A 16 year old girl has cut her wrists more than once, often after small fights at home, and also shows big mood swings and a long pattern of rocky relationships. The question asks which diagnosis fits this picture best.

1. **Borderline personality disorder:** Marked by repeated impulsive self-harm, sharp mood swings, and a lasting pattern of unstable relationships, which is exactly what is described here.
2. **Major depression:** Usually causes a suicide attempt during a low mood episode, but does not by itself explain a repeated pattern of self-harm set off by minor quarrels, or ongoing relationship instability.
3. **Histrionic personality disorder:** Centres on being overly dramatic and craving attention, not on repeated cutting behaviour and unstable relationships.
4. **Adjustment disorder:** A reaction limited to one clear stressor over a short time, not a long repeating pattern seen across many episodes.

The repeated wrist-slashing tied to trivial conflicts, along with mood instability and unstable relationships that run through her life, are the hallmark triad of borderline personality disorder.

Let's summarize:

- Recurrent impulsive self-harm is a core feature of borderline personality disorder.
- Unstable mood and unstable relationships that persist over time support this diagnosis over a single depressive episode.

- Histrionic personality disorder and adjustment disorder do not match the repeated self-harm pattern described.

So the correct answer is borderline personality disorder.

Quick Tip: Repeated impulsive self-harm plus unstable mood and relationships in a teenager points to borderline personality disorder.

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60. High resolution CT (HRCT) of the lung is a specialized CT technique for greater detail of the lung parenchyma. It utilizes:

- (A) Special lung filters
- (B) Thick collimation
- (C) Bone algorithm for image reconstruction
- (D) Large field of view

Correct Answer: (C) Bone algorithm for image reconstruction

SOLUTION:

The question asks what technical trick lets HRCT of the lung show finer detail of the lung tissue than an ordinary chest CT scan.

1. **Special lung filters:** This is not a defined, standard part of how HRCT is set up, so it does not fit.
2. **Thick collimation:** Thicker slices average more tissue into each pixel and blur fine detail, which works against the whole point of HRCT, so this is wrong.
3. **Bone algorithm for image reconstruction:** This sharp, high spatial frequency algorithm boosts edge detail, the same way it is used to bring out fine bone structure, and this same sharpening is

exactly what HRCT needs to show the lung's fine architecture, so this fits.

4. **Large field of view:** A bigger field of view means bigger pixels and less detail per unit area, the opposite of what HRCT is trying to achieve, so this is wrong.

HRCT gets its extra detail from thin slices, a small targeted field of view, and a sharp bone-style reconstruction algorithm. Among the four choices given, the bone algorithm for image reconstruction is the one that correctly describes part of the HRCT technique.

Let's summarize:

- HRCT uses thin collimation, not thick collimation.
- It uses a small field of view, not a large one.
- The sharp, edge-enhancing bone algorithm is what gives HRCT its fine parenchymal detail.

So the correct answer is bone algorithm for image reconstruction.

Quick Tip: HRCT needs thin slices, a small field of view, and a sharp bone-type reconstruction algorithm, not thick slices or a large field.

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61. Which of the following is NOT in the differential diagnosis of an anterior mediastinal mass?

- (A) Teratoma
- (B) Neurogenic tumour
- (C) Thymoma
- (D) Lymphoma

Correct Answer: (B) Neurogenic tumour

SOLUTION:

This question tests knowledge of which masses typically sit in the anterior part of the mediastinum, and which one is instead found elsewhere.

1. **Teratoma:** A germ cell tumour that is one of the standard anterior mediastinal masses, so it belongs on the list.
2. **Neurogenic tumour:** These tumours grow from nerve tissue near the spine, which places them in the posterior mediastinum, not the anterior compartment, so this one does not belong.
3. **Thymoma:** Comes from the thymus, an anterior mediastinal structure, so it belongs on the list.
4. **Lymphoma:** Frequently presents as anterior mediastinal or hilar nodal disease, so it belongs on the list too.

A simple way to remember the anterior mediastinum causes is the "4 Ts": thymoma, teratoma or other germ cell tumour, thyroid (retrosternal goitre), and terrible lymphoma. Neurogenic tumours fall outside this group because they arise from posterior mediastinal nerve structures.

Let's summarize:

- Anterior mediastinal masses follow the 4 Ts: thymoma, teratoma, thyroid, terrible lymphoma.
- Neurogenic tumours are the classic posterior mediastinal masses, arising near the spine and sympathetic chain.

So the correct answer is neurogenic tumour.

Quick Tip: Remember the anterior mediastinum 4 Ts (thymoma, teratoma, thyroid, terrible lymphoma); neurogenic tumours belong to the posterior compartment.

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62. A dense persistent nephrogram may be seen in all of the following except:

- (A) Acute ureteral obstruction
- (B) Systemic hypertension
- (C) Severe hydronephrosis
- (D) Dehydration

Correct Answer: (C) Severe hydronephrosis

SOLUTION:

A dense, persistent nephrogram happens when contrast stays trapped and concentrated inside the kidney's tubules for longer than normal. The question wants the one option among the four that does not typically produce this picture.

1. **Acute ureteral obstruction:** A sudden block raises pressure inside the tubules and slows the flow of filtrate, trapping contrast and building a dense, lasting nephrogram. This is a well-known cause.
2. **Systemic hypertension:** When blood flow to a kidney is slowed, such as in renovascular hypertension, contrast lingers and becomes concentrated, giving a dense and delayed nephrogram, which is actually used as a clue to renal artery narrowing. This is a recognised cause too.

3. **Severe hydronephrosis:** Long standing, severe hydronephrosis crushes and thins the renal cortex over time, so the kidney has little functioning tissue left to concentrate contrast. Instead of a dense nephrogram, a chronically damaged kidney like this usually shows a faint, thin nephrogram, which makes this option the exception.
4. **Dehydration:** Less blood flow plus more water reabsorption in the tubules concentrates the contrast, again giving a dense persistent nephrogram. This is a recognised cause.

Three of the four choices (acute obstruction, hypertension with reduced flow, and dehydration) share the same underlying mechanism of slowed, concentrated tubular flow that produces a dense nephrogram. Severe, chronic hydronephrosis instead works by destroying functioning kidney tissue, which weakens rather than strengthens the nephrogram.

Let's summarize:

- Dense persistent nephrograms come from slow, concentrated tubular flow, as in obstruction, dehydration, and reduced renal blood flow states like hypertension.
- Chronic severe hydronephrosis instead destroys renal parenchyma, giving a faint nephrogram rather than a dense one.

So the correct answer is severe hydronephrosis. The original source for this question did not commit to a stated answer key, so this conclusion is reasoned from nephrogram physiology.

Quick Tip: Acute obstruction, hypertension-related slow flow, and dehydration all concentrate contrast; chronic severe hydronephrosis instead damages the kidney and weakens the nephrogram.

63. Which of the following imaging modalities is most sensitive to detect early renal tuberculosis?

- (A) IV urography
- (B) Ultrasound
- (C) Computed tomography
- (D) MRI

Correct Answer: (C) Computed tomography

SOLUTION:

The question asks which of four imaging tests best catches renal tuberculosis at an early stage, when the changes in the kidney are still small.

1. **IV urography:** Shows the collecting system through excreted contrast and was the classic test used for years, but it depends on the calyces being distorted enough to show up, so very early, subtle lesions can be missed.
2. **Ultrasound:** Handy and radiation-free, but it depends heavily on the operator's skill and is not fine-grained enough to reliably catch the earliest small lesions of renal TB.
3. **Computed tomography:** Directly images the renal cortex and medulla in cross-section, so it can pick up tiny papillary necrosis, calyceal wall changes, thinning of the cortex, and small calcifications well before they are obvious on a urogram. This makes it the most sensitive of the four for early disease.
4. **MRI:** Offers excellent soft tissue detail but is weaker than CT at showing the small calcifications that are an important early clue in renal TB.

Because CT looks directly at the kidney tissue itself rather than only the shape of the collecting system, it finds the small early lesions of renal tuberculosis sooner and more reliably than IV urography, ultrasound, or MRI.

Let's summarize:

- IV urography only shows changes once the collecting system is distorted enough to be visible.
- CT directly visualises the renal parenchyma and catches small papillary, cortical, and calcification changes earlier.
- Ultrasound and MRI are useful but less sensitive than CT for the earliest renal TB findings.

So the correct answer is computed tomography. The original 2002 paper marked IV urography, which was the older standard, but current practice with CT urography favours CT as more sensitive for early disease.

Quick Tip: CT images the kidney parenchyma directly and picks up small papillary and calcification changes of renal TB earlier than a urogram.

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64. A young man with tuberculosis presents with massive recurrent hemoptysis. For angiographic treatment, which vascular structure should be evaluated first?

- (A) Pulmonary artery
- (B) Bronchial artery
- (C) Pulmonary vein
- (D) Superior vena cava

Correct Answer: (B) Bronchial artery

SOLUTION:

This question tests where massive bleeding in a TB patient usually comes from, and which vessel an interventional radiologist checks before treating it. Let's go through each option.

1. **Pulmonary artery:** This is the low pressure circuit that oxygenates blood in the alveoli. It is rarely the source of major hemoptysis, and TB does not typically damage it enough to bleed heavily.
2. **Bronchial artery:** These vessels branch off the aorta, run at systemic pressure, and supply the airways and any cavity wall. Long-standing TB makes them thick and tortuous, and they are the source of bleeding in the vast majority of massive hemoptysis cases. This is the vessel checked and treated first.
3. **Pulmonary vein:** This carries oxygenated blood away from the lung toward the heart. It plays no real part in hemoptysis and is not evaluated for embolization.
4. **Superior vena cava:** This is a large systemic vein draining the upper body into the right atrium. It has nothing to do with lung bleeding.

Since the bronchial arteries are the usual bleeding source and the vessel actually embolized, they are the structure evaluated first on angiography.

Let's summarize:

- Bronchial arteries run at systemic pressure and are the source of most massive hemoptysis, especially in TB.

- Bronchial artery embolization is the standard first-line treatment, so these arteries are studied first.

Therefore, the answer is the bronchial artery.

Quick Tip: In massive hemoptysis, the high-pressure bronchial circulation, not the pulmonary circulation, is usually the culprit and the first target on angiography.

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65. Which of the following imaging modalities is most sensitive for evaluation of extra-adrenal pheochromocytoma?

- (A) USG
- (B) CT
- (C) MRI
- (D) MIBG scan

Correct Answer: (D) MIBG scan

SOLUTION:

Step 1: Understanding the Question:

We need the imaging test that best finds pheochromocytoma tissue lying outside the adrenal gland, which can be anywhere in the sympathetic chain.

Step 2: Key Formula or Approach:

Anatomical scans, USG, CT, and MRI, only show structure in the region they cover. A functional scan instead follows a tracer that is taken up specifically by the tumor cells, wherever they sit in the body.

Step 3: Detailed Explanation:

MIBG behaves like norepinephrine and is actively absorbed by chromaffin cells through their noradrenaline transporter. A radiolabeled MIBG scan therefore surveys the whole body in one go and lights up any chromaffin tissue, including small, multiple, or ectopic extra-adrenal tumors that a CT of just the abdomen could easily miss. USG has poor sensitivity outside the adrenal bed and for small lesions. CT and MRI show anatomy well but cannot tell functioning tumor tissue from a look-alike mass, and both are usually done as regional studies rather than whole-body surveys.

Step 4: Final Answer:

MIBG scan is the most sensitive of the four options for extra-adrenal pheochromocytoma because it is a whole-body, tumor-specific functional test.

Quick Tip: Extra-adrenal or multiple pheochromocytomas need a whole-body, tissue-specific scan, not just a regional anatomical one.

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66. A 15 year old boy has 10-12 partial complex seizures per day despite an adequate 4-drug antiepileptic regimen. He gives a history of repeated high grade fever in childhood. MRI done for the epilepsy protocol shows a normal brain scan. What should be the best non-invasive strategy to make a definite diagnosis, so that he can be prepared to undergo epilepsy surgery?

- (A) Interictal scalp EEG
- (B) Video EEG
- (C) Interictal 18F-FDG PET
- (D) Video EEG with ictal 99mTc-HMPAO brain SPECT

Correct Answer: (D) Video EEG with ictal 99mTc-HMPAO brain SPECT

SOLUTION:

This question is about presurgical workup for drug-resistant epilepsy when the MRI is normal. We need the non-invasive test combination that best finds the seizure onset zone. Let's look at each option.

1. **Interictal scalp EEG:** Recorded between seizures, it can show spikes suggesting an irritable area of cortex, but spikes do not always sit exactly over the true seizure onset zone, so this alone is not definite enough for surgical planning.
2. **Video EEG:** This captures the actual clinical seizure and its scalp EEG pattern, confirming the semiology and rough region of onset, but scalp electrodes can struggle to localize precisely, especially with a normal MRI and possibly deep or subtle pathology.
3. **Interictal 18F-FDG PET:** Done between seizures, it shows an area of low glucose use around the epileptic focus. It is useful, but the hypometabolic zone it shows is usually larger and less sharply defined than the actual onset zone.
4. **Video EEG with ictal 99mTc-HMPAO SPECT:** The tracer is injected right when the seizure starts, catching the sudden rise in blood flow at the true onset zone before it fades, and video EEG times this precisely against the clinical and electrical seizure

onset. Combined, this pairing gives the sharpest, most reliable non-invasive localization when MRI is unremarkable.

Because it captures the transient ictal blood flow surge together with exact electro-clinical timing, video EEG with ictal HMPAO SPECT is the best strategy here.

Let's summarize:

- Interictal tests (scalp EEG, FDG-PET) give useful but blurrier localization.
- Ictal SPECT paired with video EEG captures the seizure onset zone at its sharpest, which is why it is chosen in MRI-negative, drug-resistant epilepsy.

So the correct answer is video EEG with ictal ^{99m}Tc -HMPAO brain SPECT.

Quick Tip: When MRI is normal in drug-resistant epilepsy, injecting the tracer right at seizure onset (ictal SPECT) plus video EEG gives the sharpest localization.

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67. Which of the following is the most radiosensitive phase of the cell cycle?

- (A) G₁
- (B) S
- (C) G₂
- (D) M

Correct Answer: (D) M

SOLUTION:

Step 1: Understanding the Concept:

Radiosensitivity changes across the cell cycle because the cell's ability to repair DNA damage and the state of its chromatin both change from phase to phase.

Step 2: Key Formula or Approach:

Think of it in terms of repair opportunity. Whichever phase gives the cell the least time or tools to fix damaged DNA before something critical happens (like chromosome separation) will be the most sensitive phase, and whichever phase gives the most repair capacity will be the most resistant.

Step 3: Detailed Explanation:

In late S phase, a fresh copy of DNA sits right next to the original strand, so homologous recombination repair works efficiently, making this the most radioresistant window. G1 has moderate sensitivity. G2 is fairly sensitive as the cell readies its machinery for division. M phase is where the chromatin is packed tightly into chromosomes and the cell is committed to dividing with essentially no pause for repair, which is why it takes the biggest hit from radiation and is ranked the most radiosensitive phase overall.

Step 4: Final Answer:

The M phase of the cell cycle is the most radiosensitive, since dividing cells with condensed chromatin have the least chance to repair radiation damage.

Quick Tip: Cells caught mid-division, with condensed chromatin and no

time to repair, are hit hardest by radiation.

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68. X-rays are produced when

- (A) Electron beam strikes the nucleus of the atom
- (B) Electron beam strikes the anode
- (C) Electron beam reacts with the electromagnetic field
- (D) Electron beam strikes the cathode

Correct Answer: (B) Electron beam strikes the anode

SOLUTION:

This question checks the basic working of an X-ray tube, and where inside it the X-rays actually come from. Let's look at each option.

1. **Electron beam strikes the nucleus of the atom:** A direct hit on the nucleus is not what happens. X-rays form when an electron swings close to a nucleus and is deflected and slowed by its field, not from a collision with the nucleus itself.
2. **Electron beam strikes the anode:** The anode is the target electrode, usually tungsten, that the accelerated electrons crash into. This impact converts kinetic energy into heat and, for a small share, into bremsstrahlung and characteristic X-rays. This is the correct source.
3. **Electron beam reacts with the electromagnetic field:** There is no such external field reaction in standard X-ray tube operation, so this does not describe the real mechanism.
4. **Electron beam strikes the cathode:** The cathode is where electrons are boiled off from a heated filament, it is the source of the beam, not its target, so no X-rays are made there.

Since X-rays are generated at the point of impact of the accelerated electrons, which is the anode, that is the correct choice.

Let's summarize:

- The cathode releases electrons, the anode is what they strike.
- X-rays come from the sudden deceleration of electrons at the anode (bremsstrahlung) and from inner-shell electron transitions there (characteristic radiation).

So X-rays are produced when the electron beam strikes the anode.

Quick Tip: Electrons are emitted from the cathode but X-rays are generated only when they slam into the anode target.

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69. An eight year old boy presents with swelling in the left eye of 3 months duration. Examination reveals proptosis of the left eye with preserved vision. The right eye is normal. CT scan reveals an intraorbital, extraconal mass lesion. Biopsy reveals embryonal rhabdomyosarcoma. Metastatic workup is normal. The standard line of treatment is

- (A) Chemotherapy only
- (B) Wide local excision
- (C) Enucleation
- (D) Chemotherapy and radiotherapy

Correct Answer: (D) Chemotherapy and radiotherapy

SOLUTION:

Step 1: Understanding the Question:

A child has a biopsy-proven embryonal rhabdomyosarcoma confined

to the orbit, with normal vision and no spread elsewhere, and we need the standard treatment for this exact situation.

Step 2: Key Formula or Approach:

Modern pediatric oncology protocols for localized rhabdomyosarcoma favor organ-sparing treatment whenever the tumor is chemosensitive and radiosensitive, saving radical surgery for tumors that cannot be controlled any other way.

Step 3: Detailed Explanation:

Orbital rhabdomyosarcoma responds strongly to combination chemotherapy, which shrinks the tumor and covers micrometastatic disease, and to radiotherapy, which mops up residual local disease in the orbit. Together these achieve excellent local control while keeping the eye and useful vision intact, which matches this boy's presentation of preserved vision. Chemotherapy alone leaves local disease under-treated. Wide excision is not feasible in the confined orbital space without major functional loss, and it is unnecessary given how well this tumor responds to chemoradiation. Enucleation is kept in reserve for a blind, disfigured, or unsalvageable eye, not for a case where vision is still good.

Step 4: Final Answer:

The standard line of treatment for this boy's localized orbital embryonal rhabdomyosarcoma is chemotherapy combined with radiotherapy.

Quick Tip: Orbital rhabdomyosarcoma responds well to chemo and radiation together, so the eye and vision can usually be saved without surgery.

70. Which morphological type of cataract is most visually handicapping?

- (A) Cortical
- (B) Nuclear
- (C) Posterior subcapsular
- (D) Zonular

Correct Answer: (C) Posterior subcapsular

SOLUTION:

This question is about which type of cataract causes the greatest visual disability for its size. The key idea is where each type sits inside the lens relative to the visual axis. Let's check each option.

1. **Cortical:** Starts as spoke-like opacities at the edge of the lens cortex. Since the central visual axis is spared in the early stages, vision often stays fairly good until the spokes grow inward, so this type is slower to disable vision.
2. **Nuclear:** Affects the central lens core and worsens gradually over years. It mostly blurs distance vision and can even improve near vision for a while as the lens becomes more powerful, so it takes a long time to become truly handicapping.
3. **Posterior subcapsular:** Sits right where light rays from the whole pupil converge on their way to the retina, exactly on the visual axis. Even a tiny opacity here blocks and scatters a large share of incoming light, causing marked glare and poor near vision, especially in bright light when the pupil is small. This makes it the most disabling type relative to its size.
4. **Zonular:** A specific layered opacity, often congenital or linked to a metabolic problem, that does not typically cause the same

severe, rapid visual handicap as a posterior subcapsular opacity.

Because it blocks the light path exactly at the visual axis, a posterior subcapsular cataract causes disproportionately severe visual handicap even when small.

Let's summarize:

- Location relative to the visual axis matters more than the overall size of the opacity.
- Posterior subcapsular cataracts sit right at the point of light convergence, causing early glare and poor near vision.

So the most visually handicapping type is the posterior subcapsular cataract.

Quick Tip: A small opacity right on the visual axis, where light rays converge, causes bigger visual trouble than a similar sized opacity elsewhere in the lens.

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71. The standard sutureless cataract surgery done with phacoemulsification and a foldable IOL has an incision of:

- (A) 1 mm - 1.5 mm
- (B) 2 mm - 2.5 mm
- (C) 3 mm - 3.5 mm
- (D) 3.5 mm - 4 mm

Correct Answer: (B) 2 mm - 2.5 mm

SOLUTION:

This question checks the incision size used in modern sutureless cataract surgery done with phacoemulsification and a foldable lens.

1. **1 mm - 1.5 mm:** Too small for the standard phaco probe and sleeve to pass through safely; this range belongs to special micro incision techniques, not the routine procedure.
2. **2 mm - 2.5 mm:** Wide enough for the phaco tip and sleeve, yet narrow enough that the corneal wound seals itself once a foldable lens is injected through it. This is the standard size.
3. **3 mm - 3.5 mm:** Larger than needed once foldable lenses came into use; this size fit older rigid lenses better and adds more astigmatism.
4. **3.5 mm - 4 mm:** Even larger, closer to an old style extracapsular cataract wound, and not needed for a foldable lens.

A wound of about 2 mm to 2.5 mm gives enough room for the instruments while staying small enough to self seal, which is why it is the standard for sutureless phacoemulsification with a foldable IOL.

Let's summarize:

- Foldable IOLs pass through a small injector opening, so the wound does not need to match the lens's unfolded size.
- A 2 mm to 2.5 mm clear corneal wound is the standard sutureless size used today.

So the correct incision range is 2 mm to 2.5 mm.

Quick Tip: A foldable IOL lets phaco use a small, self sealing corneal wound, about 2 to 2.5 mm.



72. A one year old child having leucocoria was found to have a unilateral, large retinoblastoma filling half the globe. Current therapy would involve:

- (A) Enucleation
- (B) Chemotherapy followed by local therapy
- (C) Direct laser ablation using photodynamic cryotherapy
- (D) Scleral radiotherapy followed by chemotherapy

Correct Answer: (A) Enucleation

SOLUTION:

This question tests how the size and spread of a retinoblastoma inside the eye decide its treatment.

1. **Enucleation:** When a one sided tumor already fills half the globe, the eye has no useful vision left and carries a risk of the tumor spreading outside the eye. Removing the eye is the safest and standard step in this setting.
2. **Chemotherapy followed by local therapy:** This eye sparing approach works for smaller tumors where enough healthy retina remains to keep some vision, which does not apply here.
3. **Direct laser ablation using photodynamic cryotherapy:** Laser and cryotherapy can only clear small, thin tumors; a mass filling half the globe is far too large and thick for these local methods.
4. **Scleral radiotherapy followed by chemotherapy:** Plaque radiotherapy is kept for smaller, localized tumors that failed other eye sparing treatments, not for a tumor this advanced.

Since the tumor is one sided, large, and already fills half the globe, there is no vision worth preserving, so the eye is removed to protect

the child and stop any spread.

Let's summarize:

- Small retinoblastomas get eye sparing chemotherapy plus local laser or cryotherapy.
- Large, advanced, one sided tumors filling much of the globe get enucleation.

So the correct treatment here is enucleation.

Quick Tip: A large unilateral tumor filling half the globe has no vision worth saving, so the eye is removed.

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73. A 2 year old presented with leucocoria in the right eye since 2 months. On examination, a total retinal detachment was present in the same eye. Ultrasound B scan revealed a heterogenous subretinal mass with calcification, associated with a retinal detachment. The most likely clinical diagnosis is:

- (A) Coats disease
- (B) Retinoblastoma
- (C) Toxocariasis
- (D) Retinal tuberculoma

Correct Answer: (B) Retinoblastoma

SOLUTION:

This question is really about reading one specific ultrasound clue correctly: calcification inside the mass.

1. **Coats disease:** Causes retinal detachment through leaking vessels and lipid rich subretinal fluid, but it does not form a calcified mass, so this does not match the scan.
2. **Retinoblastoma:** Grows fast, outstrips its blood supply, and deposits calcium within the tumor. A calcified, heterogenous subretinal mass with detachment in a young child is its classic ultrasound signature.
3. **Toxocariasis:** Produces an inflammatory granuloma from a larva, which is soft tissue, not a calcified mass.
4. **Retinal tuberculoma:** A rare granulomatous mass that also does not typically calcify on B scan.

Since calcification within an intraocular mass in a 2 year old is essentially specific for retinoblastoma, that is the diagnosis the scan points to.

Let's summarize:

- Retinal detachment alone can come from several childhood eye diseases.
- Calcification inside the mass is the deciding clue, and it means retinoblastoma.

So the most likely diagnosis is retinoblastoma.

Quick Tip: Calcification on B scan inside a childhood intraocular mass points strongly to retinoblastoma.



74. A 60 year old man presented with watering from his left eye since one year. Syringing revealed a patent drainage system. Rest of the ocular examination was normal. A provisional diagnosis of lacrimal pump failure was made. Confirmation of this diagnosis would be:

- (A) Dacryoscintigraphy
- (B) Dacryocystography
- (C) Pressure syringing
- (D) Canaliculus irrigation test

Correct Answer: (A) Dacryoscintigraphy

SOLUTION:

The key here is telling apart a blocked drainage channel from a channel that is open but not pumping tears well.

1. **Dacryoscintigraphy:** Places a tiny radioactive tracer on the eye and tracks it into the nose with a gamma camera during normal blinking. It measures the actual pumping action of the eyelids on the tear sac, which is exactly what needs checking when the channel is already open. This is the confirmatory test.
2. **Dacryocystography:** Uses dye and X-rays to outline the shape of the drainage channel. It is an anatomical test, and this patient's channel is already known to be open from syringing.
3. **Pressure syringing:** Still just forces fluid through under pressure, so it repeats what the first syringing already proved and adds nothing about how the tears drain during normal blinking.
4. **Canaliculus irrigation test:** Again checks whether fluid passes through the canaliculus under pressure, an anatomical patency test, not a functional one.

Because the drainage system is already open, the diagnosis rests on showing that tears fail to move through it under ordinary conditions, and dacryoscintigraphy is built to show exactly that.

Let's summarize:

- Syringing and irrigation tests check anatomy under artificial pressure.
- Dacryoscintigraphy checks physiology, how well the pump moves tears naturally.

So dacryoscintigraphy is the test that confirms lacrimal pump failure.

Quick Tip: Patent syringing rules out a block, so the next test must check tear pump function, not anatomy.

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75. While working in the neonatal ICU, your team delivers a premature infant at 27 weeks of gestation, weighing 1500 grams. How soon will you request a fundus examination by an ophthalmologist?

- (A) Immediately
- (B) 3-4 weeks after delivery
- (C) At 34 weeks gestational age
- (D) At 40 weeks gestation period

Correct Answer: (C) At 34 weeks gestational age

SOLUTION:

This question is about when, not whether, to screen a premature baby for retinopathy of prematurity (ROP).

1. **Immediately:** Too soon. The retinal vessel changes of ROP take weeks to show up, so an exam right after birth would not catch early disease and risks confusing normal immature vessels for disease.
2. **3-4 weeks after delivery:** This flat calendar rule ignores how early the baby was born. A baby born at 27 weeks needs the exam timed against its combined age since conception, not a fixed count of weeks after the delivery date.
3. **At 34 weeks gestational age:** Screening guidelines time the first exam to whichever is later, about 4 to 6 weeks after birth or about 31 to 33 weeks postmenstrual age. For a baby born at 27 weeks, this lands close to 33 to 34 weeks postmenstrual age, matching this option.
4. **At 40 weeks gestation period:** Far too late. Sight threatening ROP can progress and even need treatment before a baby reaches full term equivalent age.

Postmenstrual age, meaning gestational age at birth plus the weeks the baby has lived, is the number that decides when the retina is developed enough to check meaningfully, and for this baby that points to about 34 weeks.

Let's summarize:

- ROP screening timing is based on postmenstrual age, not the calendar date of delivery.
- A 27 week preemie reaches the screening window at around 33 to 34 weeks postmenstrual age.

So the fundus exam should be requested at about 34 weeks gestational age.

Quick Tip: For very preterm babies, the first ROP screen is timed by postmenstrual age, about 31-34 weeks, not by a fixed number of weeks after birth.

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76. A friend of yours has a spectacle correction of -6.0 D and -8.0 D. He telephones you one morning and tells you that he has started seeing some opacities floating in front of his eyes and that his vision has decreased slightly over the last few days. As an intern in the ophthalmology section, what would you do?

- (A) Reassure
- (B) Refraction and prescribe a new spectacle
- (C) Direct ophthalmoscopy
- (D) Indirect ophthalmoscopy

Correct Answer: (D) Indirect ophthalmoscopy

SOLUTION:

The clue to catch here is the combination of high myopia with new floaters and dropping vision, which points toward the retinal periphery.

1. **Reassure:** Would be reasonable for a single old floater in a normal eye, but in a high myope with new symptoms, it risks missing a retinal tear that is still treatable at this early stage.
2. **Refraction and prescribe a new spectacle:** Only corrects focusing power and says nothing about the state of the retina, so it does not address the real risk here.
3. **Direct ophthalmoscopy:** Gives a small, magnified, straight ahead view and cannot reach the far periphery, where retinal

tears in high myopes usually start.

4. **Indirect ophthalmoscopy:** Uses a headset and lens to give a wide, three dimensional view of the whole retina out to its far edge, with the option of scleral depression, which is exactly what is needed to find a peripheral tear or early detachment.

Because high myopia predisposes to peripheral retinal tears, and this patient now has fresh floaters with reduced vision, a full peripheral retinal check with indirect ophthalmoscopy is the right next step.

Let's summarize:

- High myopia raises the risk of retinal tears and detachment.
- New floaters plus vision loss in such a patient need a peripheral retinal exam, best done by indirect ophthalmoscopy.

So the correct action is indirect ophthalmoscopy.

Quick Tip: New floaters and vision drop in a high myope suggest a retinal tear; check the far periphery with indirect ophthalmoscopy.

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77. A 30 year old male presents with a history of injury to the eye with a leaf 5 days ago, and pain, photophobia, and redness of the eye for 2 days. What would be the most likely pathology?

- (A) Anterior uveitis
- (B) Conjunctivitis
- (C) Fungal corneal ulcer
- (D) Corneal laceration

Correct Answer: (C) Fungal corneal ulcer

SOLUTION:

Two details in this history matter most: what caused the injury, and how long it took for symptoms to appear.

1. **Anterior uveitis:** Can follow trauma, but it has no special link to vegetable matter injury and does not explain why the plant injury specifically matters here.
2. **Conjunctivitis:** Causes redness but usually mild discomfort rather than marked pain and photophobia, and it is not a typical outcome of a corneal injury with plant matter.
3. **Fungal corneal ulcer:** Vegetable matter injuries are the classic setup for fungal keratitis, since fungal spores live on plant surfaces. Fungal ulcers also tend to grow slowly, so pain and photophobia appearing several days after the injury, rather than right away, fits this diagnosis well.
4. **Corneal laceration:** A cut to the cornea causes sharp pain immediately at the time of injury, not a gradual onset of symptoms starting days later, so the timeline does not match.

The combination of a vegetable matter injury and a delayed, several day onset of pain, photophobia, and redness is the textbook picture of a fungal corneal ulcer.

Let's summarize:

- Plant or vegetable matter injuries carry a specific risk for fungal keratitis.
- A slow, delayed onset of symptoms after such an injury favors a fungal cause over a bacterial or mechanical one.

So the most likely pathology is a fungal corneal ulcer.

Quick Tip: Vegetable matter injury with symptoms starting days later, not right away, points to a fungal corneal ulcer.

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78. The most likely diagnosis in a newborn with a radio-opaque shadow and an air-fluid level in the chest, along with a hemivertebra of the 6th thoracic vertebra on a plain x-ray, is:

- (A) Congenital diaphragmatic hernia
- (B) Esophageal duplication cyst
- (C) Bronchogenic cyst
- (D) Staphylococcal pneumonia

Correct Answer: (B) Esophageal duplication cyst

SOLUTION:

This question gives three clues together: a cystic shadow in a newborn's chest, an air-fluid level inside that shadow, and a hemivertebra at T6. Put together, these point to a cyst that shares its origin with both the gut and the spinal column.

1. **Congenital diaphragmatic hernia:** shows bowel loops in the chest with a gas pattern and a shifted mediastinum, but there is no link to a vertebral defect, so this does not fit.
2. **Esophageal duplication cyst:** a foregut duplication cyst of the neurenteric type forms when the notochord and gut fail to separate fully in early life. It can open into the esophagus, so it fills with air and fluid, and the same defect leaves a hemivertebra or spina bifida nearby. This matches every clue in the question.

3. **Bronchogenic cyst:** comes from the breathing-tube side of the foregut, usually sits near the carina, and almost never connects to the airway or gut. Without that connection it stays fluid-filled with no air level, and it carries no link to vertebral anomalies.
4. **Staphylococcal pneumonia:** causes patchy shadowing and pneumatoceles from infection, not a single cyst tied to a spinal defect.

The air-fluid level plus the hemivertebra is the signature of split notochord syndrome, which produces a foregut duplication cyst of the esophageal/neurenteric type. This is why esophageal duplication cyst fits better than bronchogenic cyst, which some older keys list for this question.

Let's summarize:

- An air-fluid level in a chest cyst means the cyst opens into the gut or esophagus.
- A hemivertebra next to a chest cyst points to a shared notochord-gut origin, seen in neurenteric/esophageal duplication cysts, not in bronchogenic cysts.

So the most likely diagnosis is an esophageal duplication cyst.

Quick Tip: Air-fluid level plus a hemivertebra in a newborn's chest points to a foregut duplication (neurenteric) cyst, not a bronchogenic cyst.

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79. Failure to pass meconium within 48 hours of birth in a newborn with no obvious external abnormality should raise suspicion of:

- (A) Anal atresia
- (B) Congenital pouch colon
- (C) Congenital aganglionosis
- (D) Meconium ileus

Correct Answer: (C) Congenital aganglionosis

SOLUTION:

Normal newborns pass meconium within 24 to 48 hours. When that does not happen and the baby's outside anatomy looks normal, the question is asking which inside problem is most likely.

1. **Anal atresia:** this shows up on the very first check as a missing or blocked anal opening, so it does not fit a baby with no external abnormality.
2. **Congenital pouch colon:** this rare condition comes together with an anorectal malformation, so again the outside exam would not be normal.
3. **Congenital aganglionosis:** nerve cells are missing from the end of the colon, so that segment cannot relax and stool backs up behind it. The anus and the outside of the baby look completely normal because the defect is inside the bowel wall, not in the anatomy of the anus.
4. **Meconium ileus:** this blocks the small bowel, mostly in babies with cystic fibrosis, and tends to show early distension and bilious vomiting rather than being the first cause considered for simple delayed meconium.

Since the baby looks normal outside, the block has to be a functional one inside the bowel, and that is exactly what happens in Hirschsprung disease.

Let's summarize:

- Delayed meconium past 48 hours with a normal anus points to a nerve cell problem in the colon.
- Anal atresia and pouch colon are ruled out because they change how the anus looks.

So congenital aganglionosis (Hirschsprung disease) is the right answer.

Quick Tip: Normal-looking anus plus no meconium by 48 hours points to Hirschsprung disease (aganglionosis).

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80. The metabolic derangement in congenital pyloric stenosis is:

- (A) Hypochloremic alkalosis
- (B) Hyperchloremic alkalosis
- (C) Hyperchloremic acidosis
- (D) Hypochloremic acidosis

Correct Answer: (A) Hypochloremic alkalosis

SOLUTION:

Pyloric stenosis blocks the stomach outlet, so the baby keeps bringing up stomach juice by forceful vomiting. Stomach juice is mostly hydrochloric acid, so tracking what leaves the body in that vomit tells us the acid-base picture.

1. **Hypochloremic alkalosis:** vomiting removes acid (hydrogen ions) and chloride together. Losing acid raises blood pH, giving

alkalosis, and losing chloride drops the blood chloride, giving the hypochloremic part. This matches the vomiting pattern exactly.

2. **Hyperchloremic alkalosis:** chloride cannot be high when it is being vomited out in large amounts, so this does not fit.
3. **Hyperchloremic acidosis:** this pattern is seen with diarrhea or kidney bicarbonate loss, not with loss of stomach acid, so it is the wrong direction on both counts.
4. **Hypochloremic acidosis:** the chloride part is right, but losing stomach acid should raise the pH, not lower it, so calling it acidosis is wrong.

Dehydration from the vomiting also makes the kidney hold sodium and water while dumping potassium and acid in the urine, which is why the classic lab picture is described fully as hypochloremic, hypokalemic metabolic alkalosis.

Let's summarize:

- Vomiting stomach acid removes both hydrogen and chloride ions.
- The result is a rise in pH (alkalosis) with a fall in blood chloride (hypochloremic).

So the metabolic derangement in congenital pyloric stenosis is hypochloremic alkalosis.

Quick Tip: Vomiting stomach acid removes H^+ and Cl^- , so pyloric stenosis causes hypochloremic alkalosis.

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81. In neonatal cholestasis, if the serum gamma-glutamyl transpeptidase (GGT) is more than 600 IU/L, the most likely diagnosis is:

- (A) Neonatal hepatitis
- (B) Choledochal cyst
- (C) Hypothyroidism
- (D) Biliary atresia

Correct Answer: (D) Biliary atresia

SOLUTION:

GGT is an enzyme found on bile duct cells. Its level in the blood helps tell apart liver cell disease from bile duct disease in a baby with cholestasis (yellow skin from poor bile flow).

1. **Neonatal hepatitis:** mainly damages liver cells, so it usually pushes up AST and ALT sharply while GGT stays only mildly to moderately high, well under the level in this question.
2. **Choledochal cyst:** can raise GGT since it is also an obstructive problem, but it is a rare cause of neonatal cholestasis and usually comes with a cyst visible on ultrasound, so it is not the most likely answer here.
3. **Hypothyroidism:** can slow bile flow and cause cholestasis, but the GGT rise is usually mild, and the baby shows other clues like lethargy and poor feeding along with an abnormal thyroid test.
4. **Biliary atresia:** the extrahepatic bile ducts are destroyed and blocked from early life, which puts constant strain on duct cells and drives GGT to very high levels, often past 600 IU/L. It is also the single most common obstructive cause of neonatal cholestasis.

Since a GGT this high points to bile duct obstruction rather than plain liver cell injury, and biliary atresia is both obstructive and common, it fits best.

Let's summarize:

- High GGT means bile duct strain or obstruction, not just liver cell injury.
- Biliary atresia is the most common and most likely obstructive cause behind a GGT above 600 IU/L.

So the most likely diagnosis is biliary atresia.

Quick Tip: GGT over 600 in neonatal cholestasis points to bile duct obstruction, most often biliary atresia.

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82. These ventral spinal rootlets are more prone to injury during decompressive operations because they are shorter and exit in a more horizontal direction:

- (A) C5
- (B) C6
- (C) C7
- (D) T1

Correct Answer: (A) C5

SOLUTION:

After surgery to relieve pressure on the cervical spinal cord, one specific nerve root is injured far more often than the rest, causing shoulder and upper arm weakness afterward. The question is asking which root that is and why.

1. **C5:** its ventral rootlet is shorter than the others and leaves the spinal cord at a more horizontal angle. When the cord shifts back after decompression, this short, flat root gets stretched the most, which is why C5 palsy is the classic and most common nerve injury after this type of surgery.
2. **C6:** has a longer course and a steeper exit angle than C5, so it tolerates cord shift better and is injured far less often.
3. **C7:** travels an even longer, more angled course, and injury here is uncommon.
4. **T1:** exits well below the area of most cord shift after cervical decompression and is rarely affected.

The short length and horizontal exit angle of the C5 root is the anatomic reason it gets tethered and stretched when the cord drifts backward, so it is the answer, even though some older question keys also accept C6.

Let's summarize:

- A short, horizontal nerve root stretches more when the spinal cord shifts.
- C5 has this anatomy and is the most common site of postoperative palsy after cervical decompression.

So the correct answer is C5.

Quick Tip: C5 has the shortest, most horizontal ventral root, making it the classic root injured after cervical decompression.

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83. The arachnoid villi responsible for CSF absorption protrude mainly into the:

- (A) Superior sagittal sinus
- (B) Inferior sagittal sinus
- (C) Straight sinus
- (D) Transverse sinus

Correct Answer: (A) Superior sagittal sinus

SOLUTION:

CSF made inside the brain has to get back into the blood somehow, and that job belongs to small finger-like projections of the arachnoid membrane called arachnoid villi. The question asks which venous sinus they mainly poke into.

1. **Superior sagittal sinus:** this sinus runs along the top midline of the skull, right next to the subarachnoid space over the brain's surface. Arachnoid villi cluster here in the largest numbers, especially in its side pouches called venous lacunae, making it the main drainage route for CSF.
2. **Inferior sagittal sinus:** sits deep along the free edge of the falx cerebri and carries much less CSF drainage, with few villi present.
3. **Straight sinus:** mainly drains deep venous blood from structures like the great cerebral vein, not a major site for arachnoid villi.
4. **Transverse sinus:** carries blood onward toward the internal jugular vein but is not where villi concentrate.

Since the space above the brain sits closest to the superior sagittal sinus, that is where most of the CSF drains through the arachnoid villi.

Let's summarize:

- Arachnoid villi are one-way channels that drain CSF into venous blood.
- They are densest along the superior sagittal sinus, the sinus closest to the CSF-filled space over the brain.

So the correct answer is the superior sagittal sinus.

Quick Tip: Arachnoid villi cluster mainly along the superior sagittal sinus, the main CSF drainage route.

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84. The term post-traumatic epilepsy refers to seizures occurring:

- (A) Within moments of head injury
- (B) Within 7 days of head injury
- (C) Several weeks to months after head injury
- (D) Many years after head injury

Correct Answer: (D) Many years after head injury

SOLUTION:

Seizures that follow a head injury are not all the same. Doctors group them by how soon after the injury they start, because the cause and the long-term outlook differ between the groups.

1. **Within moments of head injury:** this is an immediate seizure, a brief reflex-like event tied to the impact itself, not what the term post-traumatic epilepsy is used for.
2. **Within 7 days of head injury:** this is an early post-traumatic seizure, caused by the direct short-term effects of the trauma,

such as bleeding or swelling, and it does not by itself mean the person has epilepsy.

3. **Several weeks to months after head injury:** seizures can start in this window too, but the defining feature of post-traumatic epilepsy is a lasting, recurring tendency, which develops as scar tissue slowly forms in the injured brain.
4. **Many years after head injury:** the scarring and abnormal circuit formation that cause true post-traumatic epilepsy can take a long time to mature, so this chronic seizure disorder can first appear even years after the original trauma, and the term post-traumatic epilepsy is used for this long-term, recurring pattern.

The key idea is that post-traumatic epilepsy is not just a seizure after a head injury, it is a lasting seizure disorder that can take years to develop and show itself.

Let's summarize:

- Immediate and early seizures come from the direct effects of the injury and usually settle.
- Post-traumatic epilepsy is the chronic, recurring pattern that can take years to appear as scar tissue forms.

So post-traumatic epilepsy refers to seizures that can occur many years after the head injury.

Quick Tip: Post-traumatic epilepsy is the chronic, recurring seizure disorder that can appear years after head injury, unlike early post-traumatic seizures.

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85. Treatment of squamous cell carcinoma of the anal canal is:

- (A) Cisplatin based chemotherapy followed by radical radiotherapy
- (B) Abdominoperineal resection
- (C) Radical radiotherapy
- (D) Radical radiotherapy followed by mitomycin C based chemotherapy

Correct Answer: (A) Cisplatin based chemotherapy followed by radical radiotherapy

SOLUTION:

Step 1: Think about what changed the treatment of this cancer.

Anal canal squamous cell carcinoma was once treated mainly by removing the anus and rectum. That changed once doctors found that chemotherapy paired with radiotherapy could destroy the tumor just as well while leaving the sphincter in place.

Step 2: Work out the correct order of treatment.

Chemotherapy is given first so the tumor cells become more sensitive to the radiation dose that follows. Starting with chemotherapy and then delivering radical radiotherapy is the working sequence behind this regimen.

Step 3: Test each remaining option against this idea.

Removing the anus and rectum surgically skips the sphincter sparing benefit entirely, so it is not the first choice anymore. Radiotherapy on its own drops the cure rate because it loses the chemical sensitizing effect. Placing the chemotherapy after the radical radiotherapy breaks the working order, since the sensitizing step needs to come first.

Step 4: Settle on the answer.

Only the option that starts with chemotherapy and follows with

radical radiotherapy matches the accepted combined modality approach for this cancer.

Chemotherapy followed by radical radiotherapy

Quick Tip: Anal canal SCC is cured with combined chemotherapy and radiotherapy, not surgery first; surgery is kept for salvage.

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86. All of the following are indications for surgery in gastric lymphomas, except:

- (A) Bleeding
- (B) Perforation
- (C) Residual disease following chemotherapy
- (D) Intractable pain

Correct Answer: (D) Intractable pain

SOLUTION:

Step 1: Start from the usual treatment plan.

Gastric lymphoma responds well to chemotherapy, radiotherapy, and, for the MALT type, H. pylori eradication. An operation is not the first step in a straightforward case.

Step 2: List the situations where surgery becomes necessary.

Surgery steps in for heavy bleeding that cannot be stopped medically, for a perforated stomach wall, for a blockage of the stomach outlet, and for a lump of tumor that survives a full course of chemotherapy.

Step 3: Compare the four options against that list.

Bleeding, perforation, and leftover disease after chemotherapy all appear directly on that list of surgical triggers. Pain by itself is not on the list, since it is usually settled with painkillers and by shrinking the tumor with chemotherapy or radiotherapy.

Step 4: Conclude.

The option that does not belong with the true surgical indications is intractable pain.

Intractable pain

Quick Tip: Surgery in gastric lymphoma is reserved for complications like bleeding, perforation, or leftover tumor after chemotherapy, not for pain alone.

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87. A 22 year old man presents with a solitary 2 cm space occupying lesion of mixed echogenicity in the right lobe of the liver on ultrasound. The rest of the liver is normal. Which of the following tests should be done?

- (A) Ultrasound guided biopsy of the lesion
- (B) Hepatic scintigraphy
- (C) Hepatic angiography
- (D) Contrast enhanced CT scan of the liver

Correct Answer: (D) Contrast enhanced CT scan of the liver

SOLUTION:

Step 1: Picture the clinical scene.

A healthy young man is found to have one small liver lesion by chance

on ultrasound, with the rest of the liver looking normal. This pattern usually points toward a benign growth rather than cancer.

Step 2: Ask what test settles the diagnosis safely.

The safest and most informative next step is a scan that shows how the lesion takes up contrast dye over time, since hemangiomas, focal nodular hyperplasia, and adenomas each light up in a distinctive way on a contrast enhanced CT.

Step 3: See why the other tests fall short.

A biopsy carries a real risk of bleeding if the mass turns out to be a vascular benign tumor, so it is held back until imaging is inconclusive. Scintigraphy only gives supporting evidence for one type of lesion and misses fine detail. Angiography is invasive and has largely been replaced by CT for this purpose.

Step 4: Conclude.

The correct next test is a contrast enhanced CT scan of the liver.

Contrast enhanced CT scan of the liver

Quick Tip: A young patient with a solitary liver mass in an otherwise normal liver needs non-invasive characterization by contrast CT before any biopsy is considered.

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88. Which one of the following preservatives is used while packing catgut cultures?

- (A) Isopropyl alcohol
- (B) Colloidal iodine
- (C) Glutaraldehyde
- (D) Hydrogen peroxide

Correct Answer: (A) Isopropyl alcohol

SOLUTION:

Step 1: Think about what catgut is made of.

Catgut is a collagen suture taken from animal tissue. Collagen strands can dry out, stiffen, or get infected if they are not stored correctly after sterilization.

Step 2: Work out what the storage fluid must avoid.

A good storage fluid must kill or stop bacteria without eating away at the collagen itself, since a suture that turns brittle or weak in the packet is useless in surgery.

Step 3: Match each option to its usual role.

Colloidal iodine and hydrogen peroxide are antiseptics applied to skin or wounds, not fluids meant for long-term suture storage.

Glutaraldehyde is a strong instrument disinfectant that would be too harsh on a collagen strand over time. Isopropyl alcohol, on the other hand, is mild enough to keep the strand supple while still preventing bacterial growth, which is exactly why it is the standard packing fluid for catgut.

Step 4: Conclude.

Isopropyl alcohol

Quick Tip: Catgut suture packets are preserved in isopropyl alcohol, which keeps the strand sterile and supple.

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89. Ramkumar, a 70 year old hypertensive male, was admitted to the intensive care unit with transmural anterolateral myocardial infarction. His condition was stable till the 5th day of admission, when he developed a pericardial friction rub and pleuritic chest pain which persisted despite narcotic and steroid therapy. On the 7th morning he suddenly developed marked hypotension. On examination, there was distension of jugular veins, accompanied by electromechanical dissociation. Most likely, the patient had developed:

- (A) Severe acute mitral regurgitation
- (B) Ventricular septal rupture
- (C) Right ventricular rupture
- (D) External (free wall) cardiac rupture

Correct Answer: (D) External (free wall) cardiac rupture

SOLUTION:

Step 1: Build the timeline of this patient.

A large anterolateral transmural infarct in an elderly hypertensive man is a known setup for the heart wall to give way. His course stayed stable for several days, then pericarditis appeared on day 5, and sudden collapse followed on day 7. Free wall rupture almost always shows up in this same first week window.

Step 2: Treat the pericarditis as a clue, not a coincidence.

A friction rub and persistent pleuritic pain over a dead area of heart

muscle often signals that the wall there has become thin and fragile, which is exactly the setting in which the wall can tear open.

Step 3: Match the collapse to a mechanism.

Sudden low blood pressure, full neck veins, and a heart that keeps its electrical rhythm without pumping effectively is the signature of blood suddenly flooding the pericardial sac and squeezing the heart from outside, that is, tamponade from a torn free wall.

Step 4: Eliminate the other mechanical complications.

A torn papillary muscle gives a new murmur with flash pulmonary edema, not this tamponade picture. A torn septum gives a new pansystolic murmur and a shunt, again not this exact tamponade and electromechanical dissociation combination. Right ventricular rupture alone does not fit the anterolateral distribution or the typical week-one free wall rupture pattern as well as left ventricular free wall rupture does.

Step 5: Conclude.

External (free wall) cardiac rupture

Quick Tip: Pericarditis after a transmural MI followed by sudden hypotension, distended neck veins, and electromechanical dissociation on day 5 to 7 points to free wall cardiac rupture with tamponade.

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90. A young motorist suffered injuries in a major road traffic accident. He was diagnosed to have a fracture of the left humerus. He also had fractures of multiple ribs anteriorly on both sides. On examination, the blood pressure was 80/60 mm Hg, and the heart rate was 140/minute. The patient was agitated, restless, and tachypneic. Jugular veins were distended. Air entry was adequate in both lung fields. Heart sounds were barely audible. Femoral pulses were weakly palpable but no pulsation could be felt distally. On a priority basis, the immediate intervention would be:

- (A) Rapid blood transfusion
- (B) Urgent pericardial tap
- (C) Intercostal drainage on both sides
- (D) Fixation of left femur and repair of femoral artery

Correct Answer: (B) Urgent pericardial tap

SOLUTION:

Step 1: Name the pattern first.

Low blood pressure plus full neck veins plus faint heart sounds is a well known combination called Beck's triad, and in a patient with chest trauma it means the heart is being squeezed by fluid trapped around it, that is, cardiac tamponade.

Step 2: Use the neck veins to sort out the type of shock.

If this patient were simply bleeding out from his injuries, the neck veins would collapse rather than bulge, because there would not be enough blood to fill them. Since the veins are distended instead, the problem is blood struggling to get back into a heart that is being compressed, not a lack of blood volume.

Step 3: Check the lungs before blaming them.

Both lungs are ventilating fine on exam, so a collapsed lung or a large blood collection in the chest cavity is not driving this picture, even with the rib fractures present.

Step 4: Decide what must happen first.

Tamponade kills quickly by stopping the heart from filling, so it has to be drained before anything else. A pericardial tap removes the compressing fluid and lets the heart fill and pump again.

Step 5: See why the other choices wait.

Giving blood does not solve a heart that cannot fill because it is squeezed from outside. Chest tubes are not indicated when air entry is already normal on both sides. The limb with weak pulses does need vascular repair, but a squeezed, failing heart is the more immediate threat and is treated first.

Step 6: Conclude.

Urgent pericardial tap

Quick Tip: Hypotension with distended neck veins and muffled heart sounds after chest trauma is Beck's triad; treat the tamponade first with urgent pericardiocentesis.

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91. A 35 year old farmer consulted a local medical practitioner for recurrent attacks of chest pain. His elder brother had similar complaints and had died suddenly at the age of 40 years. The farmer was advised to take nitroglycerine sublingually at the time of pain. However, the patient finds that the intensity of pain is increased by nitroglycerine. Most probably he is suffering from:

- (A) Subacute bacterial endocarditis involving the aortic valve
- (B) Hypertrophic obstructive cardiomyopathy
- (C) Degenerative mitral regurgitation
- (D) Chronic type A dissection of the aorta

Correct Answer: (B) Hypertrophic obstructive cardiomyopathy

SOLUTION:

Step 1: Read the twist in the question.

Nitroglycerine is supposed to ease chest pain by relaxing the veins and easing the heart's load. Here it does the opposite and makes the pain worse, so this cannot be a plain case of coronary artery blockage.

Step 2: Recall what makes a heart condition react this way.

In hypertrophic obstructive cardiomyopathy the thickened muscle wall already blocks the path blood takes out of the heart.

Nitroglycerine reduces the blood returning to the heart and the resistance it pumps against, which shrinks the ventricle even further and tightens that blockage, worsening the pain instead of relieving it.

Step 3: Weigh the family history.

A brother who died suddenly at 40 fits the inherited pattern of hypertrophic cardiomyopathy, which runs in families and is a leading cause of sudden cardiac death in relatively young people.

Step 4: Cross off the other diagnoses.

Endocarditis comes with fever and a changing murmur, not this specific nitrate reaction. Mitral regurgitation from wear and tear causes breathlessness and a regurgitant murmur, without this family pattern of sudden death. Aortic dissection causes sudden tearing pain radiating to the back, and does not show this particular worsening with nitroglycerine.

Step 5: Conclude.

Hypertrophic obstructive cardiomyopathy

Quick Tip: Chest pain that worsens, not improves, with nitroglycerine plus a family history of sudden death points to hypertrophic obstructive cardiomyopathy.

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92. A 30 year old man presents with pain in the right flank and hematuria. A CECT abdomen shows a large 8 x 8 cm solid mass in the right kidney and a 3 x 3 cm solid mass in the upper pole of the left kidney. What is the most appropriate surgical treatment for this patient?

- (A) Bilateral radical nephrectomy
- (B) Right radical nephrectomy and biopsy of the mass from the opposite kidney
- (C) Right radical nephrectomy and left partial nephrectomy
- (D) Right radical nephrectomy only

Correct Answer: (C) Right radical nephrectomy and left partial nephrectomy

SOLUTION:

Step 1: Identify the disease.

Flank pain, blood in the urine, and two separate solid renal masses on CECT is the classic picture of bilateral renal cell carcinoma. Renal masses that are solid and enhance on contrast CT are treated as cancer until proven otherwise.

Step 2: Apply the rule for bilateral kidney tumors.

With tumors in both kidneys, the surgical plan always tries to leave the patient with functioning kidney tissue on at least one side, since losing both kidneys means dialysis for life. This is why nephron sparing surgery, meaning partial removal of the kidney rather than the whole organ, is preferred whenever the tumor size and position allow it.

Step 3: Judge each mass on its own size.

An 8 x 8 cm mass is large. A tumor of this size usually replaces most of the kidney and cannot be removed with a safe margin while sparing the organ, so the right kidney is removed completely (radical nephrectomy).

A 3 x 3 cm mass at the upper pole is small and sits at one end of the kidney, which is exactly the setting where a partial nephrectomy, cutting out the tumor with a rim of normal tissue and closing the defect, works well and keeps the rest of the kidney functioning.

Step 4: Check why the other choices fail.

Removing both kidneys entirely throws away a kidney that could have been partly saved. Biopsy alone leaves a probable cancer sitting in the left kidney with no definitive treatment. Treating only the right side and ignoring the left mass leaves known cancer behind.

Step 5: State the answer.

Right radical nephrectomy for the large tumor and left partial nephrectomy for the small tumor gives the best balance between clearing the cancer and keeping kidney function.

Right radical nephrectomy and left partial nephrectomy

Quick Tip: Small pole tumor gets nephron sparing surgery; large tumor replacing the kidney gets radical nephrectomy.

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93. A 40 year old male patient is brought to the emergency room with a stab injury to the chest. On examination the patient is found to be hemodynamically stable. The neck veins are engorged and the heart sounds are muffled. All the following statements are true about this patient except:

- (A) Cardiac tamponade is likely to be present
- (B) Immediate emergency room thoracotomy should be done
- (C) Echo should be done to confirm pericardial blood
- (D) The entry wound should be sealed with an occlusive dressing

Correct Answer: (B) Immediate emergency room thoracotomy should be done

SOLUTION:

This question describes a stable patient with a stab wound to the chest who has two of the three signs of cardiac tamponade: engorged neck veins and muffled heart sounds. We need to find the one statement among the four that is NOT true for this patient.

1. **Cardiac tamponade is likely to be present:** True. Neck vein engorgement plus muffled heart sounds after a penetrating chest wound is the classic setup for blood collecting around the heart.
2. **Immediate emergency room thoracotomy should be done:** False. This aggressive bedside surgery is kept for patients who have no pulse or arrest in front of the team. A patient who is still hemodynamically stable should instead be worked up with imaging and taken to the operating room under controlled conditions, not cut open immediately in the emergency room.
3. **Echo should be done to confirm pericardial blood:** True. Ultrasound of the heart is quick and safe, and it is the standard way to look for fluid in the pericardial sac in a stable patient before deciding the next surgical step.
4. **The entry wound should be sealed with an occlusive dressing:** True. Covering a chest wound with an airtight dressing is basic first aid while the rest of the assessment is completed.

Three of the four statements describe correct, standard management. Only the call for immediate emergency room thoracotomy does not fit a stable patient, since that operation is meant for patients in extremis, not for one who is stable on arrival.

Let's summarize:

- Muffled heart sounds and neck vein engorgement after chest trauma suggest cardiac tamponade.
- Emergency room thoracotomy is for patients without vital signs, not for a stable patient.
- Echo and an occlusive dressing are both appropriate steps in a stable patient with a chest wound.

So the statement that is NOT true is that immediate emergency room thoracotomy should be done.

Quick Tip: Beck's triad points to tamponade, but ER thoracotomy is only for patients without vital signs.

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94. All the following statements are true about germ cell tumours of the testis except:

- (A) They constitute 90 to 95 percent of all primary testicular tumours
- (B) Seminoma is the most common tumour developing in patients with cryptorchid testes
- (C) Alpha-fetoprotein is markedly raised in all germ cell tumours
- (D) High inguinal orchidectomy is the initial surgical procedure

Correct Answer: (C) Alpha-fetoprotein is markedly raised in all germ cell tumours

SOLUTION:

The question asks us to find the one FALSE statement about germ cell tumours of the testis among four choices.

1. **They constitute 90 to 95 percent of all primary testicular tumours:** True. Germ cell tumours are by far the largest group of testicular cancers, with sex cord stromal and other rare tumours making up the small remainder.
2. **Seminoma is the most common tumour developing in patients with cryptorchid testes:** True. An undescended

testis carries a higher cancer risk, and seminoma is the type most often seen when cancer develops in such a testis.

3. Alpha-fetoprotein is markedly raised in all germ cell

tumours: False. AFP comes from yolk sac tissue, so it rises in yolk sac tumour, embryonal carcinoma and mixed tumours with yolk sac elements. A pure seminoma has no yolk sac component and does not raise AFP, so this marker is not raised in every germ cell tumour.

4. High inguinal orchidectomy is the initial surgical

procedure: True. Removing the testis through the groin with early cord ligation is the standard first surgical step for a suspicious testicular mass, done to avoid spreading tumour cells into the scrotal tissue.

Three statements hold up against what is known about testicular germ cell tumours. The claim about AFP being raised in every germ cell tumour breaks down because pure seminoma does not secrete it.

Let's summarize:

- AFP is a marker for yolk sac elements, not for germ cell tumours as a whole.
- Pure seminoma stays AFP negative, which actually helps tell it apart from non-seminomatous tumours.
- The other three statements about incidence, cryptorchidism and initial surgery are all standard and correct.

So the false statement is that alpha-fetoprotein is markedly raised in all germ cell tumours.

Quick Tip: AFP comes from yolk sac elements, so pure seminoma does not raise it.

95. A 25 year old male presents to the emergency department following a road traffic accident. On examination there is a pelvic fracture and blood at the urethral meatus. All the following are true about this patient except:

- (A) The anterior urethra is most likely the site of injury
- (B) Retrograde urethrography should be done after the patient is stabilized
- (C) A Foley catheter may be carefully passed if RGU is normal
- (D) Rectal examination may reveal a large pelvic hematoma with the prostate displaced superiorly

Correct Answer: (A) The anterior urethra is most likely the site of injury

SOLUTION:

We need to spot the one FALSE statement about a patient with a pelvic fracture and blood at the urethral meatus, a picture that points to urethral injury tied to the fracture.

1. The anterior urethra is most likely the site of injury:

False. A pelvic fracture stretches and shears the posterior urethra, most often right at the prostatomembranous junction. The anterior urethra is usually hurt by a direct straddle injury, a completely different mechanism, so it is not the likely site here.

2. Retrograde urethrography should be done after the

patient is stabilized: True. Once the patient is out of immediate danger, injecting dye into the urethra under X-ray shows whether the urethra is torn before any instrument is passed.

3. A Foley catheter may be carefully passed if RGU is

normal: True. A normal retrograde urethrogram, with no dye

leak, means the urethra is intact enough that a soft catheter can be passed gently.

4. **Rectal examination may reveal a large pelvic hematoma with the prostate displaced superiorly:** True. A high-riding prostate felt on rectal exam, sitting in a boggy hematoma, is a textbook sign of a posterior urethral tear.

Three of the four statements describe the accepted picture and workup of pelvic fracture urethral injury. The one that does not fit is the claim about the anterior urethra, since the injury in this setting sits in the posterior urethra.

Let's summarize:

- Pelvic fracture urethral injury affects the posterior, membranous urethra, not the anterior urethra.
- Retrograde urethrography before catheterization, and a high-riding prostate on rectal exam, are both standard and correct findings and steps.

So the false statement is that the anterior urethra is most likely the site of injury.

Quick Tip: Pelvic fracture injures the posterior, membranous urethra, not the anterior urethra.

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96. All the following statements are true about Peyronie's disease except:

- (A) Patients present with complaints of painful erection
- (B) The condition affects adolescent males
- (C) The condition is associated with Dupuytren's contracture of the tendons of the hand
- (D) Spontaneous regression occurs in 50 percent of the cases

Correct Answer: (B) The condition affects adolescent males

SOLUTION:

The question asks which statement about Peyronie's disease is NOT true.

1. Patients present with complaints of painful erection:

True. The stiff, non-stretching plaque inside the penis causes pain along with curving during an erection, which is a common early complaint.

2. The condition affects adolescent males: False. Peyronie's disease is a condition of adult life, usually showing up in men in their 40s to 60s. It is not a disease of adolescent males.

3. The condition is associated with Dupuytren's contracture of the tendons of the hand: True. Both conditions belong to the same family of fibrous tissue disorders, and one is a recognised risk marker for the other.

4. Spontaneous regression occurs in 50 percent of the cases: True. A meaningful share of patients, often quoted as around half, see the plaque and curvature improve on their own without treatment.

Three statements fit standard teaching on Peyronie's disease. The age claim is the odd one out, since this disease is seen in adult and older men, not adolescents.

Let's summarize:

- Peyronie's disease typically affects middle aged and older men.
- Painful erection, association with Dupuytren's contracture, and spontaneous partial improvement are all accepted features.

So the false statement is that the condition affects adolescent males.

Quick Tip: Peyronie's disease is a disease of adult and older men, not adolescents.

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97. The active immunity offered by tetanus toxoid is effectively present in nearly:

- (A) 25 percent of the patients
- (B) 50 percent of the patients
- (C) 75 percent of the patients
- (D) 100 percent of the patients

Correct Answer: (D) 100 percent of the patients

SOLUTION:

Step 1: Understand the vaccine.

Tetanus toxoid is the inactivated, toxoid form of the tetanus exotoxin, given to make the body build its own protective antibodies. This is active immunity, different from giving ready made antibodies, which would be passive immunity.

Step 2: Attach a number to "effective".

Public health teaching describes a completed primary schedule of

tetanus toxoid as protective in almost every healthy person who receives it, generally placed at about 80 to 100 percent seroconversion. In simple exam language, this is rounded to nearly 100 percent.

Step 3: Weigh the choices against this number.

A quarter or half of patients being protected, 25 percent or 50 percent, would make tetanus toxoid a weak vaccine, which does not match its real world record as a dependable, widely used immunization. Three quarters, 75 percent, sits near the bottom of the reported range, so it undercounts the protection most people actually get. The option of nearly all patients being protected fits best with how the vaccine performs after a full course.

Step 4: Acknowledge the close call.

The source answer sheet itself flags that both 75 percent and 100 percent could be defended, since the quoted range spans 80 to 100 percent. Sticking with the stated key and the general teaching that tetanus toxoid protection is close to universal after a complete course, the best single choice is nearly 100 percent.

100%

Quick Tip: A full primary course of tetanus toxoid protects nearly all recipients, about 80 to 100 percent.

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98. The commonest organism causing cellulitis is:

- (A) *Streptococcus pyogenes*
- (B) *Streptococcus faecalis*
- (C) *Streptococcus viridans*
- (D) Microaerophilic streptococci

Correct Answer: (A) *Streptococcus pyogenes*

SOLUTION:

The question asks for the germ most often responsible for cellulitis, a spreading skin and soft tissue infection without a clear pus pocket.

1. ***Streptococcus pyogenes***: This is group A streptococcus, well known for spreading fast through tissue planes because of the enzymes it produces. It is the organism most commonly blamed for typical cellulitis.
2. ***Streptococcus faecalis***: Now classed as *Enterococcus faecalis*, this organism is more linked to urinary tract, biliary and intra-abdominal infections, not the usual cause of skin cellulitis.
3. ***Streptococcus viridans***: This group lives normally in the mouth and is far better known for causing infective endocarditis and dental abscesses than skin cellulitis.
4. **Microaerophilic streptococci**: These organisms are typically part of mixed infections in low oxygen wounds, such as synergistic gangrene, rather than being the leading cause of straightforward cellulitis.

Among the four choices, only *Streptococcus pyogenes* fits the profile of the classic organism causing cellulitis.

Let's summarize:

- Cellulitis without abscess is most often caused by beta hemolytic streptococci.
- Streptococcus pyogenes, group A strep, is the single most common organism.
- The other three streptococcal species are linked to different infections, not typical cellulitis.

So the commonest organism causing cellulitis is Streptococcus pyogenes.

Quick Tip: Streptococcus pyogenes is the classic cause of non-purulent cellulitis.

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99. Which of the following is NOT a component of crush syndrome?

- (A) Myoglobinuria
- (B) Massive crushing of muscles
- (C) Acute tubular necrosis
- (D) Bleeding diathesis

Correct Answer: (D) Bleeding diathesis

SOLUTION:

Crush syndrome is the set of changes that follow long compression of a large muscle mass, as in earthquake or accident victims. We need the option that does not belong to this syndrome.

1. **Myoglobinuria:** crushed muscle releases myoglobin into the blood, which is then filtered into the urine. This is one of the

classic markers checked for after a crush injury, so it is a genuine part of the syndrome.

2. **Massive crushing of muscles:** this is the injury that sets off the whole process, so by definition it belongs to the syndrome.
3. **Acute tubular necrosis:** the myoglobin that leaks out damages the kidney tubules, especially in a dehydrated or acidotic patient. This kidney injury is a well known and expected part of crush syndrome.
4. **Bleeding diathesis:** crush syndrome centers on muscle breakdown, myoglobin release, hyperkalemia and kidney failure. An abnormal bleeding tendency is not one of its defining features, so it does not fit here.

The finding that does not fit the syndrome is bleeding diathesis, so option 4 is the answer.

Let's summarize:

- Crush syndrome runs on three linked events: muscle crush, myoglobin release and tubular kidney injury.
- Bleeding diathesis is not part of this core picture.

The correct option is bleeding diathesis.

Quick Tip: Crush syndrome is built around muscle breakdown, myoglobin release and kidney injury, not a bleeding disorder.



100. A 25 year old young woman has recurrent episodes of headache and sweating. Her mother had renal calculi and died after having a neck mass. Physical examination reveals a thyroid nodule but no clinical sign of thyrotoxicosis. Before performing thyroid surgery, the surgeon should order:

- (A) Measurement of thyroid hormones
- (B) Serial determinations of serum calcium, phosphorus, protein and alkaline phosphatase
- (C) 24 hour urine test for 5-hydroxy-indoleacetic acid excretion
- (D) Serial 24 hour test for catecholamines, metanephrines and vanillylmandelic acid excretion

Correct Answer: (D) Serial 24 hour test for catecholamines, metanephrines and vanillylmandelic acid excretion

SOLUTION:

This case is built from three clues: the patient's own headache and sweating attacks, her thyroid nodule, and her mother's history of renal calculi and death after a neck mass. Together these point to a familial syndrome that combines a thyroid cancer, a parathyroid tumour and an adrenal tumour, and we must decide what to check before thyroid surgery.

1. **Measurement of thyroid hormones:** this only tells us if the thyroid is overactive or underactive. The patient shows no sign of thyrotoxicosis already, and this test does not protect her on the operating table, so it does not answer the real danger here.
2. **Serial calcium, phosphorus, protein and alkaline phosphatase:** this checks for a parathyroid problem, which matches the mother's renal calculi, but a missed parathyroid

tumour will not kill the patient during anaesthesia. It is useful but not the most urgent step.

3. **24 hour urine 5-hydroxy-indoleacetic acid:** this is the screening test for carcinoid tumours, and nothing in the story points to carcinoid disease, so this option is out of place.

4. **Serial catecholamines, metanephrines and vanillylmandelic acid:** episodic headache and sweating are the hallmark symptoms of a pheochromocytoma, an adrenal tumour that is part of the same inherited syndrome suggested by the mother's neck mass and renal calculi. An unrecognised pheochromocytoma can cause a fatal surge in blood pressure the moment anaesthesia or surgery begins.

Because a missed pheochromocytoma can be fatal on the operating table, it has to be excluded first, using the catecholamine, metanephrine and vanillylmandelic acid test, before the thyroid is touched.

Let's summarize:

- Headache and sweating plus a family pattern of thyroid and parathyroid tumours suggest MEN2A, which also carries pheochromocytoma.
- Pheochromocytoma must be screened for and treated before any operation, since it is the one finding that can turn fatal under anaesthesia.

The correct test to order first is the serial catecholamine, metanephrine and vanillylmandelic acid excretion test.

Quick Tip: The family history fits MEN2A; always rule out pheochromocytoma before any surgery in such patients.

101. During surgery for aortic arch aneurysm under deep hypothermic circulatory arrest, which of the following anaesthetic agents, when administered prior to circulatory arrest, also provides cerebral protection?

- (A) Etomidate
- (B) Thiopental sodium
- (C) Propofol
- (D) Ketamine

Correct Answer: (B) Thiopental sodium

SOLUTION:

Deep hypothermic circulatory arrest stops blood flow completely for a limited window during aortic arch surgery. Cooling the body is the main way the brain is protected, but a drug is also given just before the arrest to add extra protection, and we have to identify it.

1. **Etomidate:** lowers brain oxygen use to some degree, but it suppresses the adrenal gland's own hormone output even with one dose. In a long, stressful cardiac case this is an unwanted side effect, so it is not the agent chosen for this role.
2. **Thiopental sodium:** a barbiturate that pushes brain activity down to burst suppression on the EEG, sharply cutting how much oxygen the brain needs. This is the long standing drug given before the circulation is stopped in aortic arch surgery.
3. **Propofol:** can reduce brain metabolism too, but it lowers blood pressure more strongly than thiopental, which is risky right before a period with no blood flow in an already fragile cardiac patient.

4. **Ketamine:** actually increases brain blood flow and oxygen use in normal practice, the opposite of what cerebral protection needs, so it is ruled out.

Among these, thiopental sodium is the one built for this exact job, cutting cerebral oxygen demand through burst suppression right before the brain loses its blood supply.

Let's summarize:

- Cerebral protection before circulatory arrest works by lowering the brain's oxygen need, and thiopental sodium is the classic drug for this.
- Etomidate risks adrenal suppression, propofol risks a bigger blood pressure drop, and ketamine raises rather than lowers brain metabolism.

The agent used for cerebral protection before circulatory arrest is thiopental sodium.

Quick Tip: Barbiturates cut brain oxygen use and cause burst suppression, the classic protective step before circulatory arrest.

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102. A 6 year old boy is scheduled for examination of the eye under anaesthesia. The father informs that for the past 6 months the child has been developing progressive weakness of both legs. His elder sibling had died at age 14 years. Which drug would you definitely avoid during anaesthetic management?

- (A) Succinylcholine
- (B) Thiopentone
- (C) Nitrous Oxide
- (D) Vecuronium

Correct Answer: (A) Succinylcholine

SOLUTION:

The child has slowly worsening weakness in both legs, and his older sibling died at just 14 years old. This pattern points toward an undiagnosed muscular dystrophy, and the question wants the drug that must be kept away from him during anaesthesia.

1. **Succinylcholine:** in muscle disease, acetylcholine receptors spread across the entire muscle membrane instead of staying at the neuromuscular junction. Succinylcholine opens all of them at once, flooding the blood with potassium fast enough to stop the heart, and it can also set off a malignant hyperthermia like reaction. This drug carries a real risk of sudden death here and must not be given.
2. **Thiopentone:** a routine induction agent with no special danger tied to muscle disease, so it stays safe to use.
3. **Nitrous oxide:** an inhaled anaesthetic gas that plays no part in the potassium surge problem, so there is no reason to withhold it.
4. **Vecuronium:** a non depolarising relaxant. It may act longer than usual in a myopathic child, but it does not trigger the dangerous potassium surge that succinylcholine does, so it can be used carefully.

Given the strong suspicion of muscular dystrophy from the history, succinylcholine is the one drug that must be avoided completely.

Let's summarize:

- Progressive leg weakness plus an early sibling death is a red flag for muscular dystrophy, even without lab confirmation.
- Succinylcholine can cause fatal hyperkalemia in such patients and must never be given.

The drug to definitely avoid is succinylcholine.

Quick Tip: Progressive limb weakness with an early sibling death suggests muscular dystrophy; never give succinylcholine here.

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103. In volume cycled respiration the inspiratory flow rate is set at:

- (A) 140-160 L/min
- (B) 110-130 L/min
- (C) 60-100 L/min
- (D) 30-50 L/min

Correct Answer: (C) 60-100 L/min

SOLUTION:

Volume cycled ventilation pushes a fixed tidal volume into the lungs on every breath, and the inspiratory flow rate is simply how fast that gas is delivered. We need the range this flow rate is normally set to.

1. **140-160 L/min:** far too fast for routine use. Such a high flow drives up peak airway pressure and delivers the breath unevenly through the lungs.

2. **110-130 L/min**: still faster than needed for standard ventilation, carrying the same risk of high pressure and uneven gas distribution.
3. **60-100 L/min**: fast enough to deliver the full tidal volume within a normal inspiratory time, yet gentle enough to keep airway pressures and gas flow patterns safe. This is the practical, commonly used range.
4. **30-50 L/min**: on the slow side, and may fail to deliver an adequate tidal volume within a normal breath time in many adult patients, so it is not the typical setting.

Balancing speed of delivery against airway pressure and even gas distribution, the flow rate that fits best is 60-100 L/min.

Let's summarize:

- Inspiratory flow rate must be fast enough to finish delivering the tidal volume in time, but not so fast that airway pressure spikes.
- 60-100 L/min is the standard working range for volume cycled ventilation.

The correct inspiratory flow rate range is 60-100 L/min.

Quick Tip: Volume cycled ventilators are usually run at a moderate inspiratory flow of about 60-100 L/min.

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104. Which of the following statements is NOT correct for vecuronium?

- (A) It has a high incidence of cardiovascular side effects
- (B) It has short duration of neuromuscular block
- (C) In usual doses, dose adjustment is not required in kidney disease
- (D) It has high lipophilic property

Correct Answer: (A) It has a high incidence of cardiovascular side effects

SOLUTION:

Vecuronium is a common non depolarising muscle relaxant, and this question wants the one statement about it that is false.

1. **High incidence of cardiovascular side effects:** vecuronium is prized precisely because it barely touches heart rate or blood pressure at normal doses, unlike pancuronium. Calling it high risk for cardiovascular side effects goes against its known profile, so this statement is false.
2. **Short duration of neuromuscular block:** compared with long acting relaxants, vecuronium wears off in a comparatively short time, which matches how it is actually used, so this statement holds up.
3. **No dose adjustment needed in kidney disease at usual doses:** since vecuronium leaves the body mainly through the liver and bile rather than the kidney, a single normal dose in a patient with kidney disease does not need special adjustment, making this statement correct.
4. **High lipophilic property:** as an aminosteroid compound, vecuronium is fat soluble, and this is exactly what allows liver uptake and biliary clearance, so this statement is also correct.

Only the claim about frequent cardiovascular side effects goes against what vecuronium is actually known for, so that is the incorrect statement.

Let's summarize:

- Vecuronium is valued for its cardiovascular stability, not for causing heart side effects.
- Its short duration, safe use in usual renal impairment doses, and lipophilic nature are all accurate.

The incorrect statement is that vecuronium has a high incidence of cardiovascular side effects.

Quick Tip: Vecuronium is known for cardiovascular stability, so calling it high risk for heart side effects is the wrong statement.

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105. A 10 year old child presented with headache, vomiting, gait instability and diplopia. On examination he had papilloedema and gait ataxia. The most probable diagnosis is:

- (A) Hydrocephalus
- (B) Brain stem Tumour
- (C) Suprasellar tumour
- (D) Midline posterior fossa tumour

Correct Answer: (D) Midline posterior fossa tumour

SOLUTION:

This 10 year old has two groups of findings: headache, vomiting and papilloedema, which signal raised pressure inside the skull, and gait instability with gait ataxia, which signals trouble in the balance centre of the brain. The diagnosis has to explain both groups at once.

1. **Hydrocephalus:** this is fluid build up inside the brain and does explain the raised pressure findings, but it is really the downstream effect of something blocking cerebrospinal fluid flow. On its own it does not account for the specific gait ataxia, so it is too broad an answer.
2. **Brain stem tumour:** this usually shows up with cranial nerve palsies and weakness from pressure on the long nerve tracts running through the brain stem, rather than the prominent truncal ataxia seen in this child, so it is a weaker fit.
3. **Suprasellar tumour:** tumours in this location, like craniopharyngioma, typically cause visual field loss and hormone problems from pressing on the optic chiasm and pituitary gland, not gait ataxia, so this does not match either.
4. **Midline posterior fossa tumour:** a tumour such as medulloblastoma growing from the cerebellar vermis presses on the fourth ventricle, blocking cerebrospinal fluid drainage and producing the headache, vomiting and papilloedema of raised pressure. At the same time it damages the vermis directly, producing truncal gait ataxia. The diplopia also fits, since raised pressure commonly stretches the sixth cranial nerve as a false localising sign.

Only a midline posterior fossa tumour ties every finding, the raised pressure signs and the gait ataxia, into one single lesion.

Let's summarize:

- Headache, vomiting and papilloedema point to raised intracranial pressure.
- Gait, truncal ataxia points to the cerebellar vermis, which sits in the midline posterior fossa.

The most probable diagnosis is a midline posterior fossa tumour.

Quick Tip: Raised ICP signs plus truncal gait ataxia in a child point to a midline posterior fossa mass like medulloblastoma.

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106. The most recent advance in non-invasive cardiac output monitoring is the use of:

- (A) PA catheter
- (B) Thermodilution technique
- (C) Echocardiography
- (D) Electrical impedance cardiograph technology

Correct Answer: (D) Electrical impedance cardiograph technology

SOLUTION:

Step 1: Group the four choices by how they are done.

A PA catheter sits inside the pulmonary artery, and thermodilution is the calculation done through that same catheter. Both need a line placed inside the heart's blood vessels, so both are invasive.

Step 2: Compare the two non-invasive choices left.

Echocardiography and impedance cardiography do not enter the body, so both qualify as non-invasive. The question is which one is the

newer, more recent addition.

Step 3: Recall what impedance cardiography adds.

Electrical impedance (bioimpedance) cardiography uses surface electrodes to track small changes in chest impedance caused by blood moving through the aorta with each heartbeat, and turns that into a continuous cardiac output value. This bedside, wire free approach was the newer non-invasive technique when it became available, compared with the long established echo technique.

Step 4: State the answer.

Electrical impedance cardiography is the most recent non-invasive advance.

Electrical impedance cardiograph technology

Quick Tip: Rule out anything that needs a catheter first; among the non-invasive options, pick the newest bedside technology.

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107. While carrying a heavy suitcase, the downward dislocation of the glenohumeral joint is resisted by all of the following muscles except:

- (A) Deltoid
- (B) Coracobrachialis
- (C) Short head of biceps
- (D) Latissimus dorsi

Correct Answer: (B) Coracobrachialis ; (C) Short head of biceps

SOLUTION:

This question asks which muscles do NOT help stop the humeral head from slipping down out of the glenoid cavity when a heavy weight loads the arm, as when carrying a suitcase.

1. **Deltoid:** Runs almost vertically over the shoulder from the scapular spine and clavicle to the humerus. Its tone is one of the main forces holding the head up against the glenoid, so it does resist downward slip.
2. **Coracobrachialis:** Starts on the coracoid process and runs down and medially to the humeral shaft. Its pull is oblique rather than vertical, so it gives little real support against the head dropping down, and the accepted key marks it as one of the two exceptions.
3. **Short head of biceps:** Also starts on the coracoid process and crosses two joints down to the radius. Like coracobrachialis, its line of pull is oblique, so it too is marked as an exception in the accepted key.
4. **Latissimus dorsi:** A broad adductor and medial rotator running from the trunk up to the front of the humerus. The accepted key groups it with deltoid as contributing to resisting inferior displacement, though this is one of the weaker, more debated parts of the answer.

Because two options, coracobrachialis and short head of biceps, are both accepted as not resisting downward dislocation, this question is treated as a multiple correct answer item, matching the original paper's own "2/3" key.

Let's summarize:

- Deltoid and supraspinatus type vertical fibre muscles resist inferior dislocation the most.

- Coracobrachialis and short head of biceps pull more obliquely and are the accepted exceptions here.

So the answer is coracobrachialis and short head of biceps.

Quick Tip: Think about which muscles cross the joint with vertical fibres to hold the humeral head up; this exact question is a known ambiguous one with two accepted answers.

• • •

108. A 19 year old boy fell from a motorbike onto his shoulder. The doctor diagnosed him as a case of Erb's paralysis. All of the following signs and symptoms will be observed except:

- (A) Loss of abduction at shoulder joint
- (B) Loss of lateral rotation
- (C) Loss of pronation at radioulnar joint
- (D) Loss of flexion at elbow joint

Correct Answer: (C) Loss of pronation at radioulnar joint

SOLUTION:

Step 1: Picture the mechanism of injury.

A fall directly onto the shoulder pushes the head away from the shoulder, stretching the upper roots of the brachial plexus, C5 and C6. This is the classic cause of Erb's palsy, and it fits the story of the motorbike fall.

Step 2: Match muscles to movements.

C5, C6 supply deltoid, supraspinatus, infraspinatus, teres minor, biceps, and brachialis. Losing these means the arm cannot abduct

well, cannot rotate outward, and cannot flex fully at the elbow. The arm hangs limp and rotated in at the side, the well known "waiter's tip" posture.

Step 3: Focus on the forearm.

Biceps is also the strongest supinator. With it out of action, the forearm cannot supinate, and because nothing pulls it the other way, it tends to sit pronated. So supination is what is lost, not pronation, pronation still works and even becomes the resting position.

Step 4: Match this back to the options.

Loss of abduction, loss of lateral rotation, and loss of elbow flexion are all real findings in Erb's palsy. Loss of pronation is not, since pronation is spared.

Step 5: Conclude.

Loss of pronation at radioulnar joint

Quick Tip: Erb's palsy hits C5, C6; work out which movements those roots control, then find the one movement that is NOT one of them.

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109. A 4 and a half year old girl child always had to wear warm socks even in summer season. On physical examination, she was found to have high blood pressure and her femoral pulse was weak as compared to her radial and carotid pulse. A chest radiograph showed remarkable notching of the ribs along their lower borders. This was due to:

- (A) Femoral artery thrombosis
- (B) Coarctation of aorta
- (C) Raynaud's disease
- (D) Takayasu's arteritis

Correct Answer: (B) Coarctation of aorta

SOLUTION:

Step 1: Work from the pulses first.

A radial pulse that feels stronger than the femoral pulse tells us the lower body is getting less blood than the upper body. That single finding already points to a block somewhere in the aorta between the arm vessels and the leg vessels.

Step 2: Add the blood pressure and rib finding.

High blood pressure measured in the arms fits with blood being pushed hard into the upper body above a narrowing, while the body opens up small side channels through the intercostal arteries to feed the lower body around the block. Over years these enlarged vessels press on and erode the lower border of the ribs, which shows up as notching on X-ray.

Step 3: Name the lesion that fits all three findings.

A narrowing of the aorta itself, coarctation of the aorta, is the one condition that produces this exact trio: high upper limb pressure, weak delayed femoral pulse, and rib notching from collateral vessels.

Step 4: Check why the other choices fail.

Femoral thrombosis gives a sudden painful pulseless leg, not chronic cold feet with hypertension. Raynaud's disease affects fingers with

cold triggered colour change and no rib notching. Takayasu's arteritis can weaken pulses but does not typically give this same rib notching pattern, and is an inflammatory large vessel disease rather than a fixed narrowing.

Step 5: Conclude.

Coarctation of aorta

Quick Tip: Weak femoral pulse plus strong radial pulse plus rib notching is the classic triad for one specific congenital lesion.

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110. A 40 year old man was repairing his wooden shed on a Sunday afternoon. By afternoon, he felt that the hammer was getting heavier and heavier. He felt pain on the lateral side of the elbow and also found that squeezing water out of a sponge hurt his elbow. Which of the following muscles are most likely involved?

- (A) Biceps brachii and supinator
- (B) Flexor digitorum superficialis
- (C) Extensor carpi radialis longus and brevis
- (D) Triceps brachii and anconeus

Correct Answer: (C) Extensor carpi radialis longus and brevis

SOLUTION:

Step 1: Note where the pain is.

The pain sits on the outside of the elbow and gets worse with gripping tasks like swinging a hammer or wringing a sponge. Pain right at that

spot, worsened by grip, is the hallmark of tennis elbow.

Step 2: Recall which tendons attach there.

The lateral epicondyle is the shared origin of the wrist extensors. Extensor carpi radialis brevis is the classic culprit, working alongside extensor carpi radialis longus, since both must contract hard to hold the wrist steady in slight extension every time the hand grips something firmly.

Step 3: Eliminate muscles from the wrong side or wrong job.

Biceps and supinator sit in front and handle flexion and supination, not gripping stability. Flexor digitorum superficialis comes from the medial epicondyle, the opposite side, giving medial pain instead. Triceps and anconeus are posterior extensors of the elbow, unrelated to lateral pain from gripping.

Step 4: Conclude.

Extensor carpi radialis longus and brevis

Quick Tip: Lateral elbow pain with gripping activities is tennis elbow; find the muscles attached at the lateral epicondyle.

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111. A 16 year old girl failed her final examination. Disgusted with life, she cut across the front of her wrist at the flexor retinaculum. She was rushed to hospital, and the surgeon noticed that the cut was superficial. All of the following structures would have been damaged except:

- (A) Ulnar nerve
- (B) Median nerve
- (C) Palmar cutaneous branch of median nerve
- (D) Superficial branch of radial artery

Correct Answer: (B) Median nerve

SOLUTION:

Step 1: Separate deep structures from superficial ones.

Only structures lying above (superficial to) the flexor retinaculum can be cut by a wound the surgeon describes as superficial. Anything travelling inside the carpal tunnel, deep to the retinaculum, is protected.

Step 2: Sort the four options.

The ulnar nerve travels through Guyon's canal, superficial to the retinaculum. The palmar cutaneous branch of the median nerve branches off before the wrist and stays superficial. The superficial branch of the radial artery, as its name says, runs in the superficial plane. All three of these sit in the path of a shallow cut.

Step 3: Look at the median nerve trunk.

The median nerve itself, not its small cutaneous branch, passes through the carpal tunnel beneath the flexor retinaculum along with the flexor tendons. This puts it in the deep plane, out of reach of a superficial wound.

Step 4: Conclude.

Since the wound was superficial, the one structure spared is the median nerve trunk.

Median nerve

Quick Tip: Ask which of these structures sits deep to the flexor retinaculum inside the carpal tunnel, that one is protected from a superficial cut.

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112. In dislocation of the jaw, displacement of the articular disc beyond the temporomandibular (TM) joint results from spasm or excessive contraction of which of the following muscles?

- (A) Buccinator
- (B) Lateral pterygoid
- (C) Masseter
- (D) Temporalis

Correct Answer: (B) Lateral pterygoid

SOLUTION:

Step 1: Ask which muscle attaches to the disc.

Of all the muscles of mastication, only the lateral pterygoid sends fibres, from its upper head, directly into the articular disc of the TMJ as well as into the neck of the condyle below.

Step 2: Follow what happens on overcontraction.

Because it attaches to both the disc and the condyle, an overactive or spastic lateral pterygoid drags them both forward, past the bony ridge in front of the joint (the articular eminence). The jaw then gets stuck open, unable to slide back, this is a TMJ dislocation with the disc carried forward too.

Step 3: Check the closing muscles.

Masseter and temporalis pull the jaw shut and upward, the opposite direction needed to cause this forward dislocation, so their overactivity would not push the disc past the eminence. Buccinator is a cheek muscle with no attachment to the joint or disc at all.

Step 4: Conclude.

Lateral pterygoid

Quick Tip: Find the muscle that attaches to the articular disc itself and pulls the mandible forward.

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113. Following surgical removal of a firm nodular cancer swelling in the right breast and exploration of the right axilla, on examination the patient was found to have a winged scapula. This occurred due to injury to the

- (A) Subscapular muscle
- (B) Coracoid process of scapula
- (C) Long thoracic nerve
- (D) Circumflex scapular artery

Correct Answer: (C) Long thoracic nerve

SOLUTION:

This question checks whether you can connect a nerve injury during axillary surgery to a specific clinical sign, the winged scapula. Axillary

clearance for breast cancer runs close to several nerves, and the long thoracic nerve is the classic one at risk.

1. **Subscapular muscle:** This muscle sits on the front of the scapula and rotates the arm inward. Damaging it does not push the scapula away from the chest wall, so it does not fit.
2. **Coracoid process of scapula:** This is just a bony hook on the scapula where some muscles attach. It carries no nerve fibers of its own, so injuring it cannot paralyse a muscle.
3. **Long thoracic nerve:** This nerve runs down the side of the chest wall on the outer surface of serratus anterior, right through the field of axillary dissection. Cutting it paralyzes serratus anterior, and the scapula's medial border springs away from the ribs, giving the winged look.
4. **Circumflex scapular artery:** This is a vessel that supplies muscles around the scapula through the triangular space. Injury to it causes bleeding or poor blood supply, not a winged scapula.

The structure that fits both the surgical field and the clinical sign is the long thoracic nerve. Its loss stops serratus anterior from anchoring the scapula, so the bone lifts off the chest wall.

Let's summarize:

- Serratus anterior keeps the scapula flat against the ribs and is powered by the long thoracic nerve.
- The long thoracic nerve runs superficially in the axilla, making it easy to injure during lymph node clearance.

So the winged scapula in this patient is caused by injury to the long thoracic nerve.

Quick Tip: Winged scapula after axillary surgery points to serratus anterior palsy from long thoracic nerve injury.

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114. A 49-year-old man suffering from carcinoma of prostate was X-rayed. He showed areas of sclerosis and collapse of T10 and T11 vertebra in X-ray. The spread of this cancer to the above vertebrae was through

- (A) Sacral canal
- (B) Lymphatic vessels
- (C) Internal vertebral plexus of veins
- (D) Superior rectal veins

Correct Answer: (C) Internal vertebral plexus of veins

SOLUTION:

Step 1: Understanding the Concept:

Prostate cancer commonly causes sclerotic bone lesions in the spine even though the prostate sits nowhere near the thoracic vertebrae. The question wants the pathway that lets cancer cells jump that distance.

Step 2: Key Formula or Approach:

Cancer can spread by direct extension, lymphatics, or blood vessels. For prostate cancer reaching mid thoracic vertebrae, the important blood route is a special valveless venous network around the spine, called the internal vertebral (Batson's) venous plexus.

Step 3: Detailed Explanation:

The prostatic venous plexus connects with this vertebral plexus. Since

the vertebral plexus has no valves, blood, and any tumour cells inside it, can flow in either direction depending on pressure changes in the chest and abdomen, for example during coughing. This allows prostate cancer cells to travel upward and lodge in the vertebral bodies, including T10 and T11, producing the sclerotic collapse seen on X-ray. The sacral canal is only a bony passage and carries no blood or cells. Lymphatic spread mainly reaches pelvic lymph nodes, not distant vertebrae in this pattern. Superior rectal veins belong to the portal drainage of the rectum and have no connection to prostate metastasis.

Step 4: Final Answer:

The correct route is the internal vertebral plexus of veins, option (3).

Quick Tip: Prostate cancer reaches distant vertebrae via Batson's valveless internal vertebral venous plexus.

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115. The approximate number of genes contained in the human genome is

- (A) 40,000
- (B) 30,000
- (C) 80,000
- (D) 1,00,000

Correct Answer: (B) 30,000

SOLUTION:

This question is really about how the estimated gene count of the human genome changed once scientists could actually read the full

DNA sequence instead of guessing from its length.

1. **40,000**: This number is higher than the confirmed count from genome sequencing and does not match the figure reported once the Human Genome Project finished its analysis.
2. **30,000**: This is the figure that came directly out of the completed genome sequence in the early 2000s, later narrowed further to about 19,000 to 20,000 protein coding genes with better annotation. Of the four choices, it sits closest to the real, confirmed number.
3. **80,000**: This was an older, pre-sequencing estimate used in some classic textbooks, based on rough calculations rather than actually reading the genome. It is now known to be far too high.
4. **1,00,000**: This was the earliest and roughest guess, also from before the genome was sequenced, and is even further from the true figure.

Once the human genome was fully sequenced, the gene count dropped to roughly 30,000 and was later refined closer to 20,000. So the best answer among the given options is 30,000, not the older textbook figure of 80,000 or 100,000.

Let's summarize:

- Pre-sequencing textbooks guessed 80,000 to 100,000 genes based on DNA length alone.
- Direct sequencing in the Human Genome Project brought the real count down to about 30,000, later refined to about 20,000.

So the approximate number of genes in the human genome, based on actual sequencing data, is 30,000.

Quick Tip: Post-Human Genome Project sequencing puts the count near 30,000, not the older 80,000-100,000 estimate.

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116. Which of the following would distinguish hydrocephalus due to aqueductal stenosis when compared to that due to Dandy-Walker phenomenon

- (A) Third ventricle size
- (B) Posterior fossa volume
- (C) Lateral ventricular size
- (D) Head circumference

Correct Answer: (B) Posterior fossa volume

SOLUTION:

Both aqueductal stenosis and Dandy-Walker malformation lead to hydrocephalus, but the site of CSF blockage is different in each, and this changes what the scan looks like.

1. **Third ventricle size:** In aqueductal stenosis the block sits just below the third ventricle, so it enlarges. In Dandy-Walker malformation the whole ventricular system upstream of the fourth ventricle can also back up, so the third ventricle can be large here too. This does not reliably tell the two apart.
2. **Posterior fossa volume:** In Dandy-Walker malformation the fourth ventricle itself is cystic and the vermis is underdeveloped, and this cyst physically enlarges the posterior fossa. In aqueductal stenosis the blockage is above the fourth ventricle, so

the posterior fossa keeps its normal size. This difference is consistent and specific.

3. **Lateral ventricular size:** Both conditions cause the lateral ventricles to dilate because CSF cannot drain past the block. This feature is shared, not distinguishing.
4. **Head circumference:** This grows with any significant hydrocephalus regardless of its cause, so it does not point to which condition is present.

The posterior fossa is the structure whose size actually differs between the two conditions: enlarged in Dandy-Walker malformation, normal in aqueductal stenosis.

Let's summarize:

- Aqueductal stenosis blocks CSF above the fourth ventricle, sparing the posterior fossa.
- Dandy-Walker malformation involves a cystic fourth ventricle that enlarges the posterior fossa.

So posterior fossa volume is the feature that distinguishes hydrocephalus due to aqueductal stenosis from that due to Dandy-Walker malformation.

Quick Tip: Dandy-Walker malformation enlarges the posterior fossa; aqueductal stenosis does not.

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117. In hemoglobin, the innate affinity of Heme for carbon monoxide is diminished by the presence of

- (A) His F-8
- (B) His E-7
- (C) Gly B-6
- (D) Thr C-4

Correct Answer: (B) His E-7

SOLUTION:

Step 1: Understanding the Concept:

Free heme by itself binds carbon monoxide far more strongly than oxygen because carbon monoxide likes to sit at a straight 180 degree angle to the iron. Inside the hemoglobin molecule this huge preference is cut down a lot, and the question asks which amino acid residue is responsible.

Step 2: Key Formula or Approach:

Two histidines flank the heme iron in each globin chain: the proximal His F8, which bonds directly to iron and anchors the heme, and the distal His E7, which sits in the binding pocket but does not bond to iron directly. The distal residue is the one that shapes how a gas molecule can approach the iron.

Step 3: Detailed Explanation:

Because His E7 occupies space directly across the pocket, it physically blocks carbon monoxide from lining up straight with the iron, the geometry carbon monoxide prefers. Carbon monoxide is forced into a bent, less stable angle instead, which weakens its binding. Oxygen, which naturally prefers a bent angle anyway, is not hindered nearly as much. This steric effect from His E7 brings hemoglobin's preference for carbon monoxide over oxygen down from about 25,000 times (as

in free heme) to about 200 times. His F8 is important for holding the heme in place but sits on the far side from the gas binding site, so it does not create this steric block. Gly B6 and Thr C4 are part of the protein scaffold and are not positioned to interfere with gas geometry at the iron.

Step 4: Final Answer:

His E7, the distal histidine, is the residue that lowers heme's natural affinity for carbon monoxide, option (2).

Quick Tip: The distal histidine (His E7) sterically hinders the linear binding geometry that carbon monoxide prefers.

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118. The activity of an enzyme having Michaelis constant (K_m) = $x \mu\text{M}$ will be 80% of V_{max} at a substrate concentration of

- (A) $2 \times \mu\text{M}$
- (B) $4 \times \mu\text{M}$
- (C) $8 \times \mu\text{M}$
- (D) $10 \times \mu\text{M}$

Correct Answer: (B) $4 \times \mu\text{M}$

SOLUTION:

Step 1: Understanding the Concept:

Enzyme velocity follows $v = \frac{V_{\text{max}}[S]}{K_m + [S]}$. This question fixes the velocity at 80% of V_{max} and asks what substrate concentration produces that, in terms of K_m .

Step 2: Key Formula or Approach:

Set $v = 0.8V_{max}$ and solve the Michaelis-Menten equation for $[S]$ in terms of K_m .

Step 3: Detailed Explanation:

$$0.8V_{max} = \frac{V_{max}[S]}{K_m + [S]}$$

Cancel V_{max} from both sides:

$$0.8 = \frac{[S]}{K_m + [S]}$$

Cross multiply:

$$0.8K_m + 0.8[S] = [S]$$

Move terms with $[S]$ to one side:

$$0.8K_m = 0.2[S]$$

Divide both sides by 0.2:

$$[S] = 4K_m$$

Given $K_m = x \mu M$, this becomes $[S] = 4x \mu M$. This makes sense because the Michaelis-Menten curve is a rectangular hyperbola that approaches V_{max} slowly, so getting to a high fraction like 80% needs a substrate level well above K_m , specifically four times as much.

Step 4: Final Answer:

$$4x \mu M$$

The enzyme reaches 80% activity at a substrate concentration of $4x \mu M$, option (2).

Quick Tip: Use $v = 0.8 V_{max}$ in the Michaelis-Menten equation and solve for $[S]$ in terms of K_m .

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119. The antibiotic that inhibits protein synthesis by premature chain termination and which structurally resembles aminoacyl tRNA is

- (A) Tetracycline
- (B) Chloramphenicol
- (C) Puromycin
- (D) Erythromycin

Correct Answer: (C) Puromycin

SOLUTION:

This question is testing the unique mechanism of puromycin, which is different from the other classic protein synthesis inhibitors because it works by disguising itself as a piece of the translation machinery rather than by blocking a step directly.

1. **Tetracycline:** Works by physically blocking the A site of the small ribosomal subunit, preventing any aminoacyl tRNA from

docking there. It does not resemble tRNA itself, it just gets in the way of tRNA binding.

2. **Chloramphenicol:** Binds the 50S subunit and blocks the peptidyl transferase center, the enzyme site that links amino acids together. This stops chain growth but does not involve mimicking a tRNA structure.
3. **Puromycin:** Its structure closely copies the aminoacyl end of a real tRNA, so the ribosome mistakes it for one and lets it enter the A site and join the growing chain. Because puromycin cannot support further chain extension after that, the protein is released early and unfinished. This matches both parts of the question: tRNA mimicry and premature chain ending.
4. **Erythromycin:** Attaches to the 50S subunit and blocks translocation, the step where the ribosome shifts along the mRNA after each peptide bond. It does not act as a tRNA mimic.

Only puromycin fits both clues in the question: it looks like aminoacyl tRNA and it causes the chain to stop early once it is incorporated.

Let's summarize:

- Puromycin mimics aminoacyl tRNA and gets built into the growing peptide chain.
- Because it cannot extend the chain further, the protein is released prematurely and is non-functional.

So the antibiotic described in the question is puromycin.

Quick Tip: Puromycin mimics the aminoacyl end of tRNA and causes premature release of the growing peptide chain.



120. In post-hepatic (obstructive) jaundice, the concentration of conjugated bilirubin in the blood is higher than that of unconjugated bilirubin because

- (A) There is an increased rate of destruction of red blood cells.
- (B) The unconjugated bilirubin is trapped by a bile stone produced in the bile duct.
- (C) The conjugation process of bilirubin in the liver remains operative without any interference.
- (D) The UDP-glucuronosyl-transferase activity is increased manifold in obstructive jaundice.

Correct Answer: (C) The conjugation process of bilirubin in the liver remains operative without any interference.

SOLUTION:

This question tests the difference between the three types of jaundice: pre-hepatic (hemolytic), hepatic, and post-hepatic (obstructive). The clue is that conjugated bilirubin, not unconjugated, is high in the blood.

1. **Increased red cell destruction:** A higher breakdown rate of red blood cells would flood the liver with unconjugated bilirubin, faster than it can be conjugated, so unconjugated bilirubin would rise. This is the pattern of hemolytic jaundice, not obstructive jaundice, so this option does not fit.
2. **Unconjugated bilirubin trapped by a bile stone:** A stone in the bile duct sits after the conjugation step, so it can only trap bilirubin that has already been conjugated, not the unconjugated form. This statement mixes up which form gets trapped.

3. **Conjugation process remains operative:** In obstructive jaundice the liver cells and the conjugating enzyme are untouched, so bilirubin keeps getting conjugated at a normal pace. Since the duct beyond the liver is blocked, this newly conjugated bilirubin has nowhere to go and spills back into the blood, which is exactly why the conjugated fraction dominates.
4. **Increased UDP-glucuronosyl-transferase activity:** There is no known rise in this enzyme's activity during duct obstruction, the enzyme works at its baseline rate, the problem is purely a blocked exit route.

The correct explanation is that conjugation continues normally in the liver cells, so the bilirubin reaching the blood is mainly in its conjugated form. This matches statement (C).

Let's summarize:

- Obstructive jaundice blocks the path after conjugation, so conjugated bilirubin regurgitates into blood.
- Hemolytic jaundice raises unconjugated bilirubin because red cell breakdown outpaces the liver's conjugating capacity.

So the reason conjugated bilirubin dominates in post-hepatic jaundice is that the conjugation step in the liver keeps working without interference.

Quick Tip: In obstructive jaundice the block is after conjugation, so the liver keeps conjugating bilirubin normally, and it backs up into blood.

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121. If a biochemical test gives the same reading for a sample on repeated testing, it is inferred that the measurement is:

- (A) Precise.
- (B) Accurate.
- (C) Specific.
- (D) Sensitive.

Correct Answer: (A) Precise.

SOLUTION:

This question checks whether you can tell apart precision, accuracy, specificity, and sensitivity, four terms that are often confused in laboratory medicine.

1. **Precise:** Precision is about repeatability, if you test the same sample again and again and keep getting the same number, the test is precise. This is exactly the situation described in the question, so this is the answer.
2. **Accurate:** Accuracy is about closeness to the true value, not about repeatability. A test could repeat the same wrong number every time, that would be precise but not accurate. The question gives no information about a true value to compare against, so accuracy cannot be the answer here.
3. **Specific:** Specificity is a screening test property, it tells us how well the test correctly rules out people who do not have the disease. It has nothing to do with getting the same reading twice on one sample.
4. **Sensitive:** Sensitivity is also a screening test property, it tells us how well the test correctly picks up people who do have the disease. Again, this has nothing to do with repeat readings on a single sample.

Getting the same reading on repeated testing of the same sample is the textbook definition of precision, so option (A) is correct.

Let's summarize:

- Precision = repeatability of a result on the same sample.
- Accuracy = closeness of a result to the true value.
- Sensitivity and specificity apply to how well a test classifies diseased versus healthy people, not to repeat readings.

So the correct term for a test that gives the same reading every time is precise.

Quick Tip: Same reading every time on the same sample is about repeatability, which is precision, not accuracy.

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122. An alpha helix of a protein is most likely to be disrupted if a missense mutation introduces the following amino acid within the alpha helical structure:

- (A) Alanine.
- (B) Aspartic acid.
- (C) Tyrosine.
- (D) Glycine.

Correct Answer: (D) Glycine.

SOLUTION:

An alpha helix needs a backbone that can hold a steady, repeating twist, stabilized by hydrogen bonds every fourth residue. Some amino

acids fit this shape easily, others fight against it. Let's check each option.

1. **Alanine:** Its side chain is just a methyl group, small and simple. This lets the backbone sit comfortably in the helical twist, alanine is in fact one of the strongest helix formers, so putting it into a helix would not disrupt it.
2. **Aspartic acid:** This side chain carries a negative charge at physiological pH. It can fit within a helix without much trouble, though several charged residues of the same sign placed close together can create some repulsion. On its own it is not a classic helix disrupter.
3. **Tyrosine:** A bulky ring-shaped side chain, it can add some steric bulk but tyrosine residues are regularly found inside natural alpha helices, so it is tolerated reasonably well.
4. **Glycine:** Its side chain is only a single hydrogen atom, giving the backbone extra freedom to rotate that other residues do not have. This flexibility works against the fixed, repeating angles an alpha helix depends on, so glycine is the classic helix breaker.

Between the four, only glycine's lack of a real side chain gives the backbone enough extra freedom to break the strict geometry of the helix.

Let's summarize:

- Alpha helices need a backbone locked into regular phi and psi angles.
- Glycine's tiny side chain gives too much backbone freedom, making it a helix breaker along with proline.

So a missense mutation that introduces glycine into a helical region is the one most likely to disrupt that alpha helix.

Quick Tip: Glycine's tiny side chain gives the backbone extra flexibility, breaking the fixed geometry of an alpha helix.

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123. During gluconeogenesis, reducing equivalents from mitochondria to the cytosol are transported by:

- (A) Malate.
- (B) Aspartate.
- (C) Glutamate.
- (D) Oxaloacetate.

Correct Answer: (A) Malate.

SOLUTION:

Gluconeogenesis needs NADH out in the cytosol, but NADH itself is generated mainly inside mitochondria and cannot cross the inner mitochondrial membrane on its own. The cell solves this by disguising the reducing power as a small molecule that can cross. Let's look at each option.

1. **Malate:** Oxaloacetate inside the mitochondria is reduced to malate, using up mitochondrial NADH in the process. Malate then passes through a transporter in the inner membrane into the cytosol, where it is oxidized back to oxaloacetate, releasing NADH exactly where gluconeogenesis needs it. This is the carrier molecule the question is asking about.
2. **Aspartate:** Aspartate is part of the malate-aspartate shuttle too, but its usual job is to help move reducing equivalents into the

mitochondria, and to carry the carbon skeleton back in the reverse direction of this specific export route, not to directly ferry NADH out for gluconeogenesis.

3. **Glutamate:** Glutamate donates or accepts an amino group in transamination reactions that support the aspartate-oxaloacetate interconversion, it acts as a helper in the shuttle chemistry but is not itself the molecule that crosses carrying the reducing equivalent.
4. **Oxaloacetate:** Oxaloacetate cannot cross the inner mitochondrial membrane through the transporter used in this pathway, this is precisely why the cell converts it to malate first.

Since malate is the molecule that physically crosses the membrane carrying the reducing equivalent, and is then oxidized in the cytosol to release NADH, it is the correct answer.

Let's summarize:

- NADH cannot cross the mitochondrial membrane directly.
- Oxaloacetate is reduced to malate inside the mitochondria, malate crosses over, and is oxidized back to oxaloacetate in the cytosol, releasing NADH there.

So malate is the carrier that exports reducing equivalents from mitochondria to the cytosol during gluconeogenesis.

Quick Tip: Oxaloacetate is reduced to malate inside mitochondria, malate crosses the membrane, then gets oxidized back in the cytosol to release NADH.

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124. Separation of proteins on the basis of molecular size is achieved by:

- (A) Chromatography on a carboxymethyl (CM) cellulose column.
- (B) Iso-electric focusing.
- (C) Gel filtration chromatography.
- (D) Chromatography on a diethylaminoethyl (DEAE) cellulose column.

Correct Answer: (C) Gel filtration chromatography.

SOLUTION:

This question is testing whether you know which protein separation method depends on the size of the molecule rather than its electrical charge. Let's go through the four techniques listed.

1. **CM cellulose chromatography:** Carboxymethyl cellulose carries a negative charge on the column matrix, so it binds and separates positively charged proteins, this is a cation exchange method, based on charge, not size.
2. **Iso-electric focusing:** This technique separates proteins according to their isoelectric point, the pH value at which each protein has zero net charge. Proteins migrate through a pH gradient until they stop at their own isoelectric point, again this is a charge based separation, not a size based one.
3. **Gel filtration chromatography:** Also known as size exclusion chromatography, this method uses porous beads packed into a column. Large proteins are excluded from the small pores and pass quickly around the beads, while small proteins wander into the pores and are delayed. Molecules therefore come out of the column strictly in order of size, largest first, smallest last. This is exactly the size based method the question is asking about.
4. **DEAE cellulose chromatography:** Diethylaminoethyl cellulose carries a positive charge on the matrix, so it binds and

separates negatively charged proteins, an anion exchange method, again based on charge, not size.

Only gel filtration chromatography sorts proteins purely by their physical size as they pass through the porous gel beads.

Let's summarize:

- CM cellulose and DEAE cellulose chromatography are ion exchange methods, based on charge.
- Iso-electric focusing is based on isoelectric point, also a charge property.
- Gel filtration chromatography alone separates strictly by molecular size.

So the technique that separates proteins on the basis of molecular size is gel filtration chromatography.

Quick Tip: Size exclusion through porous gel beads, large molecules elute first, is the basis of gel filtration chromatography.

• • •

125. At physiological pH, DNA molecules are:

- (A) Positively charged.
- (B) Negatively charged.
- (C) Neutral.
- (D) Amphipathic.

Correct Answer: (B) Negatively charged.

SOLUTION:

This question is about the charge state of DNA at the pH found inside living cells, close to pH 7.4. The backbone structure of DNA holds the answer.

1. **Positively charged:** The DNA backbone has no group that carries a positive charge at this pH, so this option does not fit.
2. **Negatively charged:** DNA is built from a repeating chain of sugar and phosphate units. Each phosphate group has an acidic hydroxyl with a low pKa, so at physiological pH it loses its proton and becomes negatively charged. With one such phosphate for every nucleotide, the whole strand carries a large net negative charge. This is the property that makes DNA move toward the positive electrode during gel electrophoresis, a technique used every day in molecular biology labs.
3. **Neutral:** This would only be true if the phosphates were not ionized, but at physiological pH they are, so DNA is far from neutral.
4. **Amphipathic:** This term describes molecules like phospholipids, which have one part that likes water and one part that avoids it. DNA's backbone is uniformly charged and water loving all along its length, it does not have this split character, so amphipathic is not the right description of its charge.

The source answer key for this paper lists amphipathic as correct, but that does not match standard biochemistry. The ionized phosphate backbone giving DNA a net negative charge is basic, well established chemistry, so the correct choice here is negatively charged, and this has been corrected accordingly.

Let's summarize:

- The phosphodiester backbone of DNA carries ionized phosphate groups at physiological pH.
- This gives DNA a strong net negative charge, the basis for techniques like gel electrophoresis.

So at physiological pH, DNA molecules are negatively charged.

Quick Tip: The phosphate backbone of DNA is ionized at physiological pH, giving DNA a net negative charge.

• • •

126. One of the following enzymes is not a protein:

- (A) DNAase.
- (B) Abzyme.
- (C) Eco RI.
- (D) Ribozyme.

Correct Answer: (D) Ribozyme.

SOLUTION:

Most enzymes in the body are proteins, but there is one well known exception that this question is testing. Let's check each option in turn.

1. **DNAase:** This is an enzyme that breaks down DNA, and like the great majority of enzymes, it is a folded chain of amino acids, so it is a protein.
2. **Abzyme:** The name is short for antibody enzyme, it refers to an antibody that also works as a catalyst. Since every antibody is built from amino acid chains, an abzyme is a protein too.

3. **Eco RI:** This is a restriction enzyme from bacteria that cuts DNA at a specific recognition sequence. It is a standard protein enzyme, widely used in molecular biology labs for cutting DNA at defined sites.
4. **Ribozyme:** This is the exception. A ribozyme is a piece of RNA that can catalyze a chemical reaction on its own, without any protein component. A well known example is the peptidyl transferase activity of the ribosome, which is carried out by ribosomal RNA, not by the ribosomal proteins. Since ribozymes are made of nucleotides, not amino acids, they are not proteins.

Three of the four options, DNAase, Abzyme, and Eco RI, are all protein based catalysts. Only the ribozyme is built from RNA instead of protein.

Let's summarize:

- Most enzymes, including DNAase, Abzyme, and Eco RI, are proteins.
- Ribozymes are catalytic RNA molecules, the key exception to "all enzymes are proteins".

So the enzyme that is not a protein is the ribozyme.

Quick Tip: Ribozymes are catalytic RNA molecules, the one common exception to enzymes being proteins.

• • •

127. The following hormone does not have any intracellular receptor:

- (A) Vitamin D₃
- (B) Cortisone
- (C) Adrenaline
- (D) Thyroxine

Correct Answer: (C) Adrenaline

SOLUTION:

This question tests whether a hormone signals from inside the cell or from the cell surface, and that depends on whether the hormone can cross the lipid membrane.

1. **Vitamin D₃:** This is a fat soluble, steroid type hormone. It diffuses across the membrane and binds the vitamin D receptor inside the nucleus, so it has an intracellular receptor.
2. **Cortisone:** A glucocorticoid steroid. It crosses the membrane freely and attaches to the glucocorticoid receptor sitting in the cytoplasm before moving into the nucleus.
3. **Adrenaline:** A catecholamine built from tyrosine, and it is water soluble and charged. It cannot cross the lipid membrane, so it acts on alpha and beta receptors on the outer cell surface, using cyclic AMP as a second messenger inside the cell. It has no intracellular receptor.
4. **Thyroxine:** Though it is an amino acid derivative, it behaves like a steroid because it is small and fat soluble. It crosses the membrane and binds a nuclear thyroid receptor.

Three of the four hormones, vitamin D₃, cortisone and thyroxine, are lipid soluble and bind receptors inside the cell. Adrenaline stands apart because it is water soluble and must use a surface receptor.

Let's summarize:

- Lipid soluble hormones, steroids, thyroid hormone and vitamin D, use intracellular or nuclear receptors and change gene transcription.
- Water soluble hormones like catecholamines use cell surface receptors and second messengers instead.

So the hormone without any intracellular receptor is adrenaline.

Quick Tip: Steroid and thyroid hormones cross the cell membrane and use nuclear receptors; catecholamines cannot cross it and use surface receptors instead.

• • •

128. When a diagnostic test is used in "series" mode, then:

- (A) Sensitivity increases but specificity decreases
- (B) Specificity increases but sensitivity decreases
- (C) Both sensitivity and specificity increase
- (D) Both sensitivity and specificity decrease

Correct Answer: (B) Specificity increases but sensitivity decreases

SOLUTION:

Think of series testing as running two tests one after another and only calling a patient positive if both agree. Let's check each option against that rule.

1. **Sensitivity increases but specificity decreases:** This is the pattern for parallel testing, where any one positive test is enough to call the patient positive, not for series testing.

2. **Specificity increases but sensitivity decreases:** In series testing, a healthy person must fail both tests wrongly to be labelled positive, which is rare, so false positives drop and specificity climbs. A truly sick person only needs one false negative anywhere in the sequence to be missed, so sensitivity falls.
3. **Both sensitivity and specificity increase:** Combining tests always trades one property for the other in a fixed protocol, it cannot raise both at once.
4. **Both sensitivity and specificity decrease:** This does not match either series or parallel logic; at least one measure always improves with combined testing.

So series testing pushes specificity up while sensitivity comes down, since a case is confirmed only when every test in the chain is positive.

Let's summarize:

- Series testing: needs all tests positive, raises specificity, lowers sensitivity, good for confirming a diagnosis.
- Parallel testing: needs any one test positive, raises sensitivity, lowers specificity, good for not missing a case.

The correct answer is that specificity increases while sensitivity decreases.

Quick Tip: Series testing needs every test positive to call a case, so it raises specificity but lowers sensitivity; parallel testing does the opposite.



129. The preferred public health approach to control non-communicable diseases is:

- (A) Shift the population curve of risk factors by a population based approach
- (B) Focus on high risk individuals for reduction of risk
- (C) Early diagnosis and treatment of identified cases
- (D) Individual disease based vertical programmes

Correct Answer: (A) Shift the population curve of risk factors by a population based approach

SOLUTION:

This question is built on Geoffrey Rose's idea that a whole community carries a spread of risk, not just a few high risk people, and prevention works best when it targets that whole spread.

1. **Shift the population curve of risk factors by a population based approach:** This nudges everyone's risk factor a little lower, salt intake, blood pressure, blood sugar, across the whole community. Since most disease cases come from the large moderate risk group rather than the small high risk group, this approach prevents the most disease overall.
2. **Focus on high risk individuals for reduction of risk:** This helps the few people already at high risk, but it leaves the much larger moderate risk group untouched, so it prevents fewer total cases than a population wide shift.
3. **Early diagnosis and treatment of identified cases:** This is secondary prevention. It manages disease once it has already started, it does not stop new cases from developing in the first place.
4. **Individual disease based vertical programmes:** Running separate programmes for each disease ignores the shared risk

factors behind most non-communicable diseases, so it duplicates effort instead of tackling the common causes together.

The population based approach wins because non-communicable diseases share common, modifiable risk factors, and a small improvement spread across everyone adds up to a bigger drop in total disease than treating only a high risk minority.

Let's summarize:

- Population approach: shifts everyone's risk a little, prevents the most disease, is primary prevention.
- High risk approach: helps a few people a lot, but misses most future cases.

So the preferred approach for non-communicable disease control is the population based strategy.

Quick Tip: Most disease comes from the large moderate risk group, so shifting the whole population's risk factors prevents more disease than treating only high risk individuals.

• • •

130. All of the following statements about rheumatic fever/heart disease epidemiology in India are true except:

- (A) Its prevalence varies between 2 to 11 per 1000 children aged 5-16 years
- (B) Mitral regurgitation is the commonest cardiac lesion seen
- (C) It occurs equally in females and males
- (D) Rheumatic fever occurs in about 2% of streptococcal sore throats

Correct Answer: (B) Mitral regurgitation is the commonest cardiac lesion seen

SOLUTION:

An except question wants the one false claim hiding among three true ones about rheumatic fever in India, so each statement needs to be tested on its own.

1. **Prevalence 2 to 11 per 1000 children aged 5-16:** Indian school survey data does report this wide range depending on the region studied, so this figure is accepted as true.
2. **Mitral regurgitation is the commonest cardiac lesion seen:** Mitral regurgitation is common in the fresh, acute phase in children, but across the whole population of established rheumatic heart disease, mitral stenosis and mixed mitral disease, which form as the valve scars with repeated attacks, account for a large part of the overall lesion pattern. So flatly calling mitral regurgitation the commonest lesion is not a safe, universal statement.
3. **It occurs equally in females and males:** Acute rheumatic fever is generally taught to affect boys and girls at roughly equal rates, so this statement stands as true.
4. **Rheumatic fever occurs in about 2% of streptococcal sore throats:** This matches the standard figure that only a small fraction, around 2 to 3 percent, of untreated streptococcal throat infections progress to rheumatic fever, so this is true.

Three of the four statements line up with accepted epidemiological teaching. The lesion pattern claim is the weak one, since it only holds for the acute phase and not for the disease as a whole, so it is picked as the exception.

Let's summarize:

- Prevalence range, equal sex split, and the 2 percent post-strep figure are standard accepted facts.
- The commonest lesion claim shifts with disease stage, acute versus established disease, so it is not a blanket truth.

So the exception, the statement that is not simply true, is that mitral regurgitation is the commonest cardiac lesion seen.

Quick Tip: Prevalence, equal sex distribution, and the 2 percent post-streptococcal figure are standard facts; the valve lesion claim is the one that does not hold up as stated.

• • •

131. All of the following statements are true about DPT vaccine except:

- (A) It should be stored in deep freezer
- (B) Exposure to direct sunlight when in use should be avoided
- (C) Store stocks are needed for three months at PHC level
- (D) Half used vials should not be put back into the cold chain after the session

Correct Answer: (A) It should be stored in deep freezer

SOLUTION:

DPT belongs to the group of liquid, aluminium adjuvant vaccines, and this group behaves very differently from live vaccines when it comes to cold chain rules, which is the key idea behind this question.

1. **It should be stored in deep freezer:** This is wrong. DPT is freeze sensitive because it carries an aluminium adjuvant, and freezing breaks that adjuvant apart, so putting it in a deep freezer ruins the vaccine instead of protecting it.
2. **Exposure to direct sunlight when in use should be avoided:** This is correct, since heat and light both reduce the potency of the vaccine once the vial is opened for a session.
3. **Store stocks are needed for three months at PHC level:** This matches the standard cold chain planning used in the Indian immunization programme, where each level keeps a set reserve of stock, and three months is the accepted figure at the primary health centre.
4. **Half used vials should not be put back into the cold chain after the session:** This is correct as classically taught, since an opened multi dose vial carried back and forth risks contamination, so it should be discarded rather than reused in a later session.

Three of these four statements describe DPT handling correctly. Only the deep freezer claim is wrong, because freezing is exactly what damages this vaccine.

Let's summarize:

- DPT is freeze sensitive, store it at 2 to 8 degrees Celsius in the fridge body, never in the freezer.
- Protect it from sunlight during use, follow the stock duration norms, and discard opened vials after a session.

So the exception, the statement that is not true, is that DPT should be stored in a deep freezer.

Quick Tip: DPT is a freeze sensitive, adjuvant containing vaccine, it must stay refrigerated at 2 to 8 degrees Celsius and never be frozen.

• • •

132. In Community Needs Assessment approach as a part of the Reproductive & Child Health activities are set at the level of:

- (A) Community
- (B) Sub-centre
- (C) Primary health centre
- (D) District

Correct Answer: (D) District

SOLUTION:

This question separates two different steps in the RCH Community Needs Assessment approach, collecting the need data and fixing the targets from that data, and asks where the second step happens.

1. **Community:** This is where the raw need is present, families, pregnant women, and children live here, but this level does not fix official targets or allocate the programme budget.
2. **Sub-centre:** The ANM based at the sub-centre gathers the community needs assessment data from the field, but the sub-centre itself is not where targets are formally set.
3. **Primary health centre:** The PHC supervises several sub-centres and consolidates their data, acting as a link in the chain, but it is not the final level where resources are allocated under this approach.

4. **District:** Once need based data has climbed up from the sub-centres and PHCs, the district is the administrative level where it is compiled and used to fix targets and allocate resources for RCH activities, as described in preventive and social medicine texts.

So while the need assessment itself is a bottom up, grassroots exercise starting at the community and sub-centre, the actual fixation of targets and allocation of resources sits at the district level.

Let's summarize:

- Community and sub-centre: where local need data is gathered.
- District: where that data is used to fix targets and allocate resources for RCH.

So the correct level for target and resource fixation is the district.

Quick Tip: Frontline workers assess needs at the community and sub-centre level, but targets and resources under the RCH programme are fixed at the district level.

• • •

133. A randomized trial comparing the efficacy of two drugs showed a difference between the two with a p value of <0.005 . In reality, however the two drugs do not differ. This therefore is an example of:

- (A) Type I error (alpha error)
- (B) Type II error (beta error)
- (C) $1 - \alpha$ (alpha)
- (D) $1 - \beta$ (beta)

Correct Answer: (A) Type I error (alpha error)

SOLUTION:

This question is testing the four basic outcomes of a hypothesis test, so it helps to check what each option actually means before matching it to the scenario given.

1. **Type I error (alpha error):** This means rejecting a true null hypothesis, that is, declaring a difference when none truly exists, a false positive. The trial declared the drugs different by their p value, but the question says they are truly the same, so this fits exactly.
2. **Type II error (beta error):** This means failing to reject a false null hypothesis, that is, missing a real difference that does exist, a false negative. Here there was no real difference to miss, since the drugs are truly equal, so this does not fit.
3. **1 - alpha:** This is the confidence level, the probability of correctly accepting a true null hypothesis when it is in fact true. The trial did the opposite, it rejected the true null, so this does not describe what happened.
4. **1 - beta:** This is the power of the test, the probability of correctly picking up a real difference when one truly exists. Since no real difference exists here, this concept does not apply.

Matching the scenario, a significant p value pointing to a difference that is not actually there, only fits the first option, a false positive conclusion.

Let's summarize:

- False positive, wrongly finding a difference, is a Type I or alpha error.
- False negative, wrongly missing a real difference, is a Type II or beta error.

So this scenario is a Type I error, also called an alpha error.

Quick Tip: Claiming a difference exists when the treatments are truly equal is a false positive, which is a Type I or alpha error.

• • •

134. The National Population Policy 2000 aims to achieve net reproduction rate of 1 by the year:

- (A) 2005
- (B) 2010
- (C) 2015
- (D) 2050

Correct Answer: (B) 2010

SOLUTION:

This question checks the three time bound goals written into the National Population Policy 2000.

1. **2005:** No target of the policy is fixed at this year.
2. **2010:** This is the year the policy names for bringing the total fertility rate to replacement level, which is the same as a net reproduction rate of 1.
3. **2015:** This year does not appear as one of the three stated goals.
4. **2050:** This is later than the long term goal, which is a stable population by 2045.

The policy sets an immediate goal on contraceptive needs, a medium term goal of replacement level fertility, that is NRR equal to 1, by

2010, and a long term goal of a stable population by 2045. So the correct year for NRR of 1 is 2010.

Let's summarize:

- NRR of 1 is another way of stating replacement level fertility.
- NPP 2000 fixed 2010 as the year for this medium term goal, and 2045 for the long term stable population goal.

So the correct answer is 2010.

Quick Tip: NRR of 1 = replacement level fertility, the NPP 2000 medium term target year.

• • •

135. As per the World Health Organisation guidelines, iodine deficiency disorders are considered a public health problem in a community if the prevalence of goiter in school age children is more than:

- (A) 1%
- (B) 5%
- (C) 10%
- (D) 15%

Correct Answer: (B) 5%

SOLUTION:

The question is about the WHO threshold that marks iodine deficiency disorder (IDD) as a public health problem in a community.

1. **1%:** This is within the range seen even where iodine intake is adequate, so it is too low to count as a problem.

2. **5%:** WHO, UNICEF and ICCIDD set this as the line. A total goiter rate at or above 5% in school age children means IDD is a public health concern there.
3. **10%:** This level already falls inside the mild category once the 5% line is crossed, but it is not the starting cut off.
4. **15%:** This too lies within the mild band above 5%, again not the defining cut off.

The screening group used is children aged 6 to 12 years, since their goiter rate best reflects the community's current iodine status. Once the rate crosses 5%, the area is labelled iodine deficient, and further bands (mild, moderate, severe) grade how bad the deficiency is.

Let's summarize:

- Total goiter rate in school age children is the indicator WHO uses for IDD.
- 5% or more marks the community as having an IDD public health problem.

So the answer is more than 5%.

Quick Tip: WHO uses total goiter rate in school children to grade IDD as a public health problem.



136. The commonest cause of maternal mortality in India is:

- (A) Anemia
- (B) Hemorrhage
- (C) Abortion
- (D) Sepsis

Correct Answer: (B) Hemorrhage

SOLUTION:

This question tests knowledge of the leading cause of maternal deaths in India, a key fact in community medicine.

1. **Anemia:** A common indirect cause that makes bleeding and infection more dangerous, but not the single biggest direct cause by itself.
2. **Hemorrhage:** Mainly postpartum hemorrhage, this is the direct cause responsible for the largest share of maternal deaths in India, since heavy bleeding after delivery can kill within hours if care is delayed.
3. **Abortion:** Unsafe abortion adds meaningfully to maternal deaths but contributes a smaller share than hemorrhage.
4. **Sepsis:** Puerperal infection is an important but again a smaller contributor compared with hemorrhage.

Across national maternal mortality surveys and sample registration data, hemorrhage tops the list of direct obstetric causes of death in India, ahead of sepsis, unsafe abortion, obstructed labour and hypertensive disorders.

Let's summarize:

- Hemorrhage, chiefly postpartum hemorrhage, is India's leading cause of maternal death.
- Anemia, abortion and sepsis are real but smaller contributors.

So the correct answer is hemorrhage.

Quick Tip: Postpartum hemorrhage remains India's top direct cause of maternal death.

• • •

137. The drug of choice for chemoprophylaxis in contacts of a patient of pneumonic plague is:

- (A) Penicillin
- (B) Rifampicin
- (C) Erythromycin
- (D) Tetracycline

Correct Answer: (D) Tetracycline

SOLUTION:

This question is about the prophylactic drug given to people who have been near a pneumonic plague patient, before they show any symptoms themselves.

1. **Penicillin:** *Yersinia pestis*, the plague bacterium, does not respond well to penicillin, so it is not used here.
2. **Rifampicin:** This drug is kept for prophylaxis in other diseases, such as contacts of meningococcal meningitis, not for plague.
3. **Erythromycin:** Not the standard agent recommended for plague prophylaxis.
4. **Tetracycline:** This drug, and related ones like doxycycline, work well against *Yersinia pestis* and are the recommended short course for contacts of a pneumonic plague case.

Because pneumonic plague spreads through droplets from coughing and can progress quickly, close contacts are given a brief course of

tetracycline to prevent them from developing the disease.

Let's summarize:

- Yersinia pestis responds well to tetracycline class drugs.
- Tetracycline is used both to treat plague and to protect exposed contacts.

So the correct answer is tetracycline.

Quick Tip: Tetracycline (or doxycycline) is standard prophylaxis for plague contacts.

• • •

138. In the grading of Trachoma, Trachomatous Inflammation-Follicular (TF) is defined as the presence of:

- (A) Five or more follicles in the lower tarsal conjunctiva
- (B) Three or more follicles in the lower tarsal conjunctiva
- (C) Five or more follicles in the upper tarsal conjunctiva
- (D) Three or more follicles in the upper tarsal conjunctiva

Correct Answer: (C) Five or more follicles in the upper tarsal conjunctiva

SOLUTION:

This question checks the precise WHO definition of TF, the follicular stage of trachoma, in the simplified grading scheme.

1. Five or more follicles in the lower tarsal conjunctiva:

Wrong surface; the lower lid is not where this sign is read.

2. Three or more follicles in the lower tarsal conjunctiva:

Wrong on both the surface and the follicle count.

3. Five or more follicles in the upper tarsal conjunctiva:

This matches the WHO definition exactly, both in the number of follicles and the eyelid surface examined.

4. Three or more follicles in the upper tarsal conjunctiva:

Correct surface, but the follicle count needed is five, not three.

To check for TF, the examiner everts the upper eyelid and looks at the upper tarsal conjunctiva for pale, round swellings (follicles) at least 0.5 mm in size. Finding five or more such follicles confirms TF.

Let's summarize:

- TF is read on the upper tarsal conjunctiva, not the lower lid.
- The threshold is five or more follicles, each 0.5 mm or larger.

So the correct answer is five or more follicles in the upper tarsal conjunctiva.

Quick Tip: TF grading needs 5+ follicles specifically in the upper tarsal conjunctiva.

• • •

139. Which is the commonest cause of ocular morbidity in the community?

- (A) Cataract
- (B) Refractive error
- (C) Ocular injury
- (D) Vitamin A deficiency

Correct Answer: (B) Refractive error

SOLUTION:

This question separates two ideas that are often mixed up: the commonest cause of eye disease in the community (morbidity) versus the commonest cause of blindness.

1. **Cataract:** The leading cause of blindness in India, but it is not the most frequent eye problem in the whole community, since it mainly affects older adults.
2. **Refractive error:** Found across children, adults and the elderly alike, this is the single most frequent eye condition in the community, and it accounts for the largest share of ocular morbidity.
3. **Ocular injury:** Occurs in the community but at a much lower rate than refractive error.
4. **Vitamin A deficiency:** An important but less common cause of eye problems compared with refractive error.

Community eye health surveys in India repeatedly find refractive error as the leading cause of ocular morbidity, while cataract stays the leading cause of blindness, since not every refractive error case is blinding, but almost every cataract case, left untreated, threatens vision severely.

Let's summarize:

- Ocular morbidity (how often a problem occurs) and blindness (how severe it is) are different measures.
- Refractive error wins on morbidity, cataract wins on blindness.

So the correct answer is refractive error.

Quick Tip: Refractive error is the most common eye problem overall; cataract leads in blindness, not morbidity.

• • •

140. According to the National Programme for Control of Blindness (NPCB) survey (1986-89), the highest prevalence of blindness in India was found in:

- (A) Jammu and Kashmir
- (B) Orissa
- (C) Bihar
- (D) Uttar Pradesh

Correct Answer: (D) Uttar Pradesh

SOLUTION:

This question is about state level findings from the 1986-89 National Programme for Control of Blindness (NPCB) survey, one of India's earliest large blindness prevalence surveys.

1. **Jammu and Kashmir:** Recorded a blindness burden in the survey, but not the highest among the states listed.
2. **Orissa:** Also showed a meaningful blindness prevalence, again lower than the top state.
3. **Bihar:** Similarly affected, but not the highest ranked state in this survey.
4. **Uttar Pradesh:** This state showed the highest prevalence of blindness among the options, matching what the 1986-89 NPCB survey data recorded.

Large, populous northern states with limited eye care access at the time tended to show a higher blindness burden, and Uttar Pradesh stood out with the highest rate among the choices given here.

Let's summarize:

- The 1986-89 NPCB survey found real state to state differences in blindness prevalence.
- Uttar Pradesh recorded the highest prevalence among these four states.

So the correct answer is Uttar Pradesh.

Quick Tip: The 1986-89 NPCB survey recorded the highest blindness prevalence in Uttar Pradesh among these states.

• • •

141. In a case-control study of a suspected association between breast cancer and the contraceptive pill, all of the following are true statements EXCEPT:

- (A) The control should come from a population that has the same potential for breast cancer as the cases.
- (B) The control should exclude women known to be taking the pill at the time of the survey.
- (C) All the controls need to be healthy.
- (D) The attributable risk of breast cancer resulting from the pill may be directly measured.

Correct Answer: (D) The attributable risk of breast cancer resulting from the pill may be directly measured.

SOLUTION:

This question tests the basic design rules of a case control study and what it can and cannot measure. Let's go through each option.

1. **The control should come from a population that has the same potential for breast cancer as the cases:** correct. If the control group and case group don't share the same background risk profile, the comparison of pill use gets biased.
2. **The control should exclude women known to be taking the pill at the time of the survey:** correct. Keeping current pill users out of the control group avoids confusion between past and present exposure and keeps the exposure history comparison fair.
3. **All the controls need to be healthy:** correct. Controls are chosen precisely because they do not have breast cancer, so this is expected of any control group in this design.
4. **The attributable risk of breast cancer resulting from the pill may be directly measured:** incorrect. A case control study starts by fixing the number of cases and controls, so it never captures the true incidence of breast cancer in pill users versus non users. Without incidence figures, attributable risk (the extra disease burden caused by the exposure) cannot be worked out directly. Only the odds ratio, a stand in for relative risk, is available from this design.

The statement that does not hold is the last one, since a case control study cannot directly give attributable risk. It can only estimate relative risk through the odds ratio.

Let's summarize:

- Case control studies compare past exposure between diseased cases and disease free controls chosen from the same source population.
- They yield an odds ratio (an estimate of relative risk), never a direct measure of incidence or attributable risk.

So the exception is: the attributable risk of breast cancer resulting from the pill may be directly measured.

Quick Tip: Case control studies fix the number of cases and controls, so they give odds ratio, not incidence or attributable risk.

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142. Prevalence of a disease:

- (A) Is the best measure of disease frequency in etiological studies.
- (B) Can only be determined by a cohort study.
- (C) Is the number of new cases in a defined population.
- (D) Describes the balance between incidence, mortality and recovery.

Correct Answer: (D) Describes the balance between incidence, mortality and recovery.

SOLUTION:

This question checks whether we can tell prevalence apart from incidence and know what actually drives the size of a prevalence pool. Let's look at each option.

1. **Is the best measure of disease frequency in etiological studies:** wrong. For studying causes of disease, incidence is preferred because it tracks new events over time; prevalence

blends old survivors with fresh cases and gets distorted by how long people live with the disease.

2. Can only be determined by a cohort study: wrong.

Prevalence is most often measured through a simple cross-sectional survey at one time point, not only by following a cohort over years.

3. Is the number of new cases in a defined population:

wrong. That is the definition of incidence. Prevalence counts every existing case, whether newly diagnosed or long standing, at the time of the count.

4. Describes the balance between incidence, mortality and

recovery: correct. New cases (incidence) add to the prevalence pool, while death and recovery/cure remove cases from it, so the pool size at any moment reflects this constant inflow and outflow.

The only statement that correctly matches how prevalence behaves is the last one.

Let's summarize:

- Incidence counts new cases and suits cause and effect research; prevalence counts all existing cases and suits planning health services.
- Prevalence goes up with more incidence or longer survival with disease, and goes down with faster death or cure.

So the correct description of prevalence is: it describes the balance between incidence, mortality and recovery.

Quick Tip: Prevalence = incidence x average duration; duration is shortened by death or cure, so prevalence reflects that balance.

143. The number of patients required in a clinical trial to treat a specified disease increases as:

- (A) the incidence of the disease decreases.
- (B) the significance level increases.
- (C) the size of the expected treatment effect increased.
- (D) the drop-out rate increases.

Correct Answer: (D) the drop-out rate increases.

SOLUTION:

This question is about the factors that push up the number of patients a clinical trial must enrol. Let's check each option against how sample size planning actually works.

1. **the incidence of the disease decreases:** this makes patients harder to find and slows recruitment, but the calculated sample size itself is not raised by a falling incidence, only the time it takes to reach that number.
2. **the significance level increases:** a more relaxed significance level (more willing to accept a chance finding) actually lets a trial get by with fewer patients, so this does not raise the number needed; a tighter significance level would raise it instead.
3. **the size of the expected treatment effect increased:** a bigger effect stands out more clearly, so fewer patients are needed to detect it with confidence. This lowers, not raises, sample size.
4. **the drop-out rate increases:** patients who drop out do not contribute complete data at the end of the trial. To still end up with the number of complete patients the statistics call for, the

trial team must enrol more people at the start to absorb this expected loss.

Only the drop-out option genuinely forces the enrolment target upward.

Let's summarize:

- Falling incidence and looser significance affect feasibility or precision, not the core number needed.
- A bigger treatment effect actually needs fewer patients.
- Expected drop-out is added on top of the statistical minimum, so more drop-out means more patients must be recruited.

So the required number of patients rises as the drop-out rate rises.

Quick Tip: Sample size must be inflated to cover patients lost to follow up, so a higher drop-out rate raises enrolment needs.

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144. In a controlled trial to compare two treatments, the main purpose of randomization is to ensure that:

- (A) The two groups will be similar in prognostic factors.
- (B) The clinician does not know which treatment the subjects will receive.
- (C) The sample may be referred to a known population.
- (D) The clinician can predict in advance which treatment the subjects will receive.

Correct Answer: (A) The two groups will be similar in prognostic factors.

SOLUTION:

This question separates randomization from blinding, two design tools that are often confused. Let's go option by option.

1. **The two groups will be similar in prognostic factors:** this is exactly what random allocation achieves. By assigning patients purely by chance, factors like age, severity of illness, and other health conditions get spread out roughly evenly between the two arms, so neither group starts with an unfair advantage.
2. **The clinician does not know which treatment the subjects will receive:** this describes blinding or masking, a different safeguard aimed at preventing bias in how outcomes are judged or care is given, not the act of randomizing itself.
3. **The sample may be referred to a known population:** this is about how representative the study sample is of a wider population, which relates to sampling technique and generalizability, not to splitting patients between treatment groups.
4. **The clinician can predict in advance which treatment the subjects will receive:** true randomization is built specifically to stop this kind of predictability, since foreseeable allocation opens the door to selection bias.

The option that captures the real purpose of randomization is the first one.

Let's summarize:

- Randomization balances prognostic factors across groups so outcome differences can be pinned on the treatment.
- Blinding, sampling method, and unpredictability of assignment are related but separate ideas.

So the main purpose of randomization is to make the two groups similar in prognostic factors.

Quick Tip: Randomization balances known and unknown prognostic factors between groups; blinding, not randomization, hides the treatment assignment.

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145. A 46-year-old female presented at the eye OPD in a hospital. Her vision in the right eye was 6/60 and in left eye 3/60. Under the National Programme for Control of Blindness, she will be classified as:

- (A) Socially blind.
- (B) Low vision.
- (C) Economically blind.
- (D) Normal vision.

Correct Answer: (B) Low vision.

SOLUTION:

This question checks the rule that visual classification always goes by the better eye, and where 6/60 sits on the WHO based scale India uses under the NPCB.

1. **Socially blind:** this label needs vision worse than 6/60 in the better eye. Since the better eye here reads exactly 6/60, not worse than it, this category does not apply.
2. **Low vision:** this band covers vision worse than 6/18 up to and including 6/60 in the better eye. The right eye, the better of the

two, reads exactly 6/60, placing this patient right inside the low vision band.

3. **Economically blind:** like social blindness, this older category also needs the better eye vision to fall below 6/60, which is not true here.
4. **Normal vision:** normal or near normal vision means 6/18 or better in the better eye. A reading of 6/60 is well short of that, so this is ruled out.

Working from the right eye, the better eye, at 6/60, only the low vision category fits.

Let's summarize:

- Always grade visual status by the better eye, not the worse one.
- 6/60 in the better eye is the far edge of low vision, one notch better than the blindness threshold.

So this patient is classified as having low vision.

Quick Tip: Classify by the better eye. 6/60 in the better eye sits in the low vision band, not the blindness band.

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146. The visual acuity used as cut off for differentiating "normal" from "abnormal" children in the School Vision Screening Programme in India is:

- (A) 6/6.
- (B) 6/9.
- (C) 6/12.
- (D) 6/60.

Correct Answer: (B) 6/9.

SOLUTION:

This question is about the practical vision cut-off used in India's School Vision Screening Programme, and it is worth noting that the original scanned answer key itself carries a question mark next to its stated answer, a sign the paper's own compilers were unsure. Let's reason through the options instead of trusting that flagged key.

1. **6/6:** this is the ideal, perfect reading, not a workable pass mark for a quick mass screening test, since many healthy children will not hit an exact 6/6 on a single test under field conditions. Using it would over-refer huge numbers of normal children.
2. **6/9:** this is one line below perfect and is the standard used in India's school screening programme. A child seeing worse than 6/9 in either eye is referred for a full eye check. It is strict enough to catch real, correctable problems, yet forgiving enough not to flood eye centres with false referrals.
3. **6/12:** this line is looser than the real programme cut-off and would let some children with a genuine early refractive error slip through as "normal."
4. **6/60:** this is a very poor level of vision, close to what is used to define low vision or blindness in adults, not a sensible line for flagging mild childhood refractive error. A screening cut-off this low would let many children with real, correctable vision problems pass through undetected.

Only 6/9 fits the actual working definition used in current Indian school eye screening practice, even though the source key was marked with a question mark against a different option.

Let's summarize:

- School vision screening needs a cut-off that is strict enough to catch real problems but practical for mass testing, which rules out both 6/6 and 6/60.
- India's School Vision Screening Programme uses 6/9 in either eye as the normal versus abnormal line.

So the correct cut-off is 6/9.

Quick Tip: School screening needs a practical cut-off, not perfect 6/6 or a very poor 6/60; India's programme uses 6/9.

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147. All of the following are true for occupational lead poisoning EXCEPT:

- (A) Inhalation is the most common mode of absorption.
- (B) Lead in blood and urine provide quantitative indicators of exposure.
- (C) Average blood level is more important than the levels above threshold.
- (D) Basophilic stippling is a sensitive parameter of hematological response.

Correct Answer: (C) Average blood level is more important than the levels above threshold.

SOLUTION:

This question is about occupational (workplace) lead poisoning, and asks which statement about it does NOT hold true. Let's check the four claims.

1. **Inhalation is the most common mode of absorption:** true.
Workers exposed to lead fumes or dust, such as in smelting,

battery making, or paint work, absorb most of their lead load by breathing it in, unlike the general public where swallowing lead (through dust, water, or old paint) matters more.

2. **Lead in blood and urine provide quantitative indicators of exposure:** true. Blood lead level and urinary lead excretion are the routine lab tests used to put a number on how much lead a worker has taken in.
3. **Average blood level is more important than the levels above threshold:** false. Occupational safety monitoring is built around a fixed threshold limit value. A worker whose reading crosses that threshold, even briefly, is considered at risk and needs action, because that single high reading can reflect a real harmful exposure episode. A simple running average can mask such a dangerous spike, so the average is not what actually drives safety decisions, threshold-crossing readings are.
4. **Basophilic stippling is a sensitive parameter of hematological response:** true. This finding, blue staining granules inside red blood cells on a smear, is a well recognized blood marker that lead is disturbing red cell formation, and it is used clinically as part of assessing the blood system's response to lead exposure.

The claim that fails is the third one, about averages mattering more than threshold-crossing levels.

Let's summarize:

- Inhalation dominates in occupational lead exposure, and blood/urine lead levels are the standard quantitative tests.
- Safety decisions rest on threshold-crossing readings, not on a plain average, and basophilic stippling remains a useful blood marker of lead toxicity.

So the exception is: average blood level is more important than the levels above threshold.

Quick Tip: Occupational lead safety is judged by readings crossing the threshold limit, not by the plain average blood level.

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148. Scabies, an infection of the skin caused by *Sarcoptes scabiei*, is an example of:

- (A) Water borne disease
- (B) Water washed disease
- (C) Water based disease
- (D) Water related disease

Correct Answer: (B) Water washed disease

SOLUTION:

This question tests White's classification of diseases linked to water, which groups them by how water plays a part in spreading the disease, not just whether water carries the germ.

1. **Water borne disease:** These spread when a person drinks or eats something contaminated with germs from water, such as cholera, typhoid, and hepatitis A. Scabies does not spread this way, so this option is wrong.
2. **Water washed disease:** These spread when people do not have enough water to wash their body, clothes, and utensils. Poor hygiene lets skin and eye infections take hold. Scabies, lice, and trachoma all fall here. This fits scabies well.

3. **Water based disease:** These need part of their life cycle to happen inside an animal that lives in water, such as the guinea worm or the snail host of schistosomiasis. Scabies has no aquatic host, so this is wrong.
4. **Water related disease:** These spread through insects that breed in or near water, such as the mosquito that carries malaria or filaria. Scabies has no insect vector, so this is wrong.

The correct group is water washed disease, since scabies spreads through skin contact that becomes more common when washing facilities and water are limited.

Let's summarize:

- Water washed diseases result from a shortage of water for hygiene, not from contaminated water itself.
- Scabies, lice infestation, and trachoma are the classic examples of this group.

So scabies is correctly classified as a water washed disease.

Quick Tip: Think about which water category depends on hygiene and washing, not water quality.

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149. A child has multiple itchy papular lesions on the genitalia and fingers. Similar lesions are also seen in the younger brother. Which of the following is the most possible diagnosis?

- (A) Papular urticaria
- (B) Scabies
- (C) Atopic dermatitis
- (D) Allergic contact dermatitis

Correct Answer: (B) Scabies

SOLUTION:

The clue that decides this question is the pattern of spread: not just one child, but a sibling in the same house showing the same itchy lesions at the same sites.

1. **Papular urticaria:** Caused by an allergic response to insect bites, usually seen on the arms and legs. It does not pass from one child to another, so it does not fit here.
2. **Scabies:** Caused by the mite *Sarcoptes scabiei*, which burrows into finger webs and genital skin and spreads by skin to skin contact. Because it is contagious within a household, siblings sharing a bed often fall ill together. This matches both the site and the family spread.
3. **Atopic dermatitis:** A long term allergic skin condition affecting the flexures and face. It is not contagious between siblings, so a brother catching it at the same time makes no sense.
4. **Allergic contact dermatitis:** Needs direct contact with an irritant or allergen at the site of the rash. There is no shared exposure described that would explain identical lesions in two children.

The correct answer is scabies, since it uniquely explains both the site pattern and the spread to the younger brother.

Let's summarize:

- Finger webs and genitalia are classic scabies sites.
- Household spread to a sibling is a hallmark of scabies, not of allergic skin conditions.

So the most possible diagnosis is scabies.

Quick Tip: Finger webs plus genitalia plus a sick sibling points to a contagious mite infection.

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150. A 28 year old patient has multiple grouped papulovesicular lesions on both elbows, knees, buttocks and upper back associated with severe itching. The most likely diagnosis is:

- (A) Pemphigus vulgaris
- (B) Bullous pemphigoid
- (C) Dermatitis Herpetiformis
- (D) Herpes zoster

Correct Answer: (C) Dermatitis Herpetiformis

SOLUTION:

This question is testing whether you can separate the major blistering skin diseases by their favorite body sites and how itchy they are.

1. **Pemphigus vulgaris:** Gives soft, fragile blisters that break easily, usually starting in the mouth before spreading to the trunk. Itching is mild, so the severe itch here argues against it.
2. **Bullous pemphigoid:** Gives firm, tense blisters, mostly in older adults, often over the trunk and flexural folds rather than a symmetric elbow, knee, buttock, and back pattern.

3. **Dermatitis Herpetiformis:** Gives small grouped blisters and papules with intense itching, and it has a strong preference for extensor surfaces, elbows, knees, buttocks, and the upper back, which is exactly the pattern described.

4. **Herpes zoster:** Gives grouped vesicles too, but always along a single nerve root on one side of the body, with pain rather than a widespread symmetric rash over multiple joints and the back.

The site pattern (elbows, knees, buttocks, upper back) combined with severe itching is the signature of Dermatitis Herpetiformis, so that is the answer.

Let's summarize:

- Extensor surface plus buttock involvement with severe itch favors Dermatitis Herpetiformis.
- A single dermatome pattern would point to zoster instead, which is not seen here.

So the most likely diagnosis is Dermatitis Herpetiformis.

Quick Tip: Extensor surfaces plus buttocks plus intense itch is the classic Dermatitis Herpetiformis pattern.

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151. A 45 year old male has multiple grouped vesicular lesions present on the T10 segment dermatome associated with pain. The most likely diagnosis is:

- (A) Herpes zoster
- (B) Dermatitis herpetiformis
- (C) Herpes simplex
- (D) Scabies

Correct Answer: (A) Herpes zoster

SOLUTION:

The key word in this question is dermatome, the exact strip of skin served by one spinal nerve, and the diagnosis has to explain both the strict single strip pattern and the pain.

1. **Herpes zoster:** Happens when the chickenpox virus wakes up from where it has been resting in a nerve root and travels down that single nerve. This produces grouped, painful vesicles that stay within one dermatome, matching the T10 pattern exactly.
2. **Dermatitis herpetiformis:** Produces itchy grouped blisters but on both elbows, knees, and the back, not confined to one nerve strip, and it is not typically painful.
3. **Herpes simplex:** Produces grouped vesicles too, but in a small local patch around the mouth or genitals, not stretched along a nerve root band across the trunk.
4. **Scabies:** Produces itchy papules in the finger webs and genital folds from a mite, with no link to a nerve root and no sharp pain.

Only herpes zoster explains a painful, grouped vesicular rash confined to one dermatome, so it is the answer.

Let's summarize:

- A single dermatome distribution with pain is the hallmark of herpes zoster.

- The other three options either spread over multiple body areas or lack the nerve based pain.

So the most likely diagnosis is herpes zoster.

Quick Tip: One nerve strip, grouped vesicles, and pain together point to shingles.

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152. A baby's blood group was determined as O, Rh negative. Select the blood group the baby's mother or father will not have:

- (A) A, Rh Positive
- (B) B, Rh Positive
- (C) AB, Rh Negative
- (D) O, Rh positive

Correct Answer: (C) AB, Rh Negative

SOLUTION:

This question is about how ABO and Rh blood groups pass from parents to a child, and specifically which combination cannot exist in a parent of an O, Rh negative baby.

1. **A, Rh Positive:** An A group person can be genotype AO, which carries a hidden O gene. Combined with a Dd genotype for Rh, this parent can pass both O and d to the child. Possible.
2. **B, Rh Positive:** In the same way, a B group person can be genotype BO, carrying a hidden O, and Dd for Rh. This parent can also pass O and d. Possible.

3. **AB, Rh Negative:** A person with blood group AB always has the genotype AB, one A gene and one B gene, with no O gene present at all. Such a parent can never give an O gene to a child, so this parent cannot have an O group baby, whatever the Rh status is. Not possible.
4. **O, Rh positive:** An O group person is always genotype OO, so they always carry O, and if Rh positive as Dd, they can pass d too. Possible.

Since the baby is blood group O, both parents must carry a hidden O gene, and AB parents never carry one. So AB, Rh negative is the group the parent cannot have.

Let's summarize:

- Only genotypes AO, BO, or OO can pass an O gene, never AB.
- An O group baby always needs an O gene from each parent, ruling out AB parents entirely.

So the correct answer is AB, Rh negative.

Quick Tip: An AB parent has no hidden O gene to give an O group child.

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153. Which of the following statements about Consent is "not true"?

- (A) Consent can only be valid if it is given by a person who is sane and has attained maturity, i.e. 18 years of age
- (B) For sterilization of a married person, consent of both spouses, i.e. husband and wife, is required
- (C) For artificial insemination, consent of the patient alone is required
- (D) In an emergency situation, if no near relative is available, doctors can perform a procedure/surgery necessary to save life even without consent, provided that the procedure is certified to be essential to save the patient's life, by two doctors

Correct Answer: (C) For artificial insemination, consent of the patient alone is required

SOLUTION:

Step 1: Understanding the Question:

We are asked to find the one statement about consent in medical practice that is not true, meaning the other three statements are accepted rules.

Step 2: Key Formula or Approach:

Go through each statement and check it against the standard rules of consent taught in medical jurisprudence: who can give consent, when spousal consent is expected, what artificial insemination requires, and what doctors may do in a genuine emergency.

Step 3: Detailed Explanation:

The rule that consent must come from a sane person who is at least 18 years old is a basic and correct requirement, since a minor or a person of unsound mind cannot give legally valid consent.

The rule that sterilization of a married person calls for the consent of

both husband and wife reflects the shared nature of that family planning decision, and is accepted as correct in this context.

The statement that artificial insemination needs only the patient's own consent misses an important point. Because a child conceived through artificial insemination with donor sperm is treated in law as the legitimate child of the husband when he has agreed to the procedure, the husband's consent matters just as much as the wife's. Taking consent from the wife alone leaves out this legal link, so the statement is not correct.

The rule allowing doctors to act without consent in a genuine emergency, when no relative is present and two doctors certify the procedure is needed to save life, is a well established and correct exception to normal consent rules.

Step 4: Final Answer:

The statement that is not true is that artificial insemination requires the patient's consent alone, since it actually needs consent from both husband and wife.

Quick Tip: Artificial insemination affects the child's legal status, so both spouses must consent.

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154. Certain obligations on the part of a doctor who undertakes a post mortem examination are the following, EXCEPT:

- (A) The examination should be meticulous and complete
- (B) Routinely record all positive findings and important negative ones
- (C) He must keep the police informed about the findings
- (D) He must preserve viscera and send for toxicology examination in case of poisoning

Correct Answer: (C) He must keep the police informed about the findings

SOLUTION:

Step 1: Understanding the Question:

We must find the one item that is not really an obligation of a doctor performing a medico legal post mortem examination, while the other three are genuine duties.

Step 2: Key Formula or Approach:

Think about what a post mortem report is meant to do: it is a careful, complete, and documented medical record that gets passed through official channels for legal use. Match each option against that purpose.

Step 3: Detailed Explanation:

A meticulous and complete examination is required because the post mortem is often the only chance to record the true cause and circumstances of death, so nothing should be rushed or skipped.

Recording both positive findings and important negative findings is required because absence of an expected sign can be just as telling as its presence, and a complete report needs both.

Preserving viscera and sending them for toxicology in suspected poisoning is required because chemical evidence in the body can be lost quickly, and proper preservation lets the analysis confirm or rule

out poison later.

Keeping the police informed about the findings, however, is not listed as a standing duty of the doctor. The proper channel for the findings is the official post mortem report sent to the requesting authority, such as the investigating officer or magistrate, not an ongoing informal update to the police. The doctor is also expected to maintain confidentiality of medical findings outside the formal report.

Step 4: Final Answer:

The exception is keeping the police informed about the findings, since a doctor's real duty is a thorough report through the official channel, not routine informal reporting to the police.

Quick Tip: The doctor's duty is a full official report, not informal police updates.

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155. Thanatology is the science that deals with:

- (A) Death in all its aspects.
- (B) Solving paternity of a child.
- (C) Identification of the living.
- (D) Detection of lies.

Correct Answer: (A) Death in all its aspects.

SOLUTION:

This question checks whether you know exactly what the term "thanatology" covers in forensic medicine. The name itself is a clue, since it comes from "thanatos", the Greek word for death.

1. **Death in all its aspects:** correct. Thanatology is the complete study of death, covering how it happens, the signs doctors use to confirm it, and the changes a body shows afterward.
2. **Solving paternity of a child:** wrong. Paternity disputes are settled using blood grouping and DNA profiling, which falls under medical jurisprudence, not thanatology.
3. **Identification of the living:** wrong. Identification of a living or dead person is its own separate subject, built around age, stature, fingerprints, and similar features.
4. **Detection of lies:** wrong. Lie detection uses polygraphy, which measures physiological responses like pulse and skin conductance, not anything related to death.

Since thanatology is defined as the science of death in every form, physiological, somatic, cellular, and molecular, the only option that fits is death in all its aspects.

Let's summarize:

- Thanatology means the complete study of death and postmortem change.
- Paternity, identification, and lie detection are separate forensic topics with their own dedicated methods.

So the correct answer is death in all its aspects.

Quick Tip: Break the word down: "thanato-" means death, "-logy" means the study of.

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156. A dead body with suspected poisoning is having hypostasis of red brown or deep blue colour. It is suggestive of poisoning due to:

- (A) Nitrates.
- (B) Carbon monoxide.
- (C) Cyanides.
- (D) Barbiturates.

Correct Answer: (A) Nitrates.

SOLUTION:

Postmortem lividity normally settles as a plain purplish patch, but some poisons give it a specific color, and forensic pathologists read this color at autopsy as a clue to the poison involved.

1. **Nitrates:** nitrates and nitrites convert hemoglobin into methemoglobin, which is chocolate brown. As the methemoglobin fraction climbs, the discoloration can also shift toward a bluish, cyanotic shade, giving the mixed red brown to deep blue picture asked about here.
2. **Carbon monoxide:** carboxyhemoglobin produces a bright cherry red or pink lividity, a completely different shade from brown or blue.
3. **Cyanides:** cyanide stops the tissues from using the oxygen carried in the blood, so the blood stays bright red, again giving a pink or cherry red lividity rather than brown or blue.
4. **Barbiturates:** barbiturate deaths usually show a plain dusky purple lividity from simple respiratory depression and hypoxia, without the specific brown tint asked about here.

Only nitrate or nitrite poisoning explains both the brown shade from methemoglobin and the blue shade from the cyanosis that follows.

Let's summarize:

- Cherry red or pink lividity points to carbon monoxide or cyanide.
- Brown lividity that can turn bluish points to methemoglobin forming poisons like nitrates and nitrites.

So the poison responsible here is a nitrate.

Quick Tip: Match the lividity color to the poison: cherry red suggests carbon monoxide or cyanide, while brown that can turn bluish suggests a methemoglobin former like nitrates.

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157. Following cranial nerve is most commonly involved in patients with sarcoidosis:

- (A) II Cranial nerve.
- (B) III Cranial nerve.
- (C) VII Cranial nerve.
- (D) IX Cranial nerve.

Correct Answer: (C) VII Cranial nerve.

SOLUTION:

Sarcoidosis can involve the nervous system, a pattern called neurosarcoidosis, and cranial nerve palsy is one of its most recognizable signs. This question asks which cranial nerve gets hit most often.

1. **II Cranial nerve:** the optic nerve can be affected, showing up as optic neuritis, but this happens far less often than facial nerve disease.

2. **III Cranial nerve:** oculomotor nerve palsy is described in sarcoidosis but stays uncommon compared to facial nerve involvement.
3. **VII Cranial nerve:** the facial nerve is the one most typically affected. It can cause weakness on one or both sides of the face, and bilateral facial palsy in particular should make a clinician think of sarcoidosis. It is also the palsy seen in Heerfordt syndrome, along with parotid swelling, uveitis, and fever.
4. **IX Cranial nerve:** glossopharyngeal nerve involvement in sarcoidosis is rare and not the classic pattern tested here.

Since facial nerve palsy is the hallmark cranial neuropathy of sarcoidosis, the correct choice is the VII cranial nerve.

Let's summarize:

- Facial nerve (VII) palsy, sometimes bilateral, is the classic cranial nerve finding in sarcoidosis.
- Optic and oculomotor nerve involvement occur but are much less common.

So the answer is the VII cranial nerve.

Quick Tip: Recall Heerfordt syndrome: parotid swelling, uveitis, fever, and facial nerve palsy in sarcoidosis.

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158. In type II respiratory failure, there is:

- (A) Low PO_2 and low PCO_2 .
- (B) Low PO_2 and high PCO_2 .
- (C) Normal PO_2 and high PCO_2 .
- (D) Low PO_2 and normal PCO_2 .

Correct Answer: (B) Low PO_2 and high PCO_2 .

SOLUTION:

Respiratory failure is graded by what the arterial blood gas shows, and the two types are separated by whether carbon dioxide is also affected.

1. **Low PO_2 and low PCO_2 :** a drop in carbon dioxide usually reflects hyperventilation, which the body uses early on to compensate for low oxygen. This does not match type II failure.
2. **Low PO_2 and high PCO_2 :** this is the classic pattern of type II, or ventilatory, failure. The lungs cannot move enough air, so both oxygen intake and carbon dioxide removal suffer together.
3. **Normal PO_2 and high PCO_2 :** respiratory failure always includes a fall in PO_2 by definition, so a normal PO_2 rules this option out.
4. **Low PO_2 and normal PCO_2 :** this fits type I failure, where only oxygenation is impaired while ventilation, and so carbon dioxide clearance, stays intact.

Since type II failure means the lungs cannot ventilate properly, carbon dioxide builds up alongside the fall in oxygen.

Let's summarize:

- Type I failure: low PO_2 , normal or low PCO_2 (an oxygenation problem).

- Type II failure: low PO₂, high PCO₂ (a ventilation problem).

So the answer is low PO₂ with high PCO₂.

Quick Tip: Type I failure is an oxygenation problem only; type II adds a ventilation problem, so carbon dioxide also rises.

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159. The most common cause of acute cor pulmonale is:

- (A) Pneumonia.
- (B) Pulmonary thrombo-embolism.
- (C) Chronic obstructive pulmonary disease.
- (D) Primary spontaneous pneumothorax.

Correct Answer: (B) Pulmonary thrombo-embolism.

SOLUTION:

Cor pulmonale is right heart strain caused by lung disease, and it comes in an acute form and a chronic form, each with a different usual cause.

1. **Pneumonia:** severe infection can stress the right heart, but it is not the typical trigger for acute cor pulmonale.
2. **Pulmonary thrombo-embolism:** a large clot suddenly blocks a major pulmonary artery branch, so pulmonary pressure spikes within a very short time. The right ventricle has no time to adapt, dilates rapidly, and can fail, which is exactly why this is the leading cause of the acute form.
3. **Chronic obstructive pulmonary disease:** COPD does cause cor pulmonale, but through years of hypoxic vasoconstriction and

vessel remodeling, which makes it the classic cause of the chronic form, not the acute one.

4. **Primary spontaneous pneumothorax:** this can occasionally cause sudden cardiorespiratory compromise, especially if tension develops, but it is a far less frequent trigger of acute cor pulmonale than a large embolus.

Because a large pulmonary embolus raises right-sided pressure almost instantly, it is the most common cause of the acute presentation.

Let's summarize:

- Acute cor pulmonale: usually from a large pulmonary embolus.
- Chronic cor pulmonale: usually from long-standing lung disease such as COPD.

So the answer is pulmonary thrombo-embolism.

Quick Tip: Acute onset plus sudden right heart strain should point straight to a large pulmonary embolus.

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160. All of the following murmurs may be heard in patients with aortic regurgitation except:

- (A) High-pitched decrescendo diastolic murmur.
- (B) Soft, low pitched mid-diastolic rumbling murmur.
- (C) Mid-systolic ejection flow murmur.
- (D) Pansystolic murmur.

Correct Answer: (D) Pansystolic murmur.

SOLUTION:

Aortic regurgitation lets blood leak back into the left ventricle during diastole, and this leak can produce more than one type of murmur, so it helps to go through each option.

1. **High-pitched decrescendo diastolic murmur:** this is the murmur made directly by the leaking blood rushing back through the aortic valve, and it is the hallmark sound of AR.
2. **Soft, low pitched mid-diastolic rumbling murmur:** this is the Austin Flint murmur. The jet of regurgitant blood strikes the anterior leaflet of the mitral valve, making it flutter and creating a soft rumble that mimics mitral stenosis, so it is a recognized part of severe AR.
3. **Mid-systolic ejection flow murmur:** because the left ventricle has to eject a much bigger volume with each beat, blood crosses the aortic valve fast enough to make a flow murmur in systole, even when the valve itself is not narrowed.
4. **Pansystolic murmur:** a pansystolic murmur means the sound spans the whole of systole with a fairly constant pressure gradient, which is the signature of mitral regurgitation, tricuspid regurgitation, or a ventricular septal defect. AR's own murmur is diastolic, and its systolic murmur is only a brief flow murmur, so a true pansystolic murmur does not belong to AR.

Three of the options describe sounds that genuinely occur in AR, while the pansystolic murmur belongs to a different set of valve lesions entirely.

Let's summarize:

- AR produces a diastolic decrescendo murmur, sometimes an Austin Flint rumble, and sometimes a short systolic flow

murmur.

- A pansystolic murmur points to mitral regurgitation, tricuspid regurgitation, or a VSD, not AR.

So the exception is the pansystolic murmur.

Quick Tip: AR gives a diastolic murmur plus, at most, a short systolic flow murmur, never a full pansystolic murmur.

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161. Following is true regarding opening snap:

- (A) It is a high-pitched diastolic sound.
- (B) It is due to opening of stenosed aortic valve.
- (C) It indicates pulmonary arterial hypertension.
- (D) It precedes the aortic component of second heart sound.

Correct Answer: (A) It is a high-pitched diastolic sound.

SOLUTION:

The opening snap is a well-known extra sound in cardiology, tied closely to one specific valve lesion, so this question is really about knowing exactly where it comes from and when it occurs.

1. **It is a high-pitched diastolic sound:** correct. In mitral stenosis, the fused but still mobile leaflets snap taut as they dome open under the pressure from the left atrium, producing a crisp, high-pitched sound right at the start of diastole.
2. **It is due to opening of stenosed aortic valve:** wrong. The opening snap is generated by the mitral valve, not the aortic

valve; the aortic valve has no equivalent "snap" sound in aortic stenosis.

3. **It indicates pulmonary arterial hypertension:** wrong. The opening snap signals mitral stenosis. Pulmonary hypertension may coexist as a complication of severe mitral stenosis, but the snap itself does not indicate it.

4. **It precedes the aortic component of second heart sound:** wrong. The sequence is actually S1, then S2 (with its aortic component A2), and only after that the opening snap. Clinicians even measure the A2 to opening snap gap, since a shorter gap means tighter, more severe stenosis.

Only the first statement matches the true nature of the opening snap.

Let's summarize:

- The opening snap is a high-pitched sound from a stenotic mitral valve snapping open in early diastole.
- It always comes after S2, and a short A2-OS gap suggests severe mitral stenosis.

So the correct statement is that it is a high-pitched diastolic sound.

Quick Tip: The opening snap comes from a stenotic mitral valve snapping open, right after S2, not before it.

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162. All the following diseases are associated with peripheral blood eosinophilia except:

- (A) Allergic bronchopulmonary aspergillosis (ABPA)
- (B) Loeffler's syndrome
- (C) Pulmonary eosinophilic granuloma
- (D) Churg-Strauss syndrome

Correct Answer: (C) Pulmonary eosinophilic granuloma

SOLUTION:

The question tests which pulmonary or systemic condition, out of the four, does not raise the eosinophil count in the blood. Let's go through each one.

1. **Allergic bronchopulmonary aspergillosis (ABPA):** This is a hypersensitivity response to *Aspergillus* colonizing the airways in asthmatics or cystic fibrosis patients. It causes a marked rise in both blood eosinophils and total IgE, so it is linked with eosinophilia.
2. **Loeffler's syndrome:** This is a short-lived lung infiltrate caused by larvae passing through the lungs, most often *Ascaris lumbricoides*. It is a classic cause of peripheral blood eosinophilia along with cough and patchy chest X-ray shadows.
3. **Pulmonary eosinophilic granuloma:** This is another name for pulmonary Langerhans cell histiocytosis, a smoking-related disease of young adults. The word "eosinophilic" here refers to eosinophils seen within the lung granulomas on biopsy, not to a raised blood eosinophil count. Blood eosinophilia is not a usual finding in this disease.
4. **Churg-Strauss syndrome:** Now called eosinophilic granulomatosis with polyangiitis, this vasculitis has asthma, sinus

disease and a very high blood eosinophil count as core diagnostic features.

Three of the four conditions (ABPA, Loeffler's syndrome and Churg-Strauss syndrome) are established causes of peripheral blood eosinophilia. Pulmonary eosinophilic granuloma stands apart because its eosinophils are tissue-based, not blood-based.

Let's summarize:

- ABPA, Loeffler's syndrome and Churg-Strauss syndrome all raise the blood eosinophil count.
- Pulmonary eosinophilic granuloma (Langerhans cell histiocytosis) does not typically raise blood eosinophils despite its name.

So the exception is pulmonary eosinophilic granuloma, option (3).

Quick Tip: Think about which disease's 'eosinophilic' name refers to tissue findings on biopsy rather than a raised blood eosinophil count.

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163. A thirty-year man presented with nausea, fever and jaundice of 5-days duration. The biochemical tests revealed a bilirubin of 6.7 mg/dl (conjugated 5.0 mg/dl) with SGOT/SGPT (AST/ALT) of 123/900 IU/ml. The serological tests showed presence of HBsAg, IgM anti-HBc and HBeAg. The most likely diagnosis is:

- (A) Chronic hepatitis B infection with high infectivity
- (B) Acute hepatitis B infection with high infectivity
- (C) Chronic hepatitis B infection with low infectivity
- (D) Acute hepatitis B infection with low infectivity

Correct Answer: (B) Acute hepatitis B infection with high infectivity

SOLUTION:

This question is testing how to read hepatitis B serology together with the clinical picture to stage the infection and judge how infectious the patient is. Let's look at each option.

1. Chronic hepatitis B infection with high infectivity:

Chronic infection would show IgG anti-HBc, not IgM. The short 5-day history and very high ALT of 900 also argue against a chronic, longstanding process, so this does not fit.

2. Acute hepatitis B infection with high infectivity: IgM anti-HBc marks a recent, acute infection, and HBeAg positivity marks active viral replication and high infectivity. Both markers plus the short symptomatic history fit an acute, highly infectious hepatitis B.

3. Chronic hepatitis B infection with low infectivity: Low infectivity chronic hepatitis B is the HBeAg negative, anti-HBe positive inactive carrier state. This patient is HBeAg positive, not HBeAg negative, so this does not fit.

4. Acute hepatitis B infection with low infectivity: The infection is acute, which is right, but HBeAg positive means high infectivity, not low, so this option is only half correct.

The IgM anti-HBc confirms the infection is acute, and the HBeAg confirms it is highly infectious, so the picture matches acute hepatitis B with high infectivity.

Let's summarize:

- IgM anti-HBc means acute or recent infection; IgG anti-HBc means past or chronic infection.

- HBeAg positive means high viral replication and high infectivity.

So the correct diagnosis is acute hepatitis B infection with high infectivity, option (2).

Quick Tip: IgM anti-HBc marks a recent infection, and HBeAg marks high viral replication and infectivity.

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164. A 29-year-old woman was found to have a hemoglobin of 7.8 g/dl, with a reticulocyte count of 0.8%. The peripheral blood smear showed microcytic, hypochromic anemia. Hemoglobin A2 and hemoglobin F levels were 2.4% and 1.3% respectively. The serum iron was low and the total iron binding capacity (TIBC) was raised at 420 mcg/dL. The most likely cause of anemia is:

- (A) Iron deficiency anemia
- (B) Beta-thalassemia minor
- (C) Sideroblastic anemia
- (D) Anemia due to chronic infection

Correct Answer: (A) Iron deficiency anemia

SOLUTION:

This question gives a microcytic hypochromic anemia and asks us to use the iron studies and hemoglobin electrophoresis to find the cause. Let's test each option against the numbers given.

- 1. Iron deficiency anemia:** A low serum iron with a raised TIBC of 420 mcg/dL is the classic iron study pattern of iron deficiency, since the body increases transferrin to try to capture whatever

iron is available. A normal hemoglobin A2 of 2.4 percent also fits, since A2 is usually normal or low in iron deficiency.

2. **Beta-thalassemia minor:** This condition typically raises hemoglobin A2 above 3.5 percent because of reduced normal beta chain production, and iron studies are usually normal. The A2 here is only 2.4 percent, and the iron studies show clear deficiency, so this does not fit.
3. **Sideroblastic anemia:** Iron overload is typical here, with a high or normal serum iron and a low TIBC, along with ringed sideroblasts on marrow examination. This is the opposite of the low iron, high TIBC pattern seen in this patient.
4. **Anemia due to chronic infection:** In anemia of chronic disease, serum iron is low but TIBC is also low or normal because iron gets trapped inside macrophages rather than lost from the body. A raised TIBC does not match this pattern.

Only the low iron, high TIBC combination with a normal hemoglobin A2 points cleanly to true iron deficiency anemia.

Let's summarize:

- Low serum iron with high TIBC means the body is iron-starved: iron deficiency anemia.
- Raised hemoglobin A2 above 3.5 percent, not low iron, is the marker of beta-thalassemia minor.

So the diagnosis is iron deficiency anemia, option (1).

Quick Tip: Low serum iron with a raised TIBC and a normal hemoglobin A2 points to iron deficiency, not thalassemia trait.



165. A 70-years-old man was administered penicillin intravenously. Within 5 minutes, he developed generalized urticaria, swelling of lips, hypotension and bronchospasm. The first choice of treatment is to administer:

- (A) Chlorpheniramine injection
- (B) Epinephrine injection
- (C) High-dose hydrocortisone tablet
- (D) Nebulized salbutamol

Correct Answer: (B) Epinephrine injection

SOLUTION:

The patient's picture of sudden urticaria, lip swelling, low blood pressure and bronchospasm right after intravenous penicillin is anaphylaxis, a life-threatening allergic emergency. Let's look at what each drug can and cannot do here.

1. **Chlorpheniramine injection:** An antihistamine calms itching and hives but has no useful effect on blood pressure or airway narrowing, so it cannot be relied on as the first drug in a reaction this severe.
2. **Epinephrine injection:** Acting on both alpha and beta receptors, epinephrine tightens blood vessels to bring blood pressure back up, opens the airway by relaxing bronchial muscle, and slows further release of the chemicals driving the reaction. It is the only drug that treats all three danger signs (skin, airway and circulation) at once and works within minutes.
3. **High-dose hydrocortisone tablet:** Steroids take several hours to act and a tablet would be poorly absorbed in a patient with low blood pressure and gut hypoperfusion, so this has no

role as an immediate treatment; it is added later to prevent a delayed relapse.

4. **Nebulized salbutamol:** This eases the wheeze but leaves the hypotension and swelling untreated, so on its own it is not enough for full-blown anaphylaxis.

Only epinephrine reverses hypotension, bronchospasm and mediator release together, which is exactly what this patient needs right away.

Let's summarize:

- Anaphylaxis is a combined skin, airway and circulatory emergency.
- Epinephrine is the only drug that treats all three problems fast enough to save the patient.

So the first choice of treatment is epinephrine injection, option (2).

Quick Tip: In anaphylaxis with hypotension and bronchospasm, only one drug acts on the airway, blood pressure and mediator release together.

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166. A patient ingested some unknown substance and presented with myoclonic jerks, seizures, tachycardia and hypotension. The ECG showed a heart rate of 120/minute with QRS interval of 0.16 seconds. The arterial blood gas revealed a pH of 7.25, PCO₂ of 30 mmHg and HCO₃ of 15 mmol/L. The most likely cause of poisoning is ingestion of:

- (A) Amanita phalloides
- (B) Ethylene glycol
- (C) Imipramine
- (D) Phencyclidine

Correct Answer: (C) Imipramine

SOLUTION:

The key clue in this vignette is the QRS interval of 0.16 seconds, a markedly widened QRS, together with seizures, tachycardia, hypotension and a high anion gap metabolic acidosis. Let's test each poison against this pattern.

1. **Amanita phalloides:** This mushroom poison causes severe vomiting and diarrhea first, then delayed liver failure over days. It does not cause an acute wide QRS or seizures soon after ingestion, so it does not match.
2. **Ethylene glycol:** This does cause a high anion gap metabolic acidosis, but through glycolic and oxalic acid buildup, along with intoxication and calcium oxalate crystals in the urine. It is not known for widening the QRS complex, so the ECG finding here does not point to it.
3. **Imipramine:** As a tricyclic antidepressant, imipramine blocks cardiac sodium channels, which widens the QRS in direct proportion to the dose taken. It also blocks acetylcholine, alpha, and histamine receptors, producing seizures, myoclonus, fast heart rate and low blood pressure together, exactly what this patient has. The metabolic acidosis follows from the seizures and poor tissue perfusion.
4. **Phencyclidine:** This causes agitation, nystagmus, and usually a rise rather than a fall in blood pressure, and it is not linked with QRS widening, so it does not fit the ECG picture.

The wide QRS interval is the strongest clue here, and among the four options only a tricyclic antidepressant like imipramine explains it along with the seizures and hypotension.

Let's summarize:

- A widened QRS after an overdose points to sodium channel blockade, a hallmark of tricyclic antidepressant toxicity.
- Ethylene glycol causes acidosis without QRS widening; Amanita and phencyclidine do not fit this timeline or ECG pattern.

So the poison responsible is imipramine, option (3).

Quick Tip: A widened QRS complex after an overdose is the classic clue for tricyclic antidepressant poisoning.

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167. Significant loss of vision in a patient with hypertension can occur due to all of the following except:

- (A) Occipital infarct
- (B) Anterior ischemic optic neuropathy
- (C) Papilloedema
- (D) Retinal hemorrhage

Correct Answer: (C) Papilloedema

SOLUTION:

This question asks us to separate the hypertension related eye and brain changes that genuinely threaten vision from the one that mostly serves as a warning sign. Let's go through each option.

1. **Occipital infarct:** A stroke in the occipital lobe destroys the brain tissue that interprets visual signals, so it causes a real and

often marked loss of the visual field, sometimes affecting one whole side of vision in both eyes.

2. **Anterior ischemic optic neuropathy:** Malignant hypertension can starve the optic nerve head of blood, damaging the nerve fibers directly. This produces sudden, often severe and permanent visual loss in the affected eye.
3. **Papilloedema:** This is swelling of the optic disc from raised intracranial pressure in severe hypertension, and it is a sign of how advanced the disease is, but the swelling itself rarely reduces central vision by much. Most patients keep near normal visual acuity unless a further complication like bleeding sets in.
4. **Retinal hemorrhage:** Bleeding into the retina, especially near or in the macula, directly damages the light-sensing tissue used for sharp central vision, so it can cause significant visual loss.

Three of the four options directly injure either the visual pathway in the brain or the light-sensing retina and optic nerve tissue.

Papilloedema stands apart because the disc swelling itself, without an added complication, usually does not take away significant vision.

Let's summarize:

- Occipital infarct, optic neuropathy and retinal hemorrhage all directly damage vision-carrying tissue.
- Papilloedema is mainly a marker of severe hypertension and by itself rarely causes major vision loss.

So the exception is papilloedema, option (3).

Quick Tip: Ask which of the four changes actually damages vision-carrying tissue rather than just signaling severe disease.

168. A 33 years old lady presents with polydipsia and polyuria. Her symptoms started soon after a road traffic accident 6 months ago. The blood pressure is 120/80 mm Hg with no postural drop. The daily urinary output is 6-8 liters. Investigations showed Na 130 mEq/l, blood glucose 65 mg/dL, plasma osmolality 268 mosmol/l and urine osmolality 45 mosmol/l. The most likely diagnosis is:

- (A) Central diabetes insipidus
- (B) Nephrogenic diabetes insipidus
- (C) Resolving acute tubular necrosis
- (D) Psychogenic polydipsia

Correct Answer: (A) Central diabetes insipidus

SOLUTION:

This vignette is testing the classic link between head trauma and diabetes insipidus, along with the ability to read plasma and urine osmolality together. Let's check each option.

1. **Central diabetes insipidus:** Head injury 6 months earlier is a well known trigger for damage to the hypothalamic-pituitary axis that makes ADH. Without ADH, the kidney cannot concentrate urine, giving the very low urine osmolality of 45 mosmol/L seen here, huge urine volumes, and heavy compensatory water drinking that can push plasma sodium and osmolality down toward the lower normal range, as seen in this patient.
2. **Nephrogenic diabetes insipidus:** Here the kidney does not respond to ADH even though it is present, usually from causes like lithium therapy, longstanding hypercalcemia or chronic kidney disease. None of these triggers are mentioned, and the

timing right after trauma fits a central cause far better than a kidney-based one.

3. **Resolving acute tubular necrosis:** This is a polyuric recovery phase after a bout of acute kidney injury, usually caused by shock or a toxic insult, and it settles within days to a couple of weeks. Six months of unchanged symptoms and no history of an acute kidney injury event rule this out.
4. **Psychogenic polydipsia:** This is compulsive excess water drinking without an organic trigger, usually seen in psychiatric patients, and there is no such history here. The clear onset after a head injury points to an organic cause instead.

The combination of a preceding head injury, huge urine output, and a very dilute urine that fails to concentrate despite a low-normal plasma osmolality fits central diabetes insipidus caused by post-traumatic damage to ADH release.

Let's summarize:

- Head trauma is a classic cause of central diabetes insipidus through hypothalamic-pituitary injury.
- A very dilute urine despite large fluid losses, with no drug or renal cause, favors a central rather than a nephrogenic or psychiatric explanation.

So the diagnosis is central diabetes insipidus, option (1).

Quick Tip: Polyuria starting after a head injury, with persistently dilute urine, points to failure of ADH release.



169. A 41 year old patient presented with chronic diarrhea for 3 months. A d-xylose absorption test was ordered to look for:

- (A) Carbohydrate malabsorption due to mucosal disease.
- (B) Carbohydrate malabsorption due to chronic pancreatitis.
- (C) Fat malabsorption due to mucosal disease.
- (D) Fat malabsorption due to chronic pancreatitis.

Correct Answer: (A) Carbohydrate malabsorption due to mucosal disease.

SOLUTION:

The D-xylose absorption test is a simple way to check whether the small intestine's lining can absorb a basic sugar without help from pancreatic enzymes or bile. Since chronic diarrhea can come from either a gut lining problem or a pancreatic problem, this test helps tell them apart.

- 1. Carbohydrate malabsorption due to mucosal disease:** D-xylose needs only an intact intestinal wall to pass into the blood. If the wall is damaged, as in celiac disease or tropical sprue, xylose stays in the gut and blood levels are low. This is exactly what the test picks up.
- 2. Carbohydrate malabsorption due to chronic pancreatitis:** Pancreatic disease affects enzyme output, not the gut wall itself, so xylose is absorbed normally here and the test result stays normal.
- 3. Fat malabsorption due to mucosal disease:** fat absorption depends on bile salts and enzymes acting on fat, xylose has nothing to do with fat, so this option does not match what the test measures.

4. Fat malabsorption due to chronic pancreatitis: same problem, xylose is a sugar test, not a fat test, and pancreatitis does not affect xylose absorption at all.

Because xylose absorption depends only on a healthy gut lining and not on pancreatic enzymes, an abnormal test means the small bowel mucosa itself is diseased, and it is a carbohydrate (sugar), not fat, that is being tested.

Let's summarize:

- D-xylose absorption needs no pancreatic enzymes, only a healthy mucosa.
- Low levels point to mucosal disease, normal levels rule out mucosal disease and instead suggest a pancreatic cause of malabsorption.

So the d-xylose test was ordered to look for carbohydrate malabsorption caused by mucosal disease.

Quick Tip: D-xylose needs only an intact gut mucosa, not pancreatic enzymes, to be absorbed.

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170. Narcolepsy is characterized by all of the following except:

- (A) Sleep paralysis.
- (B) Cataplexy.
- (C) Hallucination.
- (D) Snoring.

Correct Answer: (D) Snoring.

SOLUTION:

Concept:

Narcolepsy is best remembered through its four classic features, often called the narcolepsy tetrad.

Step 1: List the tetrad.

The four features are excessive daytime sleepiness, cataplexy (sudden muscle weakness with emotion), sleep paralysis (unable to move at sleep onset or offset), and hypnagogic/hypnopompic hallucinations (vivid dream like images while falling asleep or waking).

Step 2: Match each option against the tetrad.

Sleep paralysis and cataplexy are named members of the tetrad. Hallucination, meaning the hypnagogic/hypnopompic type, is also a named member.

Step 3: Test snoring.

Snoring comes from vibration of soft tissue in the upper airway during sleep, a mechanical breathing issue seen in obstructive sleep apnea. Narcolepsy instead comes from loss of hypocretin producing neurons in the hypothalamus, a completely different mechanism. There is no direct link between narcolepsy and snoring.

Step 4: Conclude.

Since three of the four options fit the known tetrad and snoring does not belong to it, snoring is the exception.

Snoring

Quick Tip: Recall the narcolepsy tetrad: sleepiness, cataplexy, sleep paralysis, hallucinations.

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171. A 25-year female presented with mild pallor and moderate hepatosplenomegaly. Her hemoglobin was 92 g/l and fetal hemoglobin level was 65%. She has not received any blood transfusion till date. She is most likely to be suffering from:

- (A) Thalassemia major.
- (B) Thalassemia intermedia.
- (C) Hereditary persistent fetal hemoglobin, homozygous state.
- (D) Hemoglobin D, homozygous state.

Correct Answer: (B) Thalassemia intermedia.

SOLUTION:

This question asks us to separate three conditions that can all show a raised fetal hemoglobin (HbF): thalassemia major, thalassemia intermedia, and homozygous hereditary persistent fetal hemoglobin (HPFH). The clues are a transfusion free course, mild pallor, a big liver and spleen, hemoglobin 9.2 g/dL, and HbF 65%.

1. **Thalassemia major:** this form causes severe anemia from infancy and needs lifelong regular transfusions to survive. A 25 year old who has never been transfused cannot have this diagnosis.

2. **Thalassemia intermedia:** patients keep a moderate hemoglobin without regular transfusions, but they still get hepatosplenomegaly from extra blood cell formation and carry raised HbF, sometimes quite high. This matches the clinical picture here closely.
3. **Homozygous HPFH:** gamma genes take over completely for the missing beta genes, so hemoglobin stays near normal and there is usually no anemia and no organ enlargement. Because this patient does have anemia and hepatosplenomegaly, true homozygous HPFH is not the best fit.
4. **Homozygous hemoglobin D:** this variant causes only mild hemolysis and does not push HbF up to 65%, so it does not explain the fetal hemoglobin finding.

Putting the findings together, a patient who is alive and well without transfusions, but who has real anemia, splenomegaly and a very high HbF, is best explained by thalassemia intermedia, a form where the body compensates partly with HbF but still cannot fully avoid anemia and organ enlargement.

Let's summarize:

- No transfusion history plus survival to adulthood rules out thalassemia major.
- Anemia and hepatosplenomegaly argue against true homozygous HPFH, which is normally silent.
- The overall picture, transfusion independent anemia with organ enlargement and high HbF, fits thalassemia intermedia.

So the most likely diagnosis is thalassemia intermedia, even though some printed answer keys list homozygous HPFH for this question.

Quick Tip: True homozygous HPFH is clinically silent; anemia plus splenomegaly favors thalassemia intermedia.

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172. All of the following cause high anion gap metabolic acidosis except:

- (A) Lactic acidosis.
- (B) Salicylate poisoning.
- (C) Ethylene glycol poisoning.
- (D) Ureterosigmoidostomy.

Correct Answer: (D) Ureterosigmoidostomy.

SOLUTION:

Concept:

Metabolic acidosis splits into two groups based on the anion gap: high gap, where an unmeasured acid appears, and normal gap, where bicarbonate is lost directly and chloride rises to compensate.

Step 1: Check the classic acid producing causes.

Lactic acid built up during poor tissue oxygenation is an unmeasured acid. Salicylate overdose releases organic acids. Ethylene glycol is broken down in the liver into glycolic and oxalic acids. All three add an unmeasured acid to the blood, so all three widen the anion gap.

Step 2: Check ureterosigmoidostomy.

After this surgery, urine sits in the colon, and the colon wall swaps chloride from the urine for bicarbonate, which then leaves the body in

stool. The net effect is bicarbonate loss matched by chloride gain, not the appearance of a new unmeasured acid.

Step 3: Compare the two mechanisms.

Since chloride rises as bicarbonate falls, sodium minus (chloride plus bicarbonate) stays about the same, so the anion gap stays normal here, unlike the other three causes.

Step 4: Conclude.

The one that does not raise the anion gap is ureterosigmoidostomy.

Ureterosigmoidostomy

Quick Tip: Ureterosigmoidostomy causes chloride gain and bicarbonate loss, so the gap stays normal.

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173. Chylous ascites is caused by all of the following except:

- (A) Colloid carcinoma of stomach.
- (B) Tuberculosis.
- (C) Trauma.
- (D) Nephrotic syndrome.

Correct Answer: (A) Colloid carcinoma of stomach.

SOLUTION:

Chylous ascites is a milky, fat rich fluid that builds up when lymph carrying digested fat leaks out of blocked or damaged lymphatic channels into the peritoneal cavity. The question asks which of four options is NOT a standard cause of this condition.

1. **Colloid carcinoma of stomach:** this mucinous tumor spreads over the peritoneum and produces a thick, gelatinous, mucin filled fluid, closer to what is seen in pseudomyxoma peritonei, rather than the lipid rich milky fluid of true chylous ascites, so it does not classically cause chylous ascites.
2. **Tuberculosis:** TB can infect and block mesenteric lymph nodes, backing up chyle into the peritoneal space, a well known cause of chylous ascites.
3. **Trauma:** injury or surgery near the cisterna chyli or major lymphatic trunks can tear them open, letting chyle spill directly into the abdomen.
4. **Nephrotic syndrome:** though less common, lymphatic abnormalities linked with nephrotic syndrome are accepted as a cause of chylous ascites.

Three of the four options damage or block true lymphatic/chyle carrying channels, giving the classic milky fluid, while colloid carcinoma of the stomach instead produces a mucin rich fluid through peritoneal spread, a different process altogether.

Let's summarize:

- True chylous ascites needs lymphatic blockage or injury, as seen with TB, trauma and nephrotic syndrome.
- Colloid (mucinous) gastric carcinoma instead produces a mucinous, gelatinous ascitic fluid, not classic chyle.

So the exception, the one not causing true chylous ascites, is colloid carcinoma of the stomach.

Quick Tip: True chylous ascites needs lymphatic blockage; mucinous gastric cancer instead gives gelatinous fluid.

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174. Adverse effects of Phenytoin include the following except:

- (A) Lymphadenopathy.
- (B) Ataxia.
- (C) Hypercalcemia.
- (D) Hirsutism.

Correct Answer: (C) Hypercalcemia.

SOLUTION:

Concept:

Phenytoin's side effect list is often grouped by system: nervous system (ataxia, nystagmus), skin and connective tissue (hirsutism, gum overgrowth), immune/lymphatic (pseudolymphoma with lymphadenopathy), and bone/mineral metabolism.

Step 1: Confirm the nervous system effect.

High phenytoin levels affect the cerebellum, giving ataxia, unsteady gait and nystagmus. This is a dose related, well known toxicity.

Step 2: Confirm the lymphatic effect.

Long term phenytoin can trigger a benign but alarming pseudolymphoma reaction, with swollen lymph nodes that can mimic cancer.

Step 3: Confirm the skin/connective tissue effect.

Phenytoin is notorious for causing gum overgrowth, coarse facial features and hirsutism with long term use.

Step 4: Check the calcium claim.

Phenytoin speeds up liver breakdown of vitamin D. Less active vitamin D means less calcium absorbed from food, so calcium levels tend to fall, not rise, over time. This can lead to osteomalacia in chronic users. Hypercalcemia is the opposite of what phenytoin actually does.

Step 5: Conclude.

Since three options are true phenytoin effects and calcium moves down, not up, hypercalcemia is the exception.

Hypercalcemia

Quick Tip: Phenytoin speeds vitamin D breakdown, causing hypocalcemia, not hypercalcemia.

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175. Drug induced myopathy can be caused by all of the following except:

- (A) Atorvastatin.
- (B) D-penicillamine.
- (C) Ciprofloxacin.
- (D) Chloroquine.

Correct Answer: (C) Ciprofloxacin.

SOLUTION:

Drug induced myopathy means muscle fiber damage caused directly by a medicine, showing up as weakness, pain or raised muscle enzymes. The question asks which of four drugs does NOT cause this.

1. **Atorvastatin:** statins are the most familiar drug cause of myopathy, from mild aches to serious rhabdomyolysis, through direct toxic effects on muscle cells.
2. **D-penicillamine:** this drug can set off an autoimmune reaction that inflames muscle, producing a picture much like polymyositis.
3. **Ciprofloxacin:** this fluoroquinolone is best known for harming tendons, especially the Achilles tendon, causing pain, inflammation or even rupture. This is a tendon problem, separate from the muscle fiber itself, so it does not fit as a myopathy cause.
4. **Chloroquine:** with long term use this drug piles up inside the lysosomes of muscle cells and produces a specific vacuolar myopathy.

Three of the drugs damage muscle fibers directly through toxic or autoimmune mechanisms, while ciprofloxacin's musculoskeletal risk lies in the tendons, not the muscle itself.

Let's summarize:

- Atorvastatin, D-penicillamine and chloroquine each damage muscle tissue by a different route and are established causes of myopathy.
- Ciprofloxacin instead is linked with tendinopathy and tendon rupture, not primary muscle disease.

So the drug that does not cause myopathy here is ciprofloxacin.

Quick Tip: Ciprofloxacin's musculoskeletal risk is tendon damage, not muscle fiber myopathy.

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176. The commonest site for hypertensive intracerebral bleed is:

- (A) Putamen
- (B) Cerebellum
- (C) Pons
- (D) Midbrain

Correct Answer: (A) Putamen

SOLUTION:

Step 1: Think about vessel anatomy.

Hypertensive bleeds happen where small deep arteries take blood almost straight off a bigger vessel. That sharp branching point takes the most pressure.

Step 2: Map that anatomy to the brain.

The lenticulostriate branches of the middle cerebral artery supply the putamen, and they branch off at a near right angle. This makes the

putamen the classic weak point.

Step 3: Compare with the other choices.

The cerebellum, pons and midbrain are all possible sites of hypertensive hemorrhage, but each is struck far less often than the putamen, and the pons ranks behind the thalamus in frequency.

Step 4: Settle on the answer.

Since the putamen accounts for the largest share of hypertensive bleeds, it is the correct choice.

Putamen

Quick Tip: Hypertensive bleeds strike the sharply angled lenticulostriate branches feeding the basal ganglia hardest.

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177. A 12 years old child presents with fever and cervical lymphadenopathy. Oral examination shows a gray membrane on the right tonsil extending to the anterior pillar. Which of the following medium will be ideal for the culture of the throat swab for a rapid identification of the pathogen?

- (A) Nutrient agar
- (B) Blood agar
- (C) Loeffler's serum slope
- (D) Lowenstein-Jensen medium

Correct Answer: (C) Loeffler's serum slope

SOLUTION:

Step 1: Pin down the diagnosis first.

A gray membrane over the tonsil that extends onto the anterior pillar, plus fever and neck node swelling in a child, points straight to diphtheria. Trying to peel the membrane off usually makes it bleed underneath.

Step 2: Think about what rapid means in the lab.

The question asks for speed, not just accuracy. Two media are used for *C. diphtheriae*: Loeffler's serum slope for a quick look under the microscope, and tellurite agar for a slower but more specific culture.

Step 3: Work out why Loeffler's wins on speed.

Because Loeffler's medium is rich in coagulated serum, the bacillus multiplies fast and takes on its typical shape within hours. A stained smear at 6 to 8 hours already shows the granules that mark this organism, well before a tellurite plate would show colonies.

Step 4: Eliminate the distractors.

Nutrient agar carries no special nutrients for this fastidious organism. Blood agar helps with strep throat, not diphtheria speed. Lowenstein-Jensen medium is built for the tuberculosis bacillus and would not even grow *C. diphtheriae* well.

Step 5: Land on the answer.

The medium built for a fast presumptive diagnosis of *C. diphtheriae* is Loeffler's serum slope.

Loeffler's serum slope

Quick Tip: Gray membrane on the tonsil in a child means diphtheria; pick the medium that shows the bacillus fastest.

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178. A 56 year old man has painful rashes over his right upper eyelid and forehead for the last 48 hours. He underwent chemotherapy for Non-Hodgkin's lymphoma one year ago. His temperature is 98 degree F, blood pressure 138/76 mm Hg, and pulse is 80/minute. Examination shows no other abnormalities. Which of the following is the most likely diagnosis?

- (A) Impetigo
- (B) Herpes zoster
- (C) Pyoderma gangrenosum
- (D) Erysipelas

Correct Answer: (B) Herpes zoster

SOLUTION:

Step 1: Start with the immune status.

A history of chemotherapy for lymphoma a year ago tells us this man's cell-mediated immunity is likely still weak. That single fact should make us think of reactivation infections.

Step 2: Match the rash to a nerve, not just a body part.

The right upper eyelid and forehead are covered by one nerve branch, the ophthalmic branch of the trigeminal nerve (V1). Painful skin disease that sits exactly along one nerve's territory, and stays on one side of the midline, is a strong clue.

Step 3: Recall which virus hides in nerve ganglia.

Varicella-zoster virus, the chickenpox virus, sits quietly in a dorsal root or cranial nerve ganglion for years after the first infection. When immunity drops, as it does after chemotherapy, the virus wakes up and spreads along that one nerve, causing herpes zoster.

Step 4: Check the pain and fever pattern.

Herpes zoster often causes pain before the rash even shows up, and this patient has severe pain with a rash only two days old, but a normal temperature and pulse, unlike a spreading bacterial skin infection which usually brings fever and systemic illness.

Step 5: Cross off the bacterial mimics.

Impetigo is superficial and crusted, not dermatomal or this painful. Erysipelas is a spreading, feverish bacterial infection. Pyoderma gangrenosum makes deep ulcers, usually on the legs, tied to bowel or blood disease, and does not follow a nerve.

Step 6: Conclude.

The nerve-bound painful rash in this immunocompromised man is herpes zoster.

Herpes zoster

Quick Tip: Painful rash along the V1 dermatome in an immunocompromised patient points to shingles.

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179. A 24 years old cook in a hostel mess suffered from enteric fever 2 years back. The chronic carrier state in this patient can be diagnosed by:

- (A) Vi agglutination test
- (B) Blood culture in Brain Heart Infusion broth
- (C) Widal test
- (D) C-Reactive protein

Correct Answer: (A) Vi agglutination test

SOLUTION:

Step 1: Think about where the bacteria actually live in a carrier.

After the fever settles, a small number of people keep *Salmonella typhi* living quietly in their gallbladder, often alongside gallstones. From there, bacteria are shed on and off into the stool, but they are not usually floating in the bloodstream anymore.

Step 2: Work out why blood tests of acute infection fail here.

Blood culture and the Widal test both depend on things that are strongest during active fever, live bacteria in the blood for culture, and rising O and H antibodies for Widal. Two years after the illness, neither of these is expected to still be positive or high, so both are poor choices for carrier detection.

Step 3: Bring in the Vi antigen angle.

The Vi capsular antigen is shed continuously by bacteria sitting in the gallbladder of a carrier. This constant low-level exposure keeps the Vi antibody level up in the blood long after acute illness, unlike O and H antibodies which fade.

Step 4: Confirm the logic with the test's actual use.

Because of this, the Vi agglutination test is used as a screening test to flag likely carriers, especially food handlers like cooks, so they can be

confirmed later with stool and bile culture.

Step 5: Set aside CRP.

C-Reactive protein just shows that inflammation is happening somewhere in the body, it cannot point to typhoid or to carriage.

Step 6: Conclude.

The test built to catch a chronic carrier here is the Vi agglutination test.

Vi agglutination test

Quick Tip: Chronic typhoid carriers keep shedding Vi antigen, so Vi antibody stays high long after the fever is gone.

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180. Chlamydia trachomatis serovars D-K cause:

- (A) Arteriosclerosis
- (B) Trachoma
- (C) Lymphogranuloma venereum
- (D) Urethritis

Correct Answer: (D) Urethritis

SOLUTION:

Step 1: Split Chlamydia trachomatis into its three serovar families.

This one species is split into three groups by serovar, and each group sticks to its own tissue. A to C prefers the eye, D to K prefers the genital tract, and L1 to L3 prefers lymph nodes.

Step 2: Anchor the eye group first.

A, B, Ba and C are the trachoma serovars, causing a slow scarring eye infection common where hygiene is poor. This rules out trachoma for our D-K group.

Step 3: Anchor the lymph node group.

L1, L2 and L3 cause lymphogranuloma venereum, a sexually spread disease that inflames and swells lymph nodes in the groin. This is a different serovar set from D-K, so it is also ruled out.

Step 4: Place D-K correctly.

That leaves D through K, which are the serovars adapted to the genital tract lining. They cause urethritis in men, cervicitis in women, and can also cause conjunctivitis or pneumonia in a newborn exposed during delivery.

Step 5: Dismiss arteriosclerosis.

Arteriosclerosis is a disease of blood vessel walls, and Chlamydia trachomatis has no established role in causing it.

Step 6: Conclude.

Since D-K is the genital serovar group, the disease they cause here is urethritis.

Urethritis

Quick Tip: A-C causes trachoma, D-K causes genital infections such as urethritis, L1-L3 causes LGV.



181. Mixed lymphocyte culture is used to identify:

- (A) MHC class I antigen
- (B) MHC class II antigen
- (C) B lymphocytes
- (D) T helper cells

Correct Answer: (B) MHC class II antigen

SOLUTION:

Step 1: Picture the actual experiment.

Take white blood cells from two different people and mix them in one dish. If their tissue types differ, something in the mix will react.

Step 2: Ask which tissue molecules trigger that reaction.

Helper T-cells (CD4 positive) are the ones that notice foreign HLA molecules on the other person's cells and respond by multiplying. CD4 cells are built to read MHC class II molecules specifically, not class I.

Step 3: Turn that reaction into a measurement.

More proliferation in the dish means the two people's MHC class II types (mostly HLA-DR) are more different from each other. Less proliferation means the class II types are closer to a match. This is exactly why the test is used before transplants, to gauge class II compatibility.

Step 4: Separate this from a class I test.

Testing MHC class I differences instead uses a cytotoxic T-lymphocyte assay, where CD8 cells kill mismatched target cells, a different setup from the proliferative mixed lymphocyte culture.

Step 5: Dismiss the cell-identity options.

B lymphocytes and T helper cells are both present and playing a role in the culture, but the test itself is not aimed at identifying either cell type, it is aimed at the MHC molecules driving the reaction.

Step 6: Conclude.

The mixed lymphocyte culture reads out MHC class II differences.

MHC class II antigen

Quick Tip: MLC proliferation is driven by CD4 helper T-cells reacting to an MHC class II mismatch.

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182. In a patient with AIDS, chorioretinitis is typically caused by:

- (A) Cytomegalovirus
- (B) Toxoplasma gondii
- (C) Cryptococcus neoformans
- (D) Histoplasma capsulatum

Correct Answer: (A) Cytomegalovirus

SOLUTION:

Step 1: Set the immune backdrop.

In AIDS, chorioretinitis (inflammation at the back of the eye) tends to show up only once the CD4 count has fallen very low, generally under 50 to 100 cells per microliter, which is when several dormant organisms reactivate.

Step 2: Recall the appearance that clinches CMV.

CMV retinitis has a very recognizable look on fundus exam, patchy white retinal necrosis with hemorrhages, sometimes called a pizza-pie fundus. It is by far the most common infectious cause of retinitis once CD4 counts crash in AIDS.

Step 3: Explain the reactivation mechanism.

CMV, like other herpesviruses, hides quietly in the body after first exposure. When T-cell immunity fails badly, as in late AIDS, the virus reactivates and spreads through blood to reach and damage the retina.

Step 4: Weigh the alternative organisms.

Toxoplasma gondii does cause retinochoroiditis and matters in AIDS, but it is more famous for brain abscesses, and it trails CMV as a cause of pure retinitis. Cryptococcus neoformans is the classic AIDS meningitis organism, eye involvement from it is uncommon. Histoplasma capsulatum mainly causes lung and blood-borne disease, and its eye link (ocular histoplasmosis syndrome) is a separate condition seen without AIDS.

Step 5: Conclude.

The organism that best fits AIDS-related chorioretinitis is Cytomegalovirus.

Cytomegalovirus

Quick Tip: Low CD4 count in AIDS plus a pizza-pie fundus appearance points to CMV retinitis.



183. A 17 year old girl with keratitis and severe pain in the eye came to the hospital, and Acanthamoeba keratitis was suspected. The patient gave a history of the following four points. Which one of these is NOT a risk factor for Acanthamoeba keratitis?

- (A) Extended wear contact lenses
- (B) Exposure to dirty water
- (C) Corneal trauma
- (D) Squamous blepharitis

Correct Answer: (D) Squamous blepharitis

SOLUTION:

This question checks which of the four listed points is not a genuine risk factor for Acanthamoeba keratitis in a young girl with painful keratitis.

1. **Extended wear contact lenses:** wearing lenses for long hours, especially with poor lens hygiene or tap water rinsing, is the leading risk factor for this infection. This is a true risk factor.
2. **Exposure to dirty water:** Acanthamoeba lives in soil, dust, and untreated water. Contact with dirty water, ponds, or hot tubs is a well known route of infection. This is a true risk factor.
3. **Corneal trauma:** an injury, especially one involving soil or plant matter, breaks the corneal surface and lets the ameba enter. This is a true risk factor.
4. **Squamous blepharitis:** this is chronic lid margin inflammation, usually tied to Staphylococcus and to bacterial or marginal keratitis, not to Acanthamoeba, which needs an outside source such as lenses, water, or trauma.

The point that does not belong on this risk list is squamous blepharitis, since Acanthamoeba keratitis is tied to an outside exposure route and not to lid margin disease.

Let's summarize:

- Contact lens wear and dirty water exposure are the two classic risk factors for Acanthamoeba keratitis.
- Corneal trauma opens a route for the ameba to enter the stroma.
- Blepharitis raises the risk of bacterial keratitis, not Acanthamoeba keratitis.

So the correct answer is squamous blepharitis, option 4.

Quick Tip: Acanthamoeba keratitis needs an outside exposure route: contact lenses, dirty water, or trauma. Lid margin disease does not fit that pattern.

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184. A 2 year old child is brought to the emergency department with a history of fever and vomiting. On examination he has neck rigidity. CSF examination shows polymorphs more than 2000/microL, protein 100 mg/dL, and glucose 10 mg/dL. Gram stain shows Gram negative coccobacilli. Culture shows growth of bacteria only on chocolate agar and not on blood agar. The causative agent is

- (A) Neisseria meningitidis
- (B) Haemophilus influenzae
- (C) Branhamella catarrhalis
- (D) Legionella pneumophila

Correct Answer: (B) Haemophilus influenzae

SOLUTION:

The CSF findings, high polymorphs, high protein, and low glucose, tell us this is bacterial meningitis. The Gram stain and the culture behaviour narrow down the exact bug.

1. **Neisseria meningitidis**: this organism is a diplococcus, paired like coffee beans, not a coccobacillus, and it grows fine on plain blood agar. It does not need a special factor rich medium, so it does not match the clue.
2. **Haemophilus influenzae**: this is a small Gram negative coccobacillus that must have both factor X (hemin) and factor V (NAD) to grow. Chocolate agar is made by heating blood agar, which breaks open red cells and releases both factors. Plain blood agar keeps most of the V factor locked inside intact red cells, so this organism grows on chocolate agar but not on blood agar, exactly as described. It is also a well known cause of meningitis in toddlers who have not had the Hib vaccine.
3. **Branhamella catarrhalis**: also a diplococcus, it grows on ordinary blood agar without any special factor requirement, and it rarely if ever causes meningitis.
4. **Legionella pneumophila**: this bug needs buffered charcoal yeast extract agar with iron and cysteine to grow, not chocolate agar, and it causes a lung infection, not this meningitis picture.

Only *Haemophilus influenzae* fits both the coccobacillary shape on Gram stain and the growth only on chocolate agar.

Let's summarize:

- Chocolate agar supplies factor X and factor V by lysing red cells during heating.

- *Haemophilus influenzae* needs both factors, so it fails to grow on plain blood agar.
- The Gram negative coccobacillary shape also points away from the diplococci *Neisseria* and *Moraxella*.

So the causative agent is *Haemophilus influenzae*, option 2.

Quick Tip: Growth only on chocolate agar, not blood agar, means the organism needs both factor X and factor V: think *Haemophilus influenzae*.

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185. A cook prepares sandwiches for 10 people going for a picnic. Eight out of them develop severe gastroenteritis within 4 to 6 hours of consuming the sandwiches. It is likely that on investigation the cook is found to be a carrier of

- (A) *Salmonella typhi*
- (B) *Vibrio cholerae*
- (C) *Entamoeba histolytica*
- (D) *Staphylococcus aureus*

Correct Answer: (D) *Staphylococcus aureus*

SOLUTION:

The main clue here is speed. Eight of ten people fell sick only 4 to 6 hours after the meal, and that timing alone rules most of the options out.

1. **Salmonella typhi:** typhoid takes one to three weeks to show up, and even ordinary *Salmonella* food poisoning usually takes half a day or more. Far too slow for this outbreak.

2. **Vibrio cholerae**: cholera usually spreads through contaminated water, not a single batch of sandwiches, and its incubation runs from several hours up to a few days. It does not fit a sharp point-source outbreak like this.
3. **Entamoeba histolytica**: amoebic dysentery develops over days to weeks, nowhere close to a 4 to 6 hour window.
4. **Staphylococcus aureus**: this organism commonly lives on the skin and in the nose of healthy people. A cook who carries it can contaminate food while preparing it. Left at room temperature, the bacteria grow and release a heat stable toxin into the food. Eating the toxin brings on sudden vomiting and diarrhoea within just 1 to 6 hours, matching the timeline exactly.

Since the outbreak struck so fast, it has to be a pre-formed toxin already sitting in the sandwiches, and Staphylococcus aureus is the organism best known for causing this pattern through food handler carriage.

Let's summarize:

- A short incubation of a few hours means a toxin was already in the food before it was eaten.
- Salmonella, Vibrio, and Entamoeba all need much longer to cause symptoms.
- Staphylococcus aureus carried by a food handler is the classic cause of rapid onset food poisoning like this.

So the cook is most likely a carrier of Staphylococcus aureus, option 4.

Quick Tip: A very short incubation period of a few hours points to a pre-formed toxin, the hallmark of Staphylococcus aureus food poisoning.

186. A man presents to a STD clinic with urethritis and urethral discharge. Gram stain shows numerous pus cells but no microorganism. The culture is negative on routine laboratory media. The most likely agent is

- (A) *Chlamydia trachomatis*
- (B) *Haemophilus ducreyi*
- (C) *Treponema pallidum*
- (D) *Neisseria gonorrhoeae*

Correct Answer: (A) *Chlamydia trachomatis*

SOLUTION:

This case is a puzzle by exclusion. Pus cells are present, so this is a real infection, yet no bug is seen on Gram stain and nothing grows on the routine plates. That combination is the real clue.

1. ***Chlamydia trachomatis*:** this bacterium can only live and multiply inside human cells, so it is invisible on a standard Gram stain and will not grow on ordinary bacterial media. It is the leading cause of non-gonococcal urethritis and needs special methods like cell culture or PCR to detect. This matches both negative findings perfectly.
2. ***Haemophilus ducreyi*:** this organism causes a painful genital ulcer called chancroid, not typical urethritis, and if grown, it needs a special enriched medium, and it would still be visible as small bacilli on a stain, so it does not explain a completely organism-free Gram stain.
3. ***Treponema pallidum*:** this is the cause of syphilis, presenting as a painless ulcer, and it can never be cultured on artificial media

and needs dark field microscopy, not routine Gram stain, to be seen at all. The clinical picture of urethral discharge does not fit syphilis either.

4. **Neisseria gonorrhoeae**: gonorrhoea classically shows Gram negative diplococci sitting inside pus cells on Gram stain, and it grows readily on selective chocolate agar. Since this stain shows no organism and the culture is negative, gonorrhoea is unlikely.

Only Chlamydia trachomatis explains why pus cells are seen, yet no organism appears on the stain and nothing grows on routine media.

Let's summarize:

- Gonococcal urethritis shows visible diplococci on Gram stain and grows on selective media.
- Chlamydia is an obligate intracellular organism, so it is silent on Gram stain and on routine culture.
- Chancroid and syphilis both present differently and are not the best fit here.

So the most likely agent is Chlamydia trachomatis, option 1.

Quick Tip: No organism on Gram stain plus a negative routine culture in a man with urethritis points to Chlamydia trachomatis, an obligate intracellular bacterium.

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187. All of the following groups of newborns are at an increased risk of hypoglycemia EXCEPT

- (A) Birth asphyxia
- (B) Respiratory distress syndrome
- (C) Maternal diabetes
- (D) Post term infant

Correct Answer: (D) Post term infant

SOLUTION:

Neonatal hypoglycemia has a short list of well known risk groups, built around low glucose reserves, high glucose demand, or excess insulin. Let's check each option against that list.

1. **Birth asphyxia:** a stressed, oxygen-starved newborn burns through glucose fast through anaerobic metabolism, making this a solid risk factor.
2. **Respiratory distress syndrome:** working hard to breathe raises the baby's energy needs sharply, which is another recognised driver of low blood glucose.
3. **Maternal diabetes:** high maternal glucose crosses the placenta, the fetus responds with extra insulin, and once the cord is cut and the glucose supply stops, that extra insulin drives glucose down fast. This is one of the textbook classic risk groups.
4. **Post term infant:** a baby simply born after completing a normal term, without growth restriction or placental problems, does not carry a built-in glucose handling problem the way a preterm, growth restricted, or diabetic mother's infant does.

Three of the four options load extra metabolic stress or extra insulin onto the newborn. Being post term by itself does none of that, so it stands apart from the other three.

Let's summarize:

- Perinatal stress states like asphyxia and respiratory distress push glucose use up.
- Infants of diabetic mothers face a post-birth insulin surge.
- A straightforward post term birth is not part of the classic hypoglycemia risk list.

So the exception here is the post term infant, option 4.

Quick Tip: Perinatal stress, prematurity, growth restriction, and maternal diabetes raise hypoglycemia risk; simply being born post term does not.

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188. A twenty year old woman has been brought to casualty with blood pressure 70/40 mm Hg, pulse rate 120/minute, and a positive urine pregnancy test. She should be managed by

- (A) Immediate laparotomy
- (B) Laparoscopy
- (C) Culdocentesis
- (D) Resuscitation and medical management

Correct Answer: (A) Immediate laparotomy

SOLUTION:

A young woman with a positive pregnancy test who is now in shock, with blood pressure 70/40 mm Hg and pulse 120/minute, is bleeding internally, most likely from a ruptured ectopic pregnancy. The question is what to do about it right away.

1. **Immediate laparotomy:** open surgery gives fast access to the bleeding tube, lets the surgeon stop the bleeding directly, and can

be started within minutes. Fluids and blood are given at the same time, but the actual source of blood loss can only be shut off in the operating room, so this is the step that saves her life.

2. **Laparoscopy:** this needs general anesthesia and gas insufflation of the abdomen, both of which can worsen an already unstable patient, so it is kept for patients who are stable, not for one in shock.
3. **Culdocentesis:** this old test just confirms blood behind the uterus with a needle, it does not treat anything, and running it here would only burn time that this patient does not have.
4. **Resuscitation and medical management:** fluids alone cannot plug a bleeding fallopian tube. Without surgery to stop the source, she keeps losing blood and her shock only gets worse.

The bleeding source has to be controlled surgically, and that happens fastest through immediate laparotomy, run together with resuscitation, not as a substitute for it.

Let's summarize:

- Shock with a positive pregnancy test signals a likely ruptured ectopic until proven otherwise.
- Laparoscopy and culdocentesis both cost time this unstable patient cannot afford.
- Fluids alone do not stop internal bleeding, the source must be controlled surgically.

So the correct management is immediate laparotomy, option 1.

Quick Tip: Shock plus a positive pregnancy test means a likely ruptured ectopic pregnancy: the bleeding must be controlled surgically without delay.

189. In a case of recurrent spontaneous abortion, which of the following investigations is unwanted?

- (A) Hysteroscopy
- (B) Testing for antiphospholipid antibodies
- (C) Testing for TORCH infections
- (D) Thyroid function tests

Correct Answer: (C) Testing for TORCH infections

SOLUTION:

Recurrent pregnancy loss has a focused list of causes worth testing for: uterine cavity problems, antiphospholipid antibody syndrome, thyroid disease, and chromosomal issues. Let's run through each test against that list.

1. **Hysteroscopy:** this looks directly inside the uterus for a septum, scar tissue, or fibroids pressing into the cavity, all known and fixable causes of repeated loss. It stays on the recommended list.
2. **Testing for antiphospholipid antibodies:** antiphospholipid syndrome is one of the best documented, treatable causes of recurrent loss, so checking for lupus anticoagulant and anticardiolipin antibodies is a core part of the workup.
3. **Testing for TORCH infections:** these infections are known to cause a single sporadic loss or birth defects in an ongoing pregnancy, but they have not been shown to cause a repeating pattern of loss. Because of this low yield, current guidelines advise against routine TORCH testing in recurrent pregnancy loss.

4. Thyroid function tests: untreated hypothyroidism is linked to pregnancy loss and is simple to fix once found, so checking thyroid function is a standard, useful part of the workup.

Three of these four tests, hysteroscopy, antiphospholipid antibody testing, and thyroid function testing, all target causes with real evidence behind them. TORCH testing stands apart because it targets a cause of single losses, not recurrent ones, and current guidelines do not recommend it here.

Let's summarize:

- The recurrent pregnancy loss workup targets uterine, antiphospholipid, endocrine, and genetic causes.
- TORCH infections explain isolated losses or fetal anomalies, not a repeating pattern.
- Modern guidelines list TORCH screening as unhelpful here, not thyroid testing.

So the unwanted investigation is TORCH testing, option 3. Note that the original answer key for this 2002 question named thyroid function tests as the unwanted test, but current obstetric guidelines have moved thyroid testing into the recommended workup and instead flag routine TORCH screening as the low-yield test, which is why this answer has been updated.

Quick Tip: Hysteroscopy, antiphospholipid antibody testing, and thyroid function tests are all part of the standard recurrent pregnancy loss workup; TORCH screening is not.



190. In a case of vesicular mole, all of the following are high risk factors for the development of choriocarcinoma except:

- (A) Serum HCG levels $> 1,00,000$ IU/ml
- (B) Uterus size larger than 16 weeks
- (C) Features of thyrotoxicosis
- (D) Presence of bilateral theca lutein cysts of ovary

Correct Answer: (C) Features of thyrotoxicosis

SOLUTION:

Step 1: Understanding the Question:

A hydatidiform mole can turn into a persistent trophoblastic tumour or choriocarcinoma after evacuation. Doctors use a set of markers to sort moles into low risk and high risk groups. We need to spot the option that is NOT one of these markers.

Step 2: Key Formula or Approach:

The classic set of high risk markers (often called Hammond's criteria) are: hCG level above 1,00,000 IU/ml, uterus size well beyond the expected date, theca lutein cysts bigger than 6 cm (usually bilateral), and maternal age past 40.

Step 3: Detailed Explanation:

Check each option against this list. $\text{hCG} > 1,00,000$ IU/ml matches the first marker directly. A uterus larger than 16 weeks reflects excess trophoblastic bulk and matches the second marker. Bilateral theca lutein cysts match the third marker exactly. Thyrotoxicosis, on the other hand, happens because hCG at very high levels weakly stimulates the thyroid, so it shows up alongside a high risk mole, but it is a downstream complication, not one of the markers used to define

the risk group.

This paper's own answer key left this question unmarked in 2002, showing that even then it was seen as debatable, so this answer rests on the standard Hammond framework rather than a printed key.

Step 4: Final Answer:

The finding that is not a defining high risk marker is thyrotoxicosis.

Features of thyrotoxicosis

Quick Tip: Recall Hammond's criteria for high risk mole: hCG, uterine size, theca lutein cysts, age. Thyrotoxicosis is a complication, not a defining marker.

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191. Use of oral contraceptive pills is known to protect against all of the following malignancies except:

- (A) Ovarian carcinoma
- (B) Endometrial carcinoma
- (C) Uterine sarcoma
- (D) Carcinoma cervix

Correct Answer: (D) Carcinoma cervix

SOLUTION:

Oral contraceptive pills change the hormone environment of the reproductive tract, and this has different effects on different cancers. Let's go through each option.

1. **Ovarian carcinoma:** OCPs stop ovulation, which reduces the repeated trauma and repair of the ovarian surface that is thought to drive cancer here. Protective, so not the answer.
2. **Endometrial carcinoma:** the progestin component keeps the uterine lining thin and stops unopposed estrogen from driving overgrowth. Protective, so not the answer.
3. **Uterine sarcoma:** this tumour grows from the muscle wall, and OCPs are not known to lower its risk. Not protective, but the classic teaching point is really about the cervix below.
4. **Carcinoma cervix:** cervical cancer is driven by persistent HPV infection of the cervical cells. The pill has no action against the virus, so it gives no protection, and some data even show a small increase in risk with long term use.

Since the question asks which malignancy OCPs do NOT protect against among the standard teaching set (ovary and endometrium being the protected ones), the intended answer is carcinoma cervix.

Let's summarize:

- OCPs lower the risk of ovarian and endometrial cancer.
- OCPs give no protection against cervical cancer, which is an HPV driven disease.

So the malignancy not protected against is carcinoma of the cervix.

Quick Tip: OCPs lower ovarian and endometrial cancer risk but do nothing against HPV driven cervical cancer.

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192. Use of Levo-Norgestrel Releasing intrauterine contraceptive device is helpful in all of the following conditions except:

- (A) Menorrhagia
- (B) Dysmenorrhea
- (C) Premenstrual symptoms
- (D) Pelvic inflammatory disease

Correct Answer: (D) Pelvic inflammatory disease

SOLUTION:

The LNG-IUS works by releasing a steady dose of levonorgestrel inside the uterus. Let's check each condition listed.

1. **Menorrhagia:** the device thins the endometrial lining, so monthly blood loss drops sharply. It is a standard treatment for heavy periods.
2. **Dysmenorrhea:** less endometrial bulk and lower prostaglandin release mean less cramping, so it helps with painful periods too.
3. **Premenstrual symptoms:** by flattening the usual cyclical hormone swings acting on the endometrium, many users report milder premenstrual complaints.
4. **Pelvic inflammatory disease:** this is the opposite case. Any active or recent pelvic infection is a reason to avoid inserting an IUD altogether, since the procedure can carry bacteria upward. The device does not treat PID, and PID is a contraindication to fitting it.

Let's summarize:

- LNG-IUS eases menorrhagia, dysmenorrhea, and premenstrual symptoms through local progestin action.
- Active pelvic inflammatory disease is a contraindication to insertion, not a condition it treats.

So the exception is pelvic inflammatory disease.

Quick Tip: LNG-IUS helps heavy/painful periods and PMS but active PID is a contraindication to insertion, not an indication.

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193. The most common cause of vesico-vaginal fistula in India is:

- (A) Gynae surgery
- (B) Irradiation
- (C) Obstructed labour
- (D) Trauma

Correct Answer: (C) Obstructed labour

SOLUTION:

Vesico-vaginal fistula causes a woman to leak urine constantly through the vagina, because the wall between bladder and vagina has broken down. The main cause depends heavily on where in the world the woman lives.

1. **Gynae surgery:** a bladder injury during hysterectomy is the top cause in high income countries with wide access to surgery, but surgical rates and settings differ in India, so this is not the leading cause here.
2. **Irradiation:** radiotherapy for cervical cancer can damage the bladder wall over months, but this accounts for only a small share of cases overall.
3. **Obstructed labour:** in India, many women in rural areas cannot reach a hospital quickly when labour stalls. The fetal head

presses on the bladder and vaginal tissue against the pelvic bone for hours or days, cutting off blood supply. The tissue later sloughs and a fistula opens up. This obstetric route is by far the most frequent cause in India.

4. **Trauma:** direct injury is possible but rare compared with the obstetric route.

Let's summarize:

- Developed countries: gynae surgery (hysterectomy) is the top cause of VVF.
- India and other low resource settings: prolonged obstructed labour is the top cause, due to delayed access to emergency obstetric care.

So the most common cause of VVF in India is obstructed labour.

Quick Tip: In India, delayed access to obstetric care makes obstructed labour, not surgery, the leading cause of VVF.

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194. Differential diagnosis of pre-menstrual tension includes all of the following except:

- (A) Psychiatric depressive disorder
- (B) Panic disorder
- (C) General anxiety disorder
- (D) Chronic fatigue syndrome

Correct Answer: (D) Chronic fatigue syndrome

SOLUTION:

Step 1: Understanding the Question:

Premenstrual tension causes mood swings, irritability and physical discomfort that show up before the period and settle once bleeding starts. Several other conditions can mimic it, so doctors keep a differential list. We need to pick the option that sits outside the usual differential grouping.

Step 2: Key Formula or Approach:

The classic differentials taught alongside PMT are conditions whose symptoms do NOT clear with the onset of menses, chiefly depressive disorder, panic disorder, and generalised anxiety disorder, since these mood and anxiety states can be confused with the premenstrual mood change but persist across the whole cycle.

Step 3: Detailed Explanation:

Depressive disorder can present with low mood and fatigue that look like PMT but continue after the period starts, so it must be ruled out. Panic disorder causes sudden anxiety attacks with physical symptoms that can be mistaken for premenstrual anxiety, so it is checked too. Generalised anxiety disorder causes constant, cycle independent worry that can overlap with premenstrual irritability.

Chronic fatigue syndrome is its own separate diagnosis built around long lasting unexplained tiredness with defined diagnostic criteria, and it is usually taught as a general medical condition to exclude rather than as one of the core psychiatric mood and anxiety differentials paired with PMT. This makes it the odd one out among the four options.

This paper did not print an official answer for this question in 2002, so this reasoning is based on the standard teaching grouping rather than a printed key.

Step 4: Final Answer:

The condition that stands apart from the core psychiatric differential list for PMT is chronic fatigue syndrome.

Chronic fatigue syndrome

Quick Tip: Core psychiatric differentials for PMT are depression, panic disorder, and GAD; CFS is a separate general medical diagnosis.

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195. All of the following are risk factors for postoperative infection after hysterectomy except:

- (A) Surgery for malignancy
- (B) Age > 50 yrs
- (C) Urinary catheterization > 7 days
- (D) Use of blood transfusion

Correct Answer: (B) Age > 50 yrs

SOLUTION:

Infection after hysterectomy can show up as a wound infection, pelvic abscess, or urinary tract infection. Certain patient and surgery factors raise this risk. Let's check each option.

1. **Surgery for malignancy:** cancer surgery tends to be longer and more extensive, with more tissue handling and sometimes lymph node removal, all of which increase infection risk. This is a real risk factor.

2. **Age > 50 yrs:** age by itself, using a simple cutoff of 50 years, is not part of the standard, specific list of risk factors quoted for postoperative infection after hysterectomy. Other factors like obesity, diabetes, and operative time matter far more than crossing this particular age line.
3. **Urinary catheterization > 7 days:** the longer a catheter stays in, the higher the chance bacteria travel up into the bladder, so prolonged catheterization is a clear and well documented risk factor.
4. **Use of blood transfusion:** needing a transfusion often means more blood loss and a longer, harder operation, and transfusion itself can dampen immune response a little, so this raises infection risk too.

Let's summarize:

- Real risk factors here: malignancy surgery, catheter left in more than 7 days, blood transfusion.
- A flat age cutoff of 50 years is not one of the specific established risk factors.

So the exception is age greater than 50 years.

Quick Tip: Malignancy surgery, prolonged catheter use, and transfusion raise infection risk; a simple age over 50 cutoff does not.

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196. Best parameter for estimation of fetal age by ultrasound in the third trimester is:

- (A) Femur length
- (B) Biparietal diameter
- (C) Abdominal circumference
- (D) Inter-ocular distance

Correct Answer: (B) Biparietal diameter

SOLUTION:

Ultrasound dating relies on measuring fixed fetal structures and comparing them against known growth charts. The best single parameter changes across the three trimesters. Let's look at each option for the third trimester specifically.

1. **Femur length:** useful and commonly combined with head measurements for a composite estimate, and some texts note it holds up reasonably well later in pregnancy since it is less affected by head shape changes, but it is not the parameter classically named as the standard for this stage in most exam texts.
2. **Biparietal diameter:** measured across the fetal skull at the level of the thalami, this has been the traditional core parameter for fetal age on ultrasound since the second trimester and continues to be used through the third trimester as one of the standard dating measurements.
3. **Abdominal circumference:** this measurement tracks fetal soft tissue and liver size, so it is more sensitive to nutritional status and growth restriction than to fixed skeletal age, making it a growth parameter rather than a dating parameter.
4. **Inter-ocular distance:** this is a niche measurement checked when the face or orbits look abnormal on scan, not a routine tool

for estimating gestational age.

Let's summarize:

- Biparietal diameter is the traditional standard parameter used for fetal age across pregnancy, including the third trimester.
- Abdominal circumference is better for judging growth than age, and inter-ocular distance is a specialised, not routine, measurement.

So the best parameter here is the biparietal diameter.

Quick Tip: BPD is the traditional standard measurement used for fetal age estimation on ultrasound through the third trimester.

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197. A 11-year-old boy presented with complaints of pain in the right arm near the shoulder. X-ray examination revealed an expansile lytic lesion in the upper third of the humerus. What is the most likely diagnosis?

- (A) Giant cell tumor
- (B) Unicameral bone cyst
- (C) Osteochondroma
- (D) Parosteal osteosarcoma

Correct Answer: (B) Unicameral bone cyst

SOLUTION:

Step 1: Build a short list of proximal humerus lesions.

In a growing child, a lytic area near the shoulder end of the humerus usually comes from one of four lesions: unicameral bone cyst, giant

cell tumor, osteochondroma or parosteal osteosarcoma. The trick is matching the age and the X-ray pattern to the right one.

Step 2: Use age as the first filter.

This boy is 11 years old, meaning his epiphyseal plate is still open. Giant cell tumor needs a fused, mature epiphysis to spread into, so it almost never appears before the growth plate closes. That removes giant cell tumor from the list.

Step 3: Use the X-ray pattern as the second filter.

Osteochondroma sticks out from the surface of the bone as a capped bony stalk, and parosteal osteosarcoma sits on the outer cortex as a dense sclerotic mass. Neither of these fits an expansile lytic lesion inside the shaft. That leaves the lesion sitting centrally in the metaphysis.

Step 4: Confirm the classic site.

The proximal humeral metaphysis, right under the growth plate, is the single most common location for a unicameral bone cyst in the body. The cyst is filled with clear fluid, thins the cortex from within, and can make the bone expand, which matches the X-ray description exactly.

Step 5: State the answer.

The lesion is a unicameral bone cyst.

Unicameral bone cyst

Quick Tip: Expansile lytic lesion in the metaphysis near an open growth plate in a child points to a simple (unicameral) bone cyst.

198. The classical flexion and rotation deformities at the hip and knee joints as a sequela of poliomyelitis are due to the contracture of:

- (A) Tensor fasciae latae
- (B) Gastrocnemius
- (C) Tendo Achilles
- (D) Hamstrings

Correct Answer: (A) Tensor fasciae latae

SOLUTION:

Step 1: Think about muscle balance after polio.

Polio does not weaken every muscle equally. Some groups are spared and stay strong, and if those spared muscles are not balanced by an opposing weak group, they slowly pull the joint into a fixed abnormal position. This kind of unopposed pull is what causes late polio deformities.

Step 2: Trace the action of each muscle in the options.

The tensor fasciae latae flexes and internally rotates the hip and, through its long iliotibial band, also drags the knee into flexion with outward rotation of the leg. The gastrocnemius and tendo Achilles mainly bend the ankle into equinus. The hamstrings mainly bend the knee.

Step 3: Match the deformity pattern in the question.

The question describes a combined flexion plus rotation deformity spanning both the hip and the knee, not just the ankle or just the knee. Only a muscle that crosses both the hip and the knee, and produces rotation as well as flexion, fits this description.

Step 4: Confirm the muscle.

The tensor fasciae latae, through the iliotibial tract, is the muscle that spans both joints and gives this exact flexion, abduction and rotation pattern in chronic polio patients.

Step 5: Conclude.

Tensor fasciae latae

Quick Tip: A spared, tight tensor fasciae latae (via the iliotibial band) pulls both hip and knee into flexion with rotation in old polio cases.

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199. What would be the most reliable test for an acutely injured knee of a 27-year-old athlete?

- (A) Anterior drawer test
- (B) Posterior drawer test
- (C) Lachman test
- (D) Steinmann test

Correct Answer: (C) Lachman test

SOLUTION:

Step 1: Picture the injured knee right after the accident.

A 27 year old athlete with a fresh knee injury most likely has torn the ACL. Right after the injury the joint fills with blood and the muscles around it tighten up on their own to guard the joint.

Step 2: Think about what each test needs from the knee.

The anterior drawer test needs the knee flexed all the way to 90 degrees before the tibia is pulled forward. A swollen, guarded knee usually cannot bend that far without real pain, so the test becomes hard to perform and easy to read wrong.

Step 3: See why the Lachman test avoids that problem.

The Lachman test only needs a slight bend, around 20 to 30 degrees, which a swollen knee can usually tolerate. Because the hamstrings are less able to guard the joint at this smaller angle, the true amount of forward slide of the tibia shows up more clearly. This is why it stays accurate even in a freshly injured knee.

Step 4: Set aside the wrong options.

The posterior drawer test looks for a posterior cruciate tear, which is a different ligament and different injury pattern. The Steinmann test is a provocation test for a torn meniscus, not for ligament laxity, so it does not answer the question about ligament injury reliability.

Step 5: State the conclusion.

For an acutely injured knee, the Lachman test is the more reliable choice over the anterior drawer test, because it works around the pain and spasm that block a full 90 degree flexion right after injury.

Lachman test

Quick Tip: In an acute knee injury, swelling and hamstring spasm block full flexion, so the low-flexion Lachman test outperforms the anterior drawer

test.

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200. The medial meniscus of the knee joint is injured more often than the lateral meniscus because the medial meniscus is relatively:

- (A) More mobile
- (B) Less mobile
- (C) Thinner
- (D) Attached loosely to the femur

Correct Answer: (B) Less mobile

SOLUTION:

Step 1: Compare how each meniscus sits in the joint.

The lateral meniscus is almost a full circle and has no attachment to the lateral collateral ligament, so it floats somewhat freely over the tibial surface. The medial meniscus is a more open crescent, and its rim is fixed to the deep part of the medial collateral ligament and the joint capsule.

Step 2: Turn this anatomy into a mechanical idea.

A structure that is tied down cannot slide out of the path of a moving joint surface. When the knee bends and twists at the same time, a fixed structure gets squeezed between the femur above and the tibia below, while a loosely held structure can slip aside and avoid the squeeze.

Step 3: Apply this to the two menisci.

Since the medial meniscus is firmly fixed by its ligament attachment, it has far less freedom to move during a twisting injury than the lateral

meniscus does. This lower mobility is exactly why it ends up trapped and torn more often.

Step 4: Check the distractors.

It is not more mobile, that is backwards. Its thickness is not the deciding factor in tear frequency. It is not loosely attached at the femur, the opposite is true, it is firmly tied to the medial collateral ligament.

Step 5: Conclude.

Less mobile

Quick Tip: The medial meniscus is fixed to the deep medial collateral ligament and capsule, so it cannot slide away during twisting injuries, unlike the more mobile lateral meniscus.