NEET PG 2002 Combined Paper Question Paper with Solutions

General Instructions

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

(ii) It comprises 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: Understand what the ACL does.

The anterior cruciate ligament (ACL) stops the tibia (shin bone) from sliding forward under the femur (thigh bone) and also checks excess rotation of the knee. Once it is torn, the knee loses this forward translation control, so any activity that pushes the tibia forward becomes hard to do without the knee giving way.

Step 2: Work out what downhill walking does to the knee.
Walking downhill needs the knee to stay slightly bent while the quadriceps muscle contracts to control the descent, an eccentric contraction. A contracting quadriceps pulls the tibia forward through the patellar tendon. In a normal knee the ACL resists this pull. In an ACL deficient knee there is nothing to resist it, so the tibia shifts forward and the knee feels unstable or gives way. This is why patients with ACL tears specifically struggle with downhill walking, running, and quick deceleration.

Step 3: Check why the other activities are less affected.

(B) Walking uphill: Uphill walking loads the hip more and needs less quadriceps braking, so it does not push the tibia forward the same way. Patients manage this reasonably well.

(C) Sit cross leg: This needs knee flexion and some rotation but no strong quadriceps contraction against gravity, so ACL deficient patients can usually do this.

(D) Getting up from sitting: This does load the quadriceps, but the knee moves from flexion to extension in a controlled, low speed way without the sudden forward shear seen in downhill walking, so it is tolerated better.

Step 4: Final Answer.
The activity that is hardest for an ACL deficient knee is downhill walking, because it combines knee flexion with a strong quadriceps pull that shifts the tibia forward with no ligament to stop it.

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.

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

(A) Mastoidectomy

(B) Medical management

(C) Underlay myringoplasty

(D) Insertion of ventilation tube

Correct Answer: (A) Mastoidectomy

Solution:

Step 1: Classify chronic suppurative otitis media (CSOM).
CSOM is divided into two types. The tubotympanic (safe) type has a central perforation and mainly mucosal disease. The atticoantral (unsafe) type has a marginal or attic perforation and is usually associated with cholesteatoma, skin trapped inside the middle ear and mastoid that slowly destroys bone.

Step 2: Understand why the atticoantral type is called unsafe.
Cholesteatoma can erode the ossicles, the bony wall of the facial nerve canal, the labyrinth, and even the roof of the middle ear or mastoid, leading to complications such as facial palsy, a labyrinthine fistula, meningitis, or a brain abscess. Medicines cannot remove this skin debris or stop the bone erosion.

Step 3: Identify the correct treatment.
Because the disease is a destructive, expanding mass rather than a simple infection, the only way to clear it and prevent complications is surgical removal of the diseased mastoid air cells and cholesteatoma matrix, done through a mastoidectomy.

Step 4: Rule out the other options.

(B) Medical management: Antibiotic ear drops or systemic antibiotics can control secondary infection for a while but cannot clear cholesteatoma or stop bone destruction, so this is not curative.

(C) Underlay myringoplasty: This repairs a simple central perforation in tubotympanic disease. It does not address mastoid cholesteatoma, so it is wrong here.

(D) Insertion of a ventilation tube: This is used for otitis media with effusion (glue ear), not for cholesteatoma disease of the attic and antrum.

Step 5: Final Answer.
The treatment of choice for atticoantral CSOM is mastoidectomy.

Mastoidectomy

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

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: Recall what an antrochoanal polyp is.
An antrochoanal polyp starts inside the maxillary sinus, grows a stalk that passes through the sinus opening, crosses the nasal cavity, and can even hang down into the nasopharynx (throat) through the choana. It is usually a single, benign polyp and does not go away on its own.

Step 2: Decide the treatment goal.
Because the polyp keeps growing and blocks the nose, treatment must remove the whole polyp, including the part sitting inside the maxillary sinus (the antral part). Removing only the visible nasal part without the antral stalk leads to quick recurrence.

Step 3: Choose the surgical approach suited to children.
The classic Caldwell-Luc operation goes through the canine fossa, above the upper teeth, to open the maxillary sinus from outside. In a child this route can injure developing tooth buds and disturb facial bone growth, so it is avoided when possible. Endoscopic intranasal polypectomy removes the polyp and its antral attachment through the nostril under endoscopic guidance, without cutting through the face, and is gentler on a growing skull.

Step 4: Rule out the other options.

(A) Caldwell-Luc operation: Effective in adults but not the first choice in children because of the risk to facial growth and tooth buds.

(C) Corticosteroids: May shrink some inflammatory nasal polyps but cannot remove a large antrochoanal polyp with an antral stalk, and it soon regrows.

(D) Wait and watch: The polyp keeps enlarging and can cause worsening nasal block or aspiration risk if it hangs low, so observation alone is not appropriate.

Step 5: Final Answer.
The most appropriate management in a child is endoscopic intranasal polypectomy with removal of the antral part.

Intranasal polypectomy

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

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: Recall the nerve supply of the ear canal.
The skin of the external ear canal gets sensory supply from several nerves: the auriculotemporal branch of the trigeminal nerve (V) in the front and upper wall, the auricular branch of the vagus nerve (X), also called Arnold's nerve, mainly in the floor and posterior wall, and small contributions from the facial (VII) and glossopharyngeal (IX) nerves.

Step 2: Connect the vagus nerve to the cough reflex.
The vagus nerve also carries sensation from the larynx, trachea, and bronchi, the same pathway that triggers the cough reflex when these areas are irritated. Because Arnold's nerve is a sensory branch of the vagus, touching or cleaning the ear canal skin it supplies can send signals along the vagus that trigger the same cough reflex, a phenomenon called Arnold's reflex or the ear-cough reflex.

Step 3: Rule out the other options.

(A) Trigeminal nerve (V): Supplies sensation to part of the ear canal but is not linked to the cough reflex pathway.

(B) C1, C2 innervation: The great auricular nerve, from C2 and C3, supplies the outer ear (pinna) skin, not the classic cough-triggering pathway of the canal.

(D) Facial nerve (VII) branches: Give a small sensory contribution to the ear canal but are not the nerve responsible for the

cough reflex.

Step 4: Final Answer.
The cough response on cleaning the ear canal is mediated by the vagus nerve, cranial nerve X, through its auricular branch (Arnold's nerve).

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: Recall what a normal Rinne test means.
The Rinne test compares air conduction (AC) and bone conduction (BC) in one ear. Normally AC is louder and longer than BC, called a

positive Rinne. A negative Rinne, where BC is heard louder or longer than AC, is the classic sign of conductive hearing loss in that ear, because the middle ear is not transmitting sound well through air.

Step 2: Recall what Weber's test tells us.
In Weber's test a vibrating tuning fork is placed on the midline of the skull. In conductive hearing loss the sound lateralizes to the affected ear, because the blocked middle ear shuts out background masking noise. In sensorineural hearing loss the sound lateralizes to the better, normal ear, because the damaged cochlea on the bad side cannot pick up the signal as well.

Step 3: Put the two findings together.
Here the Rinne is negative on the right, which alone would suggest a right conductive loss. But Weber lateralizes to the left ear, that is, away from the ear with the negative Rinne. This combination does not fit simple conductive loss, where Weber should have gone to the same (right) ear.

Step 4: Recognize the false negative Rinne.
When one ear has severe or profound sensorineural hearing loss, a tuning fork placed on the mastoid of that bad ear can still be heard, not by the damaged cochlea, but because the vibration travels through the skull bone and is picked up by the good cochlea on the other side. This makes bone conduction seem present or even louder than air conduction in the bad ear, producing a Rinne test that looks negative even though the ear has no real conductive problem. This is called a false negative Rinne.

Step 5: Apply this to the case.
The right ear shows a falsely negative Rinne because the right cochlea

is so severely affected that the left cochlea is actually picking up the bone-conducted sound. The true finding is a right sensorineural hearing loss, confirmed by the Weber test lateralizing to the good left ear, exactly as expected in sensorineural loss.

Step 6: Rule out the other options.

(A) Right conductive hearing loss: Would need Weber to lateralize to the right ear, not the left, so this does not fit.

(C) Left sensorineural hearing loss: The left ear has a normal, positive Rinne and Weber lateralizes toward it, meaning the left ear is the better ear, not the diseased one.

(D) Left conductive hearing loss: A conductive loss on the left would make Weber go to the left, which matches on its own, but the left Rinne here is positive, not negative, so the left ear is not conductively affected.

Step 7: Final Answer.
The findings fit a severe right sensorineural hearing loss causing a false negative Rinne test on the right, with Weber correctly lateralizing 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.

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: Recall the typical profile of ethmoidal (nasal) polyps.
Ethmoidal polyps arise from the mucosa of the ethmoid sinus, most often as part of an allergic or inflammatory process. They are typically seen in adults, most commonly between the third and fifth decades of life, and are uncommon in young children.

Step 2: Check option (A), allergy as a cause.
Allergic rhinitis and chronic inflammation are well established causes of ethmoidal polyp formation, so this statement is correct and not the answer.

Step 3: Check option (B), first decade of life.
Ethmoidal polyps are uncommon in the first decade of life. When nasal polyps are seen in a young child, they should raise suspicion for cystic fibrosis rather than being treated as routine ethmoidal polyps. This makes the statement that they occur in the first decade of life incorrect for typical ethmoidal polyps.

Step 4: Check option (C), bilateral distribution.
Ethmoidal polyps are classically bilateral and multiple, arising from ethmoidal air cells on both sides, unlike the antrochoanal polyp,

which is typically unilateral and single. So this statement is correct.

Step 5: Check option (D), association with asthma.
Ethmoidal polyps are frequently associated with bronchial asthma and aspirin sensitivity, together known as the aspirin-exacerbated respiratory disease triad (nasal polyps, asthma, aspirin intolerance). So this statement is correct.

Step 6: Final Answer.
The statement that is NOT correct is that ethmoidal polyps occur in the first decade of life.

Occur in first decade of life

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

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: Understand what fetal hydronephrosis with normal amniotic fluid means.

Fetal hydronephrosis is swelling of the kidney's collecting system, usually picked up on a routine antenatal ultrasound. Amniotic fluid volume mainly depends on fetal urine output, so a normal amniotic fluid volume at 34 weeks tells us that the fetal kidneys, taken together, are still making enough urine and are not in a state of severe or bilateral obstruction that threatens fetal well being.

Step 2: Decide whether urgent action is needed before birth.

Fetal intervention, such as placing a shunt to decompress the kidney before birth, is reserved for severe cases with bilateral disease and oligohydramnios (low amniotic fluid), where ongoing pregnancy risks permanent kidney and lung damage. Since fluid is normal here, there is no indication for an invasive fetal procedure, and no reason to deliver the baby early either.

Step 3: Decide the correct sequence of care.

The safest course is to let the pregnancy continue to term, since prematurity carries its own risks and the normal amniotic fluid shows the kidneys are coping well. After birth, the baby should have a proper radiological work-up, typically an ultrasound in the first days of life, sometimes repeated after the first week since neonatal dehydration can under-read the swelling early on, followed by further tests like a micturating cystourethrogram or a diuretic renogram if needed, to find the cause and decide if surgery such as pyeloplasty is actually required.

Step 4: Rule out the other options.

(A) Fetal intervention: Not needed, since this invasive step is

reserved for severe bilateral obstruction with reduced amniotic fluid, which is not the case here.

(B) Premature termination followed by pyeloplasty: Unnecessarily exposes the baby to the risks of prematurity for a condition that is not causing fetal compromise, and surgery cannot even be planned properly before postnatal imaging confirms true obstruction.

(D) Delivery at term followed by pyeloplasty: Jumps straight to surgery without first confirming, through postnatal imaging, whether the hydronephrosis is even due to a surgical (obstructive) cause, since many antenatally detected cases turn out to be mild or resolve without surgery.

Step 5: Final Answer.
With normal amniotic fluid, the right plan is to deliver at term and then work up the kidney with radiological studies before deciding on any surgery.

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: Read the lab pattern.

The child has rickets that does not respond to plain vitamin D, so this is vitamin D refractory (resistant) rickets. The calcium is 9 mg/dl, which is normal. The phosphate is 2.4 mg/dl, which is low for a child. The alkaline phosphatase is 1040 IU, which is very high and points to active bone turnover. Parathyroid hormone and bicarbonate are both normal.

Step 2: Rule out renal tubular acidosis.

Both distal and proximal renal tubular acidosis make the kidney lose bicarbonate or fail to excrete acid, so blood bicarbonate falls and the child develops metabolic acidosis. Here bicarbonate is normal, so neither distal RTA nor proximal RTA fits. This rules out options (A) and (D).

Step 3: Rule out vitamin D dependent rickets.

Vitamin D dependent rickets (type 1 or type 2) comes from a defect in making or responding to active vitamin D. This drops the blood calcium, and low calcium then drives up parathyroid hormone as the body tries to correct it. Here calcium is normal and PTH is normal, so this pattern does not match option (C).

Step 4: Match the pattern to hypophosphatemic rickets.
Hypophosphatemic rickets (the X-linked form is the most common type) comes from excess loss of phosphate through the kidney. The classic picture is normal calcium, low phosphate, normal PTH, normal acid-base status, and a high alkaline phosphatase from ongoing rickets activity. This fits the child exactly.

Final Answer:
The labs point to a phosphate-wasting disorder with normal calcium and PTH, which 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.

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: Recall the common neurological complications of meningitis.
Inflammation of the meninges can spread to nearby brain tissue and vessels. The complications seen often are seizures, raised intracranial pressure from brain swelling, subdural effusions or empyema (mainly in infants), hydrocephalus, cranial nerve palsies, and venous sinus thrombosis.

Step 2: Check seizures.
Seizures are a well known complication of bacterial meningitis, caused by cortical irritation, fever, electrolyte shifts such as low sodium, or vascular injury. This is a true complication, so option (A) is not the answer.

Step 3: Check increased intracranial pressure.
Meningeal inflammation causes cerebral edema and blocks normal cerebrospinal fluid flow, which raises intracranial pressure. This is one of the most feared complications and a true one, so option (B) is not the answer.

Step 4: Check subdural effusions.
Subdural effusions are common in infants with bacterial meningitis, especially with Haemophilus influenzae or pneumococcus, from inflamed bridging veins leaking fluid into the subdural space. This is a true complication, so option (D) is not the answer.

Step 5: Check cerebral hematoma.
A cerebral hematoma is a collection of blood within the brain tissue, typically from trauma, a ruptured aneurysm, or a bleeding disorder. Meningitis itself does not directly cause bleeding into the brain substance. It is not listed as a standard complication of meningitis, so this is the one that does not belong.

Final Answer:
Cerebral hematoma is not a recognized neurological complication of meningitis.

Cerebral Hematoma

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

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: Understand what "definitive diagnosis" means here.
Extrahepatic biliary atresia is a blockage or absence of the bile ducts outside the liver in a newborn. A screening test can raise suspicion, but the definitive test must directly show whether bile can pass out of the liver into the intestine.

Step 2: Check hepatobiliary scintigraphy.
This nuclear scan (HIDA scan) shows good uptake by liver cells but

no tracer reaching the bowel in biliary atresia. It is a good screening tool with high sensitivity, but it also gives false positives in severe neonatal hepatitis with heavy cholestasis, where bile also fails to reach the bowel. So it does not confirm the diagnosis on its own.

Step 3: Check alkaline phosphatase and liver biopsy.
Alkaline phosphatase is a nonspecific marker raised in many causes of cholestasis, so it cannot confirm biliary atresia by itself. Liver biopsy is the best single preoperative test, with high accuracy, but it is still a prediction based on tissue changes such as bile duct proliferation and portal fibrosis, not direct proof of an absent or blocked duct.

Step 4: Identify the true gold standard.
Per-operative cholangiography involves injecting contrast dye directly into the gallbladder or bile duct during surgery and taking an X-ray. This directly shows whether the biliary tree is patent or blocked, which is why it remains the gold standard confirmatory test for extrahepatic biliary atresia.

Note on the answer key:
The original 2002 key marks hepatobiliary scintigraphy as correct, but current teaching (Nelson's Textbook of Pediatrics and standard pediatric surgery texts) is clear that per-operative cholangiography is the true gold standard, since scintigraphy only screens and cannot reliably tell biliary atresia apart from severe neonatal hepatitis. This answer has been updated to reflect current medical knowledge.

Final Answer:

Per-operative Cholangiography

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

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: Recall how feeding tube length is measured in infants.
Two landmarks are commonly used together to size a feeding tube: the tip of the nose, the ear lobe, and either the xiphoid process (for gastric placement) or a point past the stomach (for tubes meant to sit beyond the pylorus).

Step 2: Understand the difference between gastric and transpyloric placement.
A gastric tube only needs to reach the stomach, so it is measured nose to ear to xiphoid, often called the NEX method. A transpyloric tube has to travel further, past the stomach and pylorus into the duodenum or jejunum, so a longer measurement is needed.

Step 3: Identify the correct landmark for transpyloric length.
For transpyloric feeding, the extra length is estimated using the

umbilicus as the distal landmark, giving the nose to ear to mid-umbilicus measurement, sometimes called NEMU. This adds enough length for the tube tip to sit past the pylorus.

Step 4: Rule out the other options.
Ear lobe to umbilicus alone leaves out the nose, which is part of the standard landmark set, so it undercounts the true path length. Using the knee joint as a landmark is not part of any standard feeding tube sizing method for infants, so options (C) and (D) do not fit.

Final Answer:
The transpyloric tube length is measured from the nose to the umbilicus (via the ear).

Nose to Umbilicus

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

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: Recall the layers of the epidermis.
From deep to superficial, the epidermis has the stratum germinativum (basal layer), stratum spinosum, stratum granulosum, stratum lucidum (only in thick skin), and the outermost stratum corneum.

Step 2: Understand skin maturity in a VLBW infant.
A very low birth weight infant is often born well before term, so the skin has not finished maturing. The layer that matures last, closest to birth, is the outer barrier layer.

Step 3: Identify the barrier layer.
The stratum corneum is the tough, keratin-rich outer layer that normally forms a water-tight barrier and takes shape mainly in the last weeks of pregnancy. In a preterm VLBW infant this layer is thin and poorly formed in the first week of life.

Step 4: Explain why this matters clinically.
Because the stratum corneum is underdeveloped, VLBW infants lose water rapidly through the skin (high transepidermal water loss), absorb topical substances more easily, and face a higher risk of skin infection and heat loss in the first week.

Step 5: Rule out the other layers.
The stratum germinativum is the actively dividing basal layer and is present from early fetal life. The stratum granulosum and stratum lucidum are inner layers that are also present, just thinner, but it is the outer corneum whose immaturity causes the well known clinical problems of preterm skin.

Final Answer:

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: Recall the purpose of intraosseous access.
Intraosseous (IO) access places a needle into the marrow cavity of a bone, usually the proximal tibia, to give drugs and fluids quickly when a vein cannot be found fast in an emergency.

Step 2: Recall the older teaching on age limits.
Older pediatric resuscitation teaching, closer to when this paper was written, often restricted IO access to younger children, since the marrow cavity was thought to be easier to reach and more useful before the bones became harder with age.

Step 3: Apply current resuscitation guidelines.
Current pediatric advanced life support guidance from the American

Heart Association states that IO access is an acceptable route in patients of any age, including older children and adults, whenever intravenous access cannot be obtained quickly, generally within about 2 attempts or 90 seconds in an emergency. Modern IO devices and insertion sites (proximal tibia, distal femur, proximal humerus, and sternum in adults) work across the whole age range.

Step 4: Explain why the age-limited options are outdated.
Restricting IO access to children less than 1, 5, or 6 years old reflects older practice, not current guidelines. Present-day teaching removes the age ceiling entirely for emergency IO use.

Note on the answer key:
The original 2002 key marks "less than 6 years" as correct, matching the teaching of that time. Current PALS guidance has since dropped the age restriction, so this answer has been updated to "any age" to reflect present-day practice.

Final Answer:

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.

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: Read the clinical picture carefully.
The child is gasping and unresponsive. Gasping is not effective breathing, it is a sign that the child is not moving enough air to keep oxygen and carbon dioxide levels normal. An unresponsive child with gasping breaths is in respiratory failure that is about to become respiratory arrest.

Step 2: Apply the basic emergency approach.
In any child who is not breathing effectively, the first priority is to support ventilation, not just to raise the oxygen percentage the child breathes in on their own. A child who is gasping cannot draw in enough of any oxygen concentration by themselves.

Step 3: Compare bag and mask ventilation with oxygen by mask.
Giving 100% oxygen through a simple face mask still depends on the child making their own breathing effort to pull that oxygen into the lungs. Since this child's own effort (gasping) is not enough, oxygen by mask alone will not fix the problem. Bag and mask ventilation actively pushes air and oxygen into the lungs, taking over the work of breathing that the child can no longer do adequately.

Step 4: Explain why the other options are not first.

Intubation may be needed soon after, but placing a tube takes time to prepare, bagging keeps the child oxygenated while the team gets ready for it. Nebulized salbutamol treats the underlying bronchospasm from asthma, but it does nothing for a child who is not moving enough air right now to even inhale the nebulized drug properly.

Note on the answer key:
The original key marks "100% oxygen by mask" with a question mark, showing doubt even at the time. Standard resuscitation teaching for a gasping, unresponsive child is to start assisted ventilation right away with bag and mask, so the answer has been updated to reflect this.

Final Answer:

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:

Step 1: Understanding the Question.

The child has a mix of features: confusion (a central sign), heavy salivation and lacrimation, muscle fasciculations, small pupils (miosis), and a fast heart rate with high blood pressure. We need a poison that gives this exact mixed picture.

Step 2: Key Concept.

This combination is the classic toxidrome of organophosphate poisoning. Organophosphates block the enzyme acetylcholinesterase, so acetylcholine builds up at nerve endings instead of being broken down.

The extra acetylcholine acts on two kinds of receptors at the same time:

Muscarinic receptors, which give salivation, lacrimation, sweating, urination, diarrhea, vomiting, miosis and a slow heart rate.

Nicotinic receptors, which give muscle fasciculations, weakness, and a faster heart rate with raised blood pressure through sympathetic ganglion stimulation.

Central acetylcholine buildup also causes confusion, restlessness and, in severe cases, seizures and coma.

Step 3: Detailed Explanation.

Opium causes sedation, pinpoint pupils and slow breathing, but it does not cause salivation, fasciculations or a fast heart rate, so it does not fit.

Dhatura contains atropine like alkaloids, which block acetylcholine action. It gives a dry mouth, dilated pupils, flushed skin, fast heart

rate and confusion, the opposite of the wet, small pupil picture here, so it does not fit.
Organochlorine pesticides mainly cause central nervous system over excitation, tremor and seizures, but not the marked salivation, miosis and fasciculations described.
Only organophosphate poisoning explains every feature together: the muscarinic signs (salivation, lacrimation, miosis), the nicotinic sign (fasciculations, tachycardia, hypertension) and the central sign (confusion).

Step 4: Final Answer.
The poison responsible is an organophosphate insecticide, which matches option (2).

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

16. The drug of choice for thoracic actinomycosis is

(A) Amphotericin B

(B) Penicillin

(C) Co trimoxazole

(D) Itraconazole

Correct Answer: (B) Penicillin

Solution:

Step 1: Understanding the Question.
Actinomycosis is a chronic infection that can involve the chest

(thoracic actinomycosis), and we are asked for its drug of choice.

Step 2: Key Concept.
Actinomycosis is caused by Actinomyces israelii, a filamentous, branching, gram positive bacterium that behaves like an anaerobe or microaerophilic organism, not a fungus, even though the name sounds fungal. It classically forms sulfur granules in tissue and spreads slowly across natural planes, causing sinus tracts.
Because Actinomyces species are reliably sensitive to penicillin, and the infection tends to be deep seated and fibrotic (poor drug penetration), high dose penicillin given for a long course, often several months, is the standard first line treatment.

Step 3: Detailed Explanation.
Amphotericin B is an antifungal drug. Since actinomycosis is bacterial, not fungal, amphotericin B has no role here.
Co trimoxazole and other agents such as tetracyclines or clindamycin are used mainly as alternatives in patients who are allergic to penicillin, not as the first choice.
Itraconazole is also an antifungal agent and is not active against Actinomyces.
Only penicillin covers Actinomyces reliably and is used at high dose for a prolonged period to clear deep thoracic disease.

Step 4: Final Answer.
The drug of choice for thoracic actinomycosis is penicillin, option (2).

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.

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:

Step 1: Understanding the Question.
We need to find the one FALSE statement among four claims about the cancer risk of different types of colorectal polyps.

Step 2: Key Concept.
Colorectal polyps are grouped mainly as neoplastic (adenomatous) polyps, which carry real malignant potential, and non-neoplastic polyps, such as hyperplastic, hamartomatous, or inflammatory (pseudo) polyps, which are largely benign growths without their own cancer risk, unless a genetic syndrome is present.

Step 3: Detailed Explanation.
Option (1), familial adenomatous polyposis: patients carry hundreds to thousands of adenomatous polyps, and without treatment nearly all of them progress to colorectal cancer by middle age. So this statement is true.
Option (2), pseudopolyps of ulcerative colitis: these are inflammatory, regenerative islands of mucosa left behind after repeated ulceration and healing in chronic colitis. They are not neoplastic tissue and do

not themselves turn malignant. The raised cancer risk in long standing ulcerative colitis comes from dysplasia developing in the flat surrounding mucosa, not from the pseudopolyps. So calling pseudopolyps high risk for malignancy is false, this is the exception we are looking for.
Option (3), villous adenoma: among adenomatous polyps, villous architecture carries the highest risk of harboring cancer compared with tubular or tubulovillous types, especially in larger polyps. So this statement is true.
Option (4), juvenile polyps: these are hamartomatous polyps typically seen in children, and isolated sporadic juvenile polyps carry little or no malignant potential (the risk rises only in juvenile polyposis syndrome with multiple polyps). So this statement is true.

Step 4: Final Answer.
The false statement is that pseudopolyps of ulcerative colitis carry a high risk of malignancy, option (2).

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

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:

Step 1: Understanding the Question.

The patient has a lung and kidney combination, cough with coughing up blood plus glomerulonephritis, and the key laboratory clue is a raised c-ANCA. We must pick the disease this antibody pattern points to.

Step 2: Key Concept.

c-ANCA is directed against proteinase 3 (PR3) inside neutrophils and is strongly linked with Wegener's granulomatosis, now also called granulomatosis with polyangiitis (GPA). This disease is a small vessel vasculitis that classically involves the upper airway, lungs and kidneys together, giving a triad of sinus or nasal disease, pulmonary nodules or hemorrhage, and rapidly progressive glomerulonephritis.

Step 3: Detailed Explanation.

Goodpasture's syndrome also causes lung hemorrhage plus glomerulonephritis, but it is driven by anti glomerular basement membrane antibodies, not ANCA, so a raised c-ANCA argues against it.

Classic polyarteritis nodosa is a medium vessel vasculitis that spares the lungs and does not cause glomerulonephritis (it affects renal arteries, causing infarcts and hypertension rather than glomerular

inflammation), and it is typically ANCA negative.
Kawasaki syndrome is a vasculitis of childhood that mainly affects the coronary arteries and presents with fever, rash, conjunctivitis and mucosal changes, not this lung-kidney, ANCA positive picture.
Only Wegener's granulomatosis fits both the clinical lung plus kidney picture and the c-ANCA positivity described.

Step 4: Final Answer.
The most likely diagnosis is Wegener's granulomatosis (granulomatosis with polyangiitis), option (3).

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.

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:

Step 1: Understanding the Question.
We need the gene family whose normal job is to control body shape and organ patterning as the embryo develops, that is, morphogenesis.

Step 2: Key Concept.

Homeobox genes, also called Hox genes, code for transcription factors that switch on the right combination of downstream genes in the right body segment at the right time. They set up the head to tail (anteroposterior) body plan and guide limb and organ patterning, so they directly regulate normal morphogenesis.

Step 3: Detailed Explanation.

FMR-1 is the familial mental retardation gene. Its abnormal expansion causes fragile X syndrome, which affects neuronal function and intellectual development, not general body patterning.

P-16 (CDKN2A) is a tumor suppressor gene that slows the cell cycle by inhibiting cyclin dependent kinases, protecting against cancer, not a morphogenesis regulator.

PTEN is also a tumor suppressor, a phosphatase that opposes the PI3K-AKT growth pathway, and its loss is linked with cancers of the breast, prostate and other organs, again a growth control gene rather than a body-plan gene.

Only homeobox genes are the master regulators of segmental body patterning during embryogenesis, and mutations in them classically cause patterning defects such as syndactyly or polydactyly.

Step 4: Final Answer.

The gene regulating normal morphogenesis during development is the homeobox gene, option (2).

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:

Step 1: Understanding the Question.
We must pick the one familial syndrome among the four that is NOT linked with pheochromocytoma, a tumor of the adrenal medulla that secretes catecholamines.

Step 2: Key Concept.
The well established familial pheochromocytoma-associated conditions include multiple endocrine neoplasia type 2 (MEN 2A and 2B), von Hippel-Lindau disease, and neurofibromatosis type 1 (also called von Recklinghausen disease), all of which arise from neural crest related or tumor suppressor gene defects. Sturge-Weber syndrome, a neurocutaneous disorder with a facial port wine stain and leptomeningeal vascular malformation, has occasionally been reported with catecholamine secreting tumors in the older literature this question is based on, reflecting a shared neural crest origin theory.

Step 3: Detailed Explanation.
Option (2), von Recklinghausen disease (neurofibromatosis type 1): this is one of the classic familial syndromes with a recognized, though

not extremely common, association with pheochromocytoma.
Option (3), MEN type IIb: this syndrome (medullary thyroid carcinoma, mucosal neuromas, marfanoid habitus) is one of the strongest and most consistent associations with pheochromocytoma, occurring in a large proportion of patients.
Option (1), Sturge-Weber syndrome: per the classic teaching this question is drawn from, this is grouped with the syndromes described as pheochromocytoma-associated, so within this question it is treated as a true association.
Option (4), Prader-Willi syndrome: this is an imprinting disorder causing hypotonia, obesity, hypogonadism and intellectual disability due to loss of paternal genes on chromosome 15. It has no established mechanistic or clinical link to adrenal medullary tumors at all.

Step 4: Final Answer.
The syndrome NOT associated with pheochromocytoma is Prader-Willi syndrome, option (4).

Note on this override check: the paper's stated key (option 4, Prader-Willi) was verified against current knowledge and is retained, since Prader-Willi has no recognized pheochromocytoma link while all three other listed syndromes do (or are taught to) in standard references.

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

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:

Step 1: Understanding the Question.
Lead poisoning causes a hypochromic anemia, meaning red cells are pale from low hemoglobin content, even though the marrow usually has enough iron. We need the exact mechanism behind this.

Step 2: Key Concept.
Heme is built through a multi step pathway inside developing red cells, and two of its key enzymes, delta-aminolevulinic acid dehydratase (ALA dehydratase) and ferrochelatase, are extremely sensitive to lead. Lead binds and blocks these sulfhydryl dependent enzymes, so the pathway cannot finish making heme even when iron is present.

Step 3: Detailed Explanation.
With ALA dehydratase blocked, delta-ALA piles up before the block and heme production slows down. With ferrochelatase blocked, the final step of inserting iron into protoporphyrin IX fails, so protoporphyrin accumulates in red cells (seen as raised free erythrocyte protoporphyrin, an important clue used in lead poisoning testing) while hemoglobin content falls.

The result is red cells that are small and pale (microcytic, hypochromic), often with basophilic stippling on the smear from residual ribosomal RNA, even though total body iron is not the problem.
Option (2) is incorrect because lead does not act by displacing iron off transferrin; the anemia comes from blocked heme synthesis, not blocked iron transport.
Option (3) is incorrect because lead's toxicity to red cell precursors is mainly through enzyme inhibition inside the synthetic pathway, not by damaging the outer cell membrane as the primary mechanism of this anemia.
Option (4) is incorrect because ferritin to hemosiderin conversion is not the step lead interferes with; the defect is earlier, in heme synthesis itself.

Step 4: Final Answer.
The hypochromic anemia of lead poisoning is due to inhibition of enzymes involved in heme biosynthesis, option (1).

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

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:

Step 1: Understanding the Question:
The question asks which change seen under the electron microscope tells us that a cell has crossed the point of no return and cannot recover, that is irreversible injury.

Step 2: Key Formula or Approach:
Cell injury is graded as reversible or irreversible based on structural changes. Reversible injury shows swelling of the cell and organelles, ER dilation, ribosome detachment, and chromatin clumping, all of which can return to normal if the stress is removed. Irreversible injury adds severe mitochondrial damage that cannot be undone, most classically large amorphous flocculent densities inside the mitochondrial matrix.

Step 3: Detailed Explanation:
Dilatation of the endoplasmic reticulum happens early in cell injury and is fully reversible once normal oxygen and ATP supply return.
Dissociation of ribosomes from the rough endoplasmic reticulum is also an early, reversible change, it reduces protein synthesis but the ribosomes reattach if the cell recovers.
Flocculent densities in mitochondria are large, amorphous, cottonwool like deposits that form inside a severely swollen and

damaged mitochondrion. This change appears only after the cell has lost the ability to recover and is taken as a marker of irreversible injury.
Myelin figures are whorled membrane fragments that can appear in both reversible and irreversible injury, since they simply mark membrane damage and phospholipid rearrangement, so they do not by themselves point to irreversible injury.

Step 4: Final Answer:
The finding that specifically marks irreversible cell injury is flocculent densities in the mitochondria.

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

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:

Step 1: Understand the Fenton reaction.
The Fenton reaction is a chemical reaction in which hydrogen peroxide reacts with a transition metal, usually iron, to make a highly reactive hydroxyl free radical. It is one of the main ways the body

generates dangerous free radicals from hydrogen peroxide.

Step 2: Write the reaction.
The reaction is

$$Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + OH^- + OH^\bullet$$

Here the ferrous ion (Fe^{2+}) donates an electron to hydrogen peroxide, breaking it apart and forming the hydroxyl radical ($OH^\bullet$). In the process the ferrous ion is oxidised to the ferric ion (Fe^{3+}).

Step 3: Check each option against this reaction.
Radiant energy absorbed by water can split water into radicals through radiolysis, but this is not the Fenton reaction, it is a separate, radiation-driven process.
Hydrogen peroxide being formed by myeloperoxidase describes how the enzyme system makes reactive oxygen species inside phagocytes, it is not the Fenton step itself, which needs a metal ion, not an enzyme.
Nitric oxide turning into peroxynitrite anion is a different free radical pathway, where nitric oxide combines with superoxide, again this does not use iron and is not Fenton chemistry.
The conversion of ferrous ions into ferric ions is exactly the electron transfer step that defines the Fenton reaction and directly produces the hydroxyl radical.

Step 4: Final answer.
Free radical generation by the Fenton reaction happens when ferrous ions are converted into ferric ions.

Ferrous ions are converted into ferric ions

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

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:

Step 1: Understanding the Question:
Familial amyloidotic polyneuropathy is an inherited disease where amyloid deposits build up in peripheral nerves. We need to identify which protein forms these deposits.

Step 2: Key Formula or Approach:
Amyloid disease is named by the precursor protein that misfolds and deposits as amyloid fibrils. AA amyloidosis comes from serum amyloid associated protein, AL amyloidosis comes from immunoglobulin light chains, and ATTR amyloidosis comes from transthyretin, a transport protein made in the liver that normally carries thyroxine and retinol.

Step 3: Detailed Explanation:
In the inherited form, a point mutation in the transthyretin gene

changes the protein's shape so it misfolds easily and deposits as amyloid fibrils in peripheral nerves, this is called familial amyloidotic polyneuropathy or hereditary ATTR amyloidosis.
Amyloid associated protein forms AA amyloidosis, which is linked to chronic inflammatory disease and mainly deposits in the kidney, liver, and spleen, not primarily in nerves as an inherited disease.
Mutant calcitonin is not a recognised amyloid precursor, calcitonin itself is not implicated in this disease, though calcitonin levels are checked in medullary thyroid cancer for an unrelated reason.
Normal, unmutated transthyretin can deposit as amyloid too, but that happens in old age as senile systemic amyloidosis, mainly affecting the heart, it is not the familial nerve disease, which specifically needs a mutant form of the protein.

Step 4: Final Answer:
Familial amyloidotic polyneuropathy is caused by deposition of 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:

Step 1: Recall the two patterns of splenic amyloidosis.
Amyloid deposits in the spleen in one of two patterns, and pathology gives each a separate name based on where the deposits sit.

Step 2: Describe sago spleen.
When amyloid deposits are limited to the splenic follicles, that is the white pulp, the follicles look like firm, glassy granules scattered through the spleen, resembling grains of sago, so this pattern is called sago spleen.

Step 3: Describe lardaceous spleen.
When amyloid instead deposits along the walls of the splenic sinusoids and the surrounding connective tissue framework in the red pulp, the deposits can fuse together into large sheets. The cut surface of the spleen then looks pale, waxy, and firm like cured pork fat, which is called lard, giving this pattern the name lardaceous spleen.

Step 4: Match the pattern to the option.
Since lardaceous spleen comes from amyloid in the sinusoids of the red pulp, and not the white pulp follicles, penicillary arteries, or the trabeculae, the correct site is the sinusoids of the red pulp.

Step 5: Final answer.
Lardaceous spleen results from amyloid deposition in the sinusoids of the red pulp.

Sinusoids of red pulp

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

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:

Step 1: Understanding the Question:
The clinical picture is a 42 year old man with fever, weakness, and bleeding gums, pancytopenia on smear, and a bone marrow with 26 percent blasts, frequent Auer rods, mature myeloid cells, and a neutrophil with the Pelger-Huet anomaly. We must decide which cytochemical stain will most likely be positive in this marrow.

Step 2: Key Formula or Approach:
Blasts above 20 percent with Auer rods point to a myeloid blast population, which by current WHO criteria falls under acute myeloid leukemia with myelodysplasia related changes rather than the older

category of refractory anemia with excess blasts in transformation. Cytochemical stains help confirm the lineage of blasts: myeloperoxidase and Sudan Black B stain myeloid blasts and are strongly positive wherever Auer rods are seen, since Auer rods themselves are made of fused primary granules rich in myeloperoxidase. Nonspecific esterase marks monocytic blasts, acid phosphatase is used mainly for hairy cell leukemia and some T cell lineage markers, and toluidine blue stains mast cell and basophil granules.

Step 3: Detailed Explanation:
Auer rods are needle shaped, crystalline structures made of condensed primary granules, and they are strongly myeloperoxidase positive, this is in fact the classic teaching point, the presence of Auer rods is direct evidence of myeloid differentiation and predicts a positive myeloperoxidase stain.
The mature myeloid cells and the Pelger-Huet neutrophil in the marrow also support a myeloid lineage process, not a lymphoid or monocytic one.
Acid phosphatase would be relevant if we suspected hairy cell leukemia or a T lymphoblastic process, neither fits this myeloid picture.
Nonspecific esterase would be positive in monocytic or myelomonocytic leukemia, but there is no mention of monocytic morphology here, and the Auer rods point away from a purely monocytic process.
Toluidine blue stains basophilic or mast cell granules and is not relevant to a myeloblastic marrow with Auer rods.
Note on the source key: the original paper's explanation only describes the disease as RAEB-T without stating an explicit answer letter. Based on the marrow findings, particularly the Auer rods, the

cytochemical stain most likely to be positive is myeloperoxidase, so that is the answer used here.

Step 4: Final Answer:
The cytochemical stain most likely to be positive in this myeloblastic marrow with Auer rods is myeloperoxidase.

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

27. Dystrophic calcification is seen in:

(A) Rickets

(B) Hyperparathyroidism

(C) Atheromatous plaque

(D) Vitamin A intoxication

Correct Answer: (C) Atheromatous plaque

Solution:

Step 1: Understanding the Question:
We need to identify which of the given conditions shows dystrophic calcification, that is calcium deposition in damaged or dead tissue while blood calcium levels stay normal.

Step 2: Key Formula or Approach:
Calcification is split into two types. Dystrophic calcification happens in areas of cell injury, necrosis, or degeneration, with normal serum calcium, common examples are atheromatous plaques, caseous

tuberculous necrosis, fat necrosis, and damaged heart valves.
Metastatic calcification happens in normal tissue because the blood calcium or phosphate level is raised, common causes are hyperparathyroidism, vitamin D or vitamin A excess, and conditions like rickets related bone and mineral disturbances.

Step 3: Detailed Explanation:
Rickets is a disorder of bone mineralisation from vitamin D deficiency, when it is treated or when there is an associated disturbance, it is linked with abnormal calcium and phosphate handling, and calcification here follows the metastatic pattern of raised mineral levels, not local tissue death.
Hyperparathyroidism raises blood calcium directly by increasing bone resorption, and this high calcium can deposit in normal tissues such as kidney and lung, which is metastatic calcification.
Atheromatous plaque contains areas of lipid deposition, cell death, and degeneration in the arterial wall, calcium salts deposit locally in this damaged tissue while the blood calcium level stays normal, this is the classic example of dystrophic calcification.
Vitamin A intoxication can raise blood calcium and contribute to metastatic calcification, it is not an example of local dystrophic change.

Step 4: Final Answer:
Dystrophic calcification, calcification in locally damaged tissue with a normal blood calcium level, is seen in atheromatous plaque.

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

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:

Step 1: Recall what each storage disease accumulates.
Lysosomal storage diseases result from a missing or faulty enzyme, so a specific lipid builds up inside cells, mainly inside phagocytic cells such as macrophages.

Step 2: Identify the enzyme defect linked to sphingomyelin.
Niemann-Pick disease, types A and B, comes from a deficiency of the enzyme sphingomyelinase, which normally breaks down sphingomyelin. Without this enzyme, sphingomyelin builds up inside lysosomes of macrophages and other cells, these lipid filled macrophages are called foam cells.

Step 3: Rule out the other diseases.
Gaucher's disease is caused by a deficiency of glucocerebrosidase, and the stored lipid is glucocerebroside, not sphingomyelin, and its macrophages are called Gaucher cells, with a crumpled tissue paper appearance.
Tay-Sachs disease is caused by a deficiency of hexosaminidase A, and the stored lipid is GM2 ganglioside, mainly in neurons, not sphingomyelin.
Down's syndrome is a chromosomal disorder from trisomy 21, it is

not a lysosomal storage disease and has no link to sphingomyelin accumulation.

Step 4: Final answer.
Accumulation of sphingomyelin in phagocytic cells is a feature of Niemann-Pick disease.

Niemann-Pick Disease

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

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:

Step 1: Recall what an Aschoff body is.
An Aschoff body (or Aschoff nodule) is the classic microscopic marker of active rheumatic carditis. It forms in the myocardium, most often around small blood vessels, during acute rheumatic fever.

Step 2: List the parts that make up an Aschoff body.

Each nodule has a core of swollen, degenerate collagen showing fibrinoid necrosis. Around this core sit plump activated macrophages called Anitschkow cells, which have a wavy, caterpillar shaped chromatin strand and are considered pathognomonic for rheumatic fever. Some of these macrophages fuse together to form multinucleated Aschoff giant cells. Lymphocytes and a few plasma cells surround the whole nodule.

Step 3: Check option 1, Anitschkow cells.
These are a core, defining component of the Aschoff body, so this feature IS seen. It is not the exception.

Step 4: Check option 3, Giant cells.
The multinucleated Aschoff giant cells form from fused Anitschkow macrophages and are a standard, well documented part of the lesion. This feature IS seen, so it is not the exception either.

Step 5: Check option 4, Fibrinoid necrosis.
The central zone of degenerate, eosinophilic collagen is fibrinoid necrosis, and it forms the core the other cells gather around. This feature IS seen.

Step 6: Check option 2, Epithelioid cells.
Epithelioid cells are macrophages that have taken on an elongated, pink, epithelium like shape, and they are the hallmark of true granulomas such as those in tuberculosis or sarcoidosis. Standard descriptions of the Aschoff body do not list epithelioid cells or granuloma formation as one of its components; the macrophages here are Anitschkow cells and Aschoff giant cells, not epithelioid cells. So this is the feature that is NOT seen.

Step 7: Final answer.

Since Anitschkow cells, giant cells and fibrinoid necrosis are all genuine parts of the Aschoff body, and epithelioid cells are not, the correct exception is epithelioid cells.

Epithelioid cells

Note on the source answer key:

The original printed key for this paper marks "Giant cells" as the answer. Aschoff giant cells are a textbook-confirmed component of the Aschoff body, so that option cannot be the exception. This entry has been corrected to Epithelioid cells, which is the option with no place in the standard description of an Aschoff body.

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

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: Recall what asbestosis is.
Asbestosis is a chronic pneumoconiosis caused by long-term inhalation of asbestos fibers, most often in workers exposed during mining, insulation work or shipbuilding. It mainly damages the lower lobes of the lungs.

Step 2: List the classic findings of asbestos-related lung disease.
Inhaled asbestos fibers reach the small airways and alveoli, where they trigger chronic inflammation and progressive scarring. This produces diffuse interstitial fibrosis, worst in the lower and subpleural zones. The pleura itself reacts by forming fibrous pleural thickening and, very often, discrete calcific pleural plaques on the parietal pleura, which are considered a hallmark of past asbestos exposure.

Step 3: Check option 1, diffuse pulmonary interstitial fibrosis.
This is the defining lesion of asbestosis itself, so it IS a feature.

Step 4: Check option 2, fibrous pleural thickening.
Diffuse fibrous thickening of the pleura is a well recognised finding after heavy asbestos exposure, so it IS a feature.

Step 5: Check option 4, calcific pleural plaques.
Calcified plaques on the parietal pleura, often seen on chest X-ray as holly leaf shaped densities, are one of the most specific markers of asbestos exposure. This IS a feature.

Step 6: Check option 3, emphysema.
Emphysema is destruction of alveolar walls with permanent air space enlargement, and it is linked to smoking and alpha-1 antitrypsin deficiency, not to asbestos fiber deposition. Asbestosis causes fibrosis

and scarring, which is the opposite pattern to the airspace destruction of emphysema. Emphysema is not part of the standard description of asbestosis.

Step 7: Final answer.
The three genuine features of asbestosis are interstitial fibrosis, pleural thickening and calcific pleural plaques. Emphysema is not one of them.

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.

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:

Step 1: Recall the main drugs used in parkinsonism.
The major drug classes are levodopa (with carbidopa), dopamine

agonists, amantadine, and antimuscarinic (anticholinergic) agents. Each acts differently and has its own side effect profile.

Step 2: Check option 1, amantadine and ankle oedema.
Amantadine is known to cause peripheral oedema, most often around the ankles, along with livedo reticularis, a mottled purplish skin discoloration on the legs. This is a well documented adverse effect. This statement IS true.

Step 3: Check option 2, levodopa and tremor.
Levodopa works best against bradykinesia (slowness of movement) and rigidity. Its effect on tremor is present but usually the weakest and slowest of the three cardinal signs to respond, tremor often improving last, if at all. So calling levodopa "particularly effective" for tremor is incorrect.

Step 4: Check option 3, amantadine versus levodopa.
Amantadine has a faster onset of action than levodopa but is a much weaker drug overall, used mainly for mild symptoms or as an add-on. Levodopa remains the most effective drug for parkinsonism. So amantadine being "more effective" is incorrect.

Step 5: Check option 4, antimuscarinics in drug-induced parkinsonism.
Antimuscarinics do help in drug-induced parkinsonism, but only the centrally acting ones (drugs that cross into the brain, such as trihexyphenidyl), because the problem there is an imbalance between dopamine and acetylcholine in the brain, not in peripheral tissue. The statement as worded, calling all antimuscarinics effective, is too broad and therefore not strictly correct.

Step 6: Final answer.

Only the statement about amantadine causing ankle oedema is fully correct as written.

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.

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 PGF2alpha cause vasoconstriction.

(B) PGE1 and PGI2 inhibit platelet aggregation, whereas TXA2 facilitates aggregation.

(C) PGE2 has marked oxytocic action while PGF2alpha has tocolytic action.

(D) PGE2 is a bronchodilator whereas PGF2alpha is a bronchoconstrictor.

Correct Answer: (C) PGE2 has marked oxytocic action while PGF2alpha has tocolytic action.

Solution:

Step 1: Recall the main prostaglandins and their receptors.
Prostaglandins (PGE2, PGI2, PGF2alpha) and thromboxane (TXA2) are eicosanoids derived from arachidonic acid. They act through different G-protein coupled receptors on smooth muscle, platelets and other tissues, so their effects can differ sharply even though they come from the same pathway.

Step 2: Check option 1, vascular smooth muscle.
PGE2 and PGI2 (prostacyclin) act on receptors that raise cyclic AMP in vascular smooth muscle, causing relaxation and vasodilation. TXA2 and PGF2alpha act on a different receptor that raises intracellular calcium, causing vasoconstriction. This statement matches standard pharmacology, so it IS correct.

Step 3: Check option 2, platelet aggregation.
PGI2 (and to a lesser extent PGE1) raises platelet cyclic AMP, which blocks aggregation, giving prostacyclin its anti-thrombotic role. TXA2 does the opposite, lowering cyclic AMP and driving platelets to aggregate and vessels to constrict, which is why aspirin's anti-platelet effect works by blocking TXA2 synthesis. This statement IS correct.

Step 4: Check option 4, bronchial smooth muscle.
PGE2 relaxes bronchial smooth muscle and is a bronchodilator, while PGF2alpha contracts it and is a bronchoconstrictor, which is why PGF2alpha analogues are avoided in asthmatics. This statement IS correct.

Step 5: Check option 3, action on the uterus.
Both PGE2 and PGF2alpha contract uterine smooth muscle and are oxytocic (uterotonic); this is exactly why both are used clinically to induce labour or terminate pregnancy (for example, PGF2alpha

analogues like carboprost are used for postpartum hemorrhage and abortion). Neither of them is tocolytic; a tocolytic drug is one that relaxes the uterus, such as ritodrine or nifedipine. So calling PGF2alpha "tocolytic" is wrong; it should be oxytocic like PGE2, not its opposite.

Step 6: Final answer.
The statement that is NOT correct is the one about PGE2 having oxytocic action while PGF2alpha has tocolytic action, because PGF2alpha is oxytocic too, not tocolytic.

PGE2 has marked oxytocic action while PGF2alpha has tocolytic action

Quick Tip: Both PGE2 and PGF2alpha 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:

Step 1: Recall how lithium is handled by the kidney.
Lithium is filtered freely at the glomerulus and then reabsorbed mainly in the proximal convoluted tubule, alongside sodium and water, using the same transporters. A smaller amount is also reabsorbed later in the collecting duct through sodium channels.

Step 2: Think about how thiazide diuretics change this.
Thiazide diuretics such as hydrochlorothiazide block sodium reabsorption in the distal convoluted tubule. This causes mild volume depletion, and the kidney responds by increasing sodium (and, along with it, lithium) reabsorption in the proximal tubule to conserve fluid. Since more lithium is pulled back into the blood, its renal clearance falls and its blood level can rise toward toxic levels. This drug-lithium interaction is one of the most important and frequently tested in pharmacology.

Step 3: Check the other diuretics.
Acetazolamide acts on the proximal tubule directly by blocking carbonic anhydrase; it actually increases lithium excretion rather than decreasing it. Furosemide, a loop diuretic, acts on the thick ascending limb of the loop of Henle; because its natriuretic mechanism is different from a thiazide's, it produces much less compensatory proximal reabsorption, so it has only a weak, clinically less significant effect on lithium clearance and is generally considered safer to combine with lithium than a thiazide. Spironolactone works on the collecting duct by blocking aldosterone; it is not the classic drug linked to lithium toxicity.

Step 4: Final answer.
Hydrochlorothiazide is the diuretic that most significantly decreases

renal lithium clearance and raises serum lithium levels, making regular monitoring necessary when the two are combined.

Hydrochlorothiazide

Note on the source answer key:
The original printed key for this paper marks furosemide as the answer. The well documented pharmacology, and the reason lithium levels are monitored closely in practice, points to thiazide diuretics, not loop diuretics, as the ones that reduce lithium clearance. This entry has been corrected to hydrochlorothiazide.

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.

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:

Step 1: Recall what makes Mycoplasma unique among bacteria.
Mycoplasma are the smallest free-living organisms capable of self-replication. Their single defining feature is that they completely lack a rigid cell wall, unlike almost all other bacteria.

Step 2: Check option 1, binary fission.
Like other bacteria, Mycoplasma reproduce by binary fission, splitting one cell into two. This IS true.

Step 3: Check option 3, cell-free culture media.
Mycoplasma can be grown on special cell-free artificial media, such as PPLO (pleuropneumonia-like organism) agar enriched with serum, unlike obligate intracellular organisms such as Chlamydia and viruses. This IS true.

Step 4: Check option 4, requirement for sterols.
Because Mycoplasma has no cell wall, its plasma membrane must be reinforced with cholesterol (a sterol) for stability, so growth media for Mycoplasma must supply sterols, usually from added serum. This IS true.

Step 5: Check option 2, sensitivity to beta-lactams.
Beta-lactam antibiotics (penicillins, cephalosporins) work by blocking the enzymes that build peptidoglycan, the material that makes up a bacterial cell wall. Since Mycoplasma has no cell wall at all, there is no peptidoglycan for these drugs to attack, so Mycoplasma is inherently resistant to the entire beta-lactam group. Effective drugs against Mycoplasma instead target protein or DNA synthesis, such as macrolides, tetracyclines or fluoroquinolones. This statement is FALSE, making it the exception.

Step 6: Final answer.
The statement that is not true for Mycoplasma is that it is sensitive to beta-lactam antibiotics.

Are sensitive to the beta-lactam group of antibiotics

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

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:

Step 1: Recall the arachidonic acid pathway.
Arachidonic acid released from cell membranes is metabolised through two main routes: the cyclooxygenase (COX) pathway, which makes prostaglandins and thromboxane, and the lipoxygenase pathway. The 5-lipoxygenase (5-LOX) enzyme specifically converts arachidonic acid into leukotrienes.

Step 2: Recall what leukotrienes do.
Leukotrienes made through 5-LOX, particularly LTC4, LTD4 and

LTE4 (together called the cysteinyl leukotrienes, or the old name "slow reacting substance of anaphylaxis"), cause strong and prolonged bronchoconstriction, increase mucus secretion, and raise vascular permeability in the airway. LTB4 additionally attracts and activates neutrophils, adding to airway inflammation.

Step 3: Connect this to bronchial asthma.
In asthma, these leukotrienes are major drivers of bronchospasm and airway inflammation. Blocking 5-LOX with a drug such as zileuton stops leukotriene production at its source, and leukotriene receptor blockers such as montelukast work on the same pathway further downstream. Both approaches reduce bronchoconstriction and are used as add-on therapy in asthma. So inhibition of 5-LOX IS useful here.

Step 4: Check the other options.
Cardiac failure is managed with drugs like diuretics, ACE inhibitors and beta blockers; the leukotriene pathway is not a standard target here. Hepatic failure is managed by treating the underlying cause and supportive care, again not through 5-LOX blockade. Arthritis (osteoarthritis and rheumatoid arthritis) is treated mainly through COX inhibition (NSAIDs) and disease-modifying drugs; 5-LOX inhibitors are not first-line or established therapy for arthritis.

Step 5: Final answer.
5-lipoxygenase inhibition is specifically useful in bronchial asthma, where it blocks the leukotrienes responsible for bronchoconstriction and airway inflammation.

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: Know what controls the speed of induction.
How fast a patient goes to sleep with an inhaled agent depends mainly on the blood:gas partition coefficient. A low coefficient means the drug does not dissolve much in blood, so the alveolar and brain levels rise fast and induction is quick.

Step 2: Compare the coefficients of the four drugs.
Halothane has a coefficient of about 2.4, the highest of the group, so it works slowly. Isoflurane is about 1.4, faster than halothane but still slow. Sevoflurane is about 0.65 to 0.69. Desflurane is the lowest, about 0.42, which on paper makes it the fastest of all.

Step 3: Check why desflurane is not used for induction.

Desflurane is pungent and irritates the airway. Breathing it in through a mask causes coughing, breath holding, and even laryngospasm, so it cannot be used to put a patient to sleep. It is only added after the airway is already secured, usually for keeping the patient asleep during surgery.

Step 4: Match sevoflurane to real mask induction.
Sevoflurane has a low blood:gas coefficient and a pleasant, non irritating smell. This combination lets it be breathed in smoothly through a mask without coughing, so the brain level climbs fast and the patient goes to sleep quickly. This is why it is the standard drug for inhalational induction, especially in children.

Step 5: Rule out isoflurane and halothane.
Both of these have higher blood:gas coefficients than sevoflurane, so they dissolve more in blood before the brain level rises. Induction with either is noticeably slower and less smooth.

Final Answer:
Among the options that can actually be used for smooth inhalational induction, sevoflurane is the fastest.

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: Split the options into their two families.
Mivacurium, atracurium, and vecuronium are all non depolarising muscle relaxants. Suxamethonium, also called succinylcholine, is the one depolarising agent in the list.

Step 2: Recall what suxamethonium does to muscle before it paralyses it.
Suxamethonium first opens the acetylcholine receptor and causes visible muscle fasciculations, small uncoordinated twitches, before flaccid paralysis sets in. This burst of muscle spindle activity sends a wave of extra afferent traffic up to the brain.

Step 3: Connect this to intracranial pressure.
The afferent barrage raises cerebral electrical activity and cerebral blood flow, which pushes up intracranial pressure for a short window after the drug is given. This is why suxamethonium is used with caution, and often with a pretreatment dose of a non depolarising agent, in patients who already have raised intracranial pressure such as head injury.

Step 4: Check the non depolarising agents.
Mivacurium, atracurium, and vecuronium block the receptor without

first triggering fasciculations. There is no afferent barrage, so none of them raise intracranial pressure the way suxamethonium does.

Final Answer:
Only the depolarising agent in the list carries this risk.

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: Recall where mu receptors sit and what they usually do.
Mu opioid receptors are spread through the brainstem, midbrain, spinal cord, and autonomic centres. Along with pain relief, their activation produces a set of classic side effects that examiners like to test.

Step 2: Walk through the true mu effects.

Miosis, pupil constriction, comes from stimulation of the Edinger-Westphal nucleus. Bradycardia comes from increased vagal tone at the brainstem cardiovascular centres. Hypothermia comes from a mu mediated reset of the hypothalamic temperature set point, so opioids lower core body temperature. All three of these are well documented mu receptor actions.

Step 3: Check the airway effect.
Opioids, morphine in particular, are known to release histamine from mast cells and can trigger bronchoconstriction and even wheeze in susceptible patients. This is the opposite of bronchodilation. There is no accepted mu receptor pathway that relaxes bronchial smooth muscle.

Step 4: Match this to the question.
The question asks for the one effect that is NOT produced by mu receptor activation. Since bronchodilation is not on the list of genuine mu effects, and if anything the opposite can occur, this is the odd one out.

Final Answer:

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.

39. Pulmonary fibrosis is a common complication with

(A) 6 mercaptopurine

(B) Vincristine

(C) Bleomycin

(D) Adriamycin

Correct Answer: (C) Bleomycin

Solution:

Step 1: Recall each drug's class and its typical toxicity.
6 mercaptopurine is an antimetabolite whose main worries are bone marrow suppression and liver toxicity. Vincristine is a vinca alkaloid best known for peripheral neuropathy from its effect on microtubules. Adriamycin, also called doxorubicin, is an anthracycline whose signature toxicity is dose related cardiomyopathy and heart failure.

Step 2: Focus on bleomycin.
Bleomycin is an antitumour antibiotic that works by causing free radical mediated DNA strand breaks. Its dose limiting toxicity is not on the bone marrow, unlike most chemotherapy drugs, but on the lungs.

Step 3: Understand why the lung is the target.
Lung tissue has low levels of the enzyme bleomycin hydrolase compared with other organs, so the drug is not broken down efficiently there. This lets reactive oxygen species build up in lung tissue and damage alveolar cells, which over time leads to interstitial pneumonitis and progressive pulmonary fibrosis.

Step 4: Note the clinical relevance.
This toxicity is cumulative dose dependent and is worsened by high

inspired oxygen, so anaesthetists are taught to avoid giving high concentration oxygen to patients who have received bleomycin.

Final Answer:
Of the four drugs, only bleomycin has pulmonary fibrosis as its defining toxicity.

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: Rule out the gases with no vascular role.
Nitrous oxide is an anaesthetic and analgesic gas, not a pulmonary vasodilator. Nitrogen is an inert gas with no signalling action in the body. Nitrogen dioxide is a toxic pollutant gas that damages lung tissue rather than treating it. None of these three lower pulmonary

artery pressure.

Step 2: Recall the biology of nitric oxide.
Nitric oxide is a naturally made signalling molecule that activates guanylate cyclase in vascular smooth muscle, raising cyclic GMP and causing relaxation of that muscle. In the lung, this relaxes the pulmonary arterioles.

Step 3: See why it works selectively on the lung.
When nitric oxide is given by inhalation, it reaches only the ventilated alveoli and the vessels right next to them. As soon as it enters the blood stream it is bound tightly by haemoglobin and inactivated, so it does not reach the systemic circulation. This gives a vasodilator effect that is local to the lung, without dropping the systemic blood pressure.

Step 4: Connect this to clinical use.
Because of this selective action, inhaled nitric oxide is used in adults and in newborns to lower a raised pulmonary artery pressure, for example in acute respiratory distress syndrome or pulmonary hypertension, while sparing the rest of the circulation.

Final Answer:

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.

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: Note that all four drugs here are local anaesthetics.
Lignocaine, bupivacaine, cocaine, and dibucaine all block sodium channels in nerve fibres, but they differ in how well they cross mucous membranes and in how dangerous they are if absorbed in a large amount.

Step 2: Check which of these are actually used topically.
Lignocaine penetrates mucosa well and is a standard topical agent for the mouth, throat, and skin. Cocaine has natural vasoconstrictor activity along with anaesthetic action, which is why it is still used topically in some ENT and ophthalmic procedures on mucosa. Dibucaine is a potent amide anaesthetic used in topical creams and ointments.

Step 3: Focus on bupivacaine.
Bupivacaine penetrates mucous membranes poorly, so it gives an unreliable topical effect. More importantly, it carries a much higher risk of cardiotoxicity than lignocaine, because it binds cardiac sodium channels more avidly and dissociates from them more slowly, which can trigger serious ventricular arrhythmias if enough drug is

absorbed.

Step 4: Put the two facts together.
Because bupivacaine both penetrates mucosa poorly and is unusually dangerous if it is absorbed systemically, its topical or surface use on mucous membranes is avoided in practice. It is reserved for infiltration and nerve block techniques where the dose and site are controlled.

Final Answer:

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.

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: Recall the main danger of succinylcholine.
Succinylcholine opens acetylcholine receptors briefly before blocking

them, and this triggers potassium to leave muscle cells and enter the blood. In a normal patient this rise is small and harmless, but in certain conditions it becomes large enough to cause dangerous hyperkalaemia and cardiac arrest.

Step 2: Explain tetanus.
Tetanus toxin causes prolonged muscle spasm and effectively immobilises muscle, which leads to extra, or extrajunctional, acetylcholine receptors spreading across the muscle membrane. Succinylcholine acting on this much larger receptor pool causes an exaggerated potassium leak, so it is avoided.

Step 3: Explain closed head injury and stroke.
Succinylcholine causes fasciculations that transiently raise cerebral blood flow and intracranial pressure, which is unsafe in a head injury where pressure may already be high. Stroke, especially with hemiparesis, also causes disuse of the weak muscle and receptor upregulation similar to other denervation states, adding a hyperkalaemia risk on top of the intracranial pressure concern. Both are treated as relative contraindications.

Step 4: Examine hepatic failure.
Succinylcholine is broken down in the plasma by pseudocholinesterase, which is made in the liver. Severe liver failure can lower pseudocholinesterase levels enough to prolong the drug's action, giving a longer than expected paralysis, but this is a dosing and duration issue, not a receptor upregulation or hyperkalaemia risk. It does not carry the same danger as the other three conditions.

Step 5: Put it together.
Tetanus, closed head injury, and stroke all carry a real risk with

succinylcholine, either through hyperkalaemia or raised intracranial pressure, so they are contraindications. Hepatic failure only prolongs the drug's effect and is not a true contraindication.

Final Answer:

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: Understanding the Question:
The question asks for the drug that is the treatment of choice for cryptococcal meningitis, a fungal infection of the brain and its covering caused by Cryptococcus neoformans.

Step 2: Key Formula or Approach:

Cryptococcal meningitis is a serious, life threatening infection seen mostly in patients with weak immunity, such as those with HIV or on long term steroids. The standard approach uses a fast acting fungicidal drug first, then switches to a lighter drug once the infection is under control.

Step 3: Detailed Explanation:
Amphotericin B is fungicidal, it kills the fungus directly by binding to ergosterol in its cell membrane and punching holes in it. Because cryptococcal meningitis can kill quickly if untreated, doctors start with Amphotericin B, often combined with flucytosine, to clear the infection fast, then move the patient to fluconazole for weeks of follow up treatment.
Fluconazole is useful for milder disease and for long term maintenance after the infection is controlled, but on its own it works too slowly to be the first choice in a sick patient.
Itraconazole is not used as first line therapy for this infection because it does not cross into the brain as well as fluconazole or amphotericin B do.
Flucytosine is never used alone, since the fungus can quickly become resistant to it. It is always paired with amphotericin B to boost the killing effect, not used by itself.

Step 4: Final Answer:
Amphotericin B remains the drug of choice to start treatment of cryptococcal meningitis, so the correct option is (D).

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

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: Understanding the Question:
The question asks why a big dose of prednisolone, a glucocorticoid, causes the lymphocyte count to fall within hours in a patient who has lymphocytic leukemia.

Step 2: Key Formula or Approach:
Glucocorticoids act on lymphocytes in two ways. Within hours they cause redistribution of lymphocytes out of the blood into lymph nodes, spleen and bone marrow, and they also trigger programmed cell death, called apoptosis, in lymphocytes. Malignant lymphocytes in lymphocytic leukemia are especially sensitive to this apoptotic effect, which is why steroids are actually part of leukemia treatment.

Step 3: Detailed Explanation:
A large dose of prednisolone switches on genes inside the lymphocyte that push the cell toward apoptosis, a controlled form of cell death. Leukemic lymphocytes carry glucocorticoid receptors in high numbers, so they die off quickly and in large numbers once exposed to the steroid. This massive apoptosis is what drops the blood

lymphocyte count within hours, well before any effect on the bone marrow could show up.
Bone marrow depression from steroids, if it happens at all, takes much longer to develop and mainly affects marrow producing other cell lines, not an acute drop in circulating lymphocytes.
Activation of cytotoxic cells would raise immune activity, not explain a fall in the lymphocyte count itself, and steroids in fact suppress cytotoxic T cell activity rather than switch it on.
Stimulation of Natural killer cell activity is also incorrect, since glucocorticoids generally dampen immune cell activity rather than stimulate it, and NK cell stimulation would not explain a drop in lymphocyte numbers.

Step 4: Final Answer:
The rapid lymphocytopenia is caused by massive apoptosis of the leukemic lymphocytes, so the correct option is (A).

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: Understanding the Question:
The question asks which substance normally inhibits the release of prolactin from the anterior pituitary gland.

Step 2: Key Formula or Approach:
Prolactin is unusual among pituitary hormones because its main control from the hypothalamus is inhibitory rather than stimulatory. The hypothalamus releases a substance called prolactin inhibiting factor, or PIF, into the portal blood supplying the pituitary, and this factor has been identified as dopamine.

Step 3: Detailed Explanation:
Dopamine travels down the tuberoinfundibular pathway from the hypothalamus to the anterior pituitary and binds to D2 receptors on lactotroph cells, the cells that make prolactin. This binding switches off prolactin gene expression and secretion. Anything that reduces dopamine, such as dopamine receptor blocking drugs, removes this brake and raises prolactin levels instead of lowering them.
Haloperidol is a dopamine receptor antagonist used in psychiatry, it blocks the very receptor dopamine uses, so it actually raises prolactin levels, one of its well known side effects, rather than lowering them.
GABA has some inhibitory effects on hormone release in general, but it is not the physiological prolactin inhibiting factor, dopamine holds that role.
Neurophysin is a carrier protein bound to oxytocin and vasopressin as they travel down the axon to the posterior pituitary, it has no role in prolactin control.

Step 4: Final Answer:

Dopamine is the physiological inhibitor of prolactin secretion, so the correct option is (D).

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

46. The production of cervical mucus is stimulated by

(A) Progesterone

(B) Estradiol

(C) Estriol

(D) Pregnenolone

Correct Answer: (B) Estradiol

Solution:

Step 1: Understanding the Question:
The question asks which hormone drives the glands of the cervix to produce mucus.

Step 2: Key Formula or Approach:
Cervical mucus changes character across the menstrual cycle under the influence of the two main ovarian hormones, estrogen and progesterone, which act in opposite directions on the cervical glands.

Step 3: Detailed Explanation:
Around the time of ovulation, rising levels of estradiol, the main and most potent estrogen produced by the growing ovarian follicle, act on the cervical glands to increase both the amount and the water content

of the mucus. This produces the thin, clear, stretchy mucus that lets sperm swim through easily around ovulation, the classic fertile type mucus.
Progesterone, which rises after ovulation from the corpus luteum, has the opposite effect, it makes the mucus thick, scanty and sticky, forming a plug that blocks sperm and bacteria from entering the uterus.
Estriol is a weaker estrogen mainly produced in large amounts during pregnancy by the placenta, it is not the main driver of cyclical cervical mucus changes.
Pregnenolone is an early steroid hormone precursor made from cholesterol, it is a building block for other steroid hormones but has no direct action on the cervical glands.

Step 4: Final Answer:
Estradiol stimulates the production of abundant, thin cervical mucus, so the correct option is (B).

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

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: Understanding the Question:

The question asks why a nerve impulse, once it has passed through a point on the axon, does not turn around and depolarize that same point again.

Step 2: Key Formula or Approach:

Right after an action potential fires at a point on the membrane, that point enters a refractory period, made of an absolute refractory period, when the sodium channels are inactivated and cannot open no matter how strong the stimulus, followed by a relative refractory period when a much stronger than normal stimulus is needed.

Step 3: Detailed Explanation:

As the action potential moves forward, the area just behind it has sodium channels sitting in the inactivated state, they need the membrane to repolarize back to resting potential before they can reset and be ready to open again. Because these channels cannot reopen so soon, that area cannot be depolarized a second time immediately, which forces the impulse to move only forward, this is what keeps conduction one directional.

Hyperpolarization does occur briefly after an action potential, but it is not the main reason backward depolarization is blocked, hyperpolarization actually makes the membrane further from threshold, while the true block comes from sodium channels being unable to open, the refractory state.

It is not self propagating is factually wrong, a nerve impulse is by definition self propagating, that is exactly how it travels along the whole length of the axon.

The condition is always orthodromic describes the normal forward

direction of conduction, it is a description of the result, not the reason, so it does not answer why the area behind stays undepolarized.

Step 4: Final Answer:
The area just behind the moving impulse is in its refractory period, so the correct option is (B).

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

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: Understanding the Question:
The question asks by how much the cerebral metabolic rate falls when brain temperature drops by 10 degree Centigrade.

Step 2: Key Formula or Approach:
Cerebral oxygen consumption, which reflects the metabolic rate of the brain, follows a temperature dependent relationship described by the

Q10 coefficient. For brain tissue this Q10 is close to 2 to 2.4, and the accepted clinical teaching is that cerebral metabolic rate falls by about 6 to 7 percent for every 1 degree Centigrade drop in temperature.

Step 3: Detailed Explanation:
If the metabolic rate falls by roughly 6 to 7 percent for each degree, then over a full 10 degree drop these small drops compound together. Working this out shows the cerebral metabolic rate ends up at roughly half of its starting value, in other words it falls by about 50 percent. This is why hypothermia is used clinically to protect the brain during surgery or after cardiac arrest, cooling the brain by 10 degree Centigrade roughly halves its oxygen demand, giving it more tolerance to periods of reduced blood flow.
A drop of 10 percent or 30 percent would be far too small to explain the strong protective effect seen with moderate hypothermia in clinical practice. A drop of 70 percent would be an overestimate for a 10 degree change.

Step 4: Final Answer:
A 10 degree Centigrade fall in temperature lowers the cerebral metabolic rate by about 50 percent, so the correct option is (C). Note: the source answer key marked this as option (B), 30 percent, with an uncertain mark next to it in the original scan. Based on the well established Q10 relationship for cerebral metabolism, about 6 to 7 percent fall per degree Centigrade, compounding to roughly 50 percent over 10 degrees, the medically accurate answer is 50 percent, option (C), and it has been corrected here.

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

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: Understanding the Question:
The question asks which hormone level, measured from blood drawn on day 20 of the cycle, confirms whether ovulation actually took place.

Step 2: Key Formula or Approach:
In a normal 28 day cycle, ovulation happens around day 14. Day 20 falls in the mid luteal phase, the phase that follows ovulation and is marked by the corpus luteum, the structure left behind after the egg is released, actively secreting progesterone.

Step 3: Detailed Explanation:
Progesterone is secreted almost only by the corpus luteum, so a clearly raised serum progesterone in the mid luteal phase, around day 20 to 22 of a 28 day cycle, is direct proof that a follicle ruptured and released an egg, forming a functioning corpus luteum. If the cycle was anovulatory, no corpus luteum forms and progesterone stays low

throughout, this is exactly why a mid luteal progesterone test is the standard clinical way to confirm ovulation.
FSH does rise before ovulation to drive follicle growth, but by day 20 it has already settled to a low, non specific level and does not by itself prove that ovulation occurred.
LH shows a sharp surge just before ovulation, around day 13 to 14, but by day 20 this surge is long over, so a day 20 LH level does not reliably confirm that ovulation actually happened.
Oestradiol peaks just before the LH surge to trigger ovulation, but its luteal phase level is a less reliable single marker of ovulation compared to progesterone, since oestradiol is produced by growing follicles in general, not only by a functioning corpus luteum.

Step 4: Final Answer:
A mid luteal serum progesterone level, drawn on day 20, is the valid way to confirm that a cycle was ovulatory, so the correct option is (D).

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

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:

Step 1: Understanding the Question:

The question asks which finding is NOT typical when the right middle cerebral artery (MCA) territory is infarcted. The right MCA supplies the right cerebral hemisphere, which in almost all right handed people, and most left handed people too, is the non dominant hemisphere for language.

Step 2: Key Concept:

A right MCA stroke damages the right motor cortex, right face area, and right corticobulbar fibers. Since the right hemisphere is not the language hemisphere in most people, language function stays largely intact. Aphasia is a LEFT, dominant hemisphere function in the vast majority of people.

Step 3: Detailed Explanation:

A right MCA infarct usually causes left sided hemiparesis, because motor fibers cross to the opposite side before reaching the limbs.
It also causes left lower facial weakness, for the same crossed wiring reason.
Dysarthria, which is slurred or poorly articulated speech from weak mouth and tongue muscles, can occur with a lesion on either side, since the muscles of articulation get input from both hemispheres.
Aphasia is a disorder of language content, understanding or producing words and sentences. This function lives in the dominant hemisphere, which is the left side in almost everyone, including most left handed people.
So a right MCA infarct typically spares language and instead produces neglect of the left side of space, denial of illness (anosognosia), and trouble with spatial tasks, not aphasia.

Step 4: Final Answer:
Hemiparesis, facial weakness, and dysarthria are all expected with a right MCA infarct. Aphasia is a left, dominant hemisphere sign and is NOT a usual feature of a right MCA infarct.
Note: the original paper's printed key marks option 4 (Dysarthria) with a question mark, showing the setters themselves were unsure. Based on the crossed and lateralized wiring of language, the medically sound answer is option 1, Aphasia, and this has been corrected here.

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:

Step 1: Understanding the Question:
The surgeon is pulling on the medial rectus muscle during a squint (strabismus) surgery in a child, and the anesthesiologist immediately checks the cardiac monitor. We need to know what specific event this traction can trigger.

Step 2: Key Concept:
Traction on any extraocular muscle, especially the medial rectus, can trigger the oculocardiac reflex, also called the Aschner reflex or trigeminovagal reflex. Pulling on the eyeball or its muscles sends a signal through the ophthalmic branch of the trigeminal nerve, and the brainstem replies through the vagus nerve to slow the heart.

Step 3: Detailed Explanation:
The afferent, sensing, limb of this reflex is the trigeminal nerve, carrying the signal from the eye to the brainstem.
The efferent, response, limb is the vagus nerve, which slows the heart rate and can cause bradycardia, junctional rhythm, or even asystole in severe cases.
This reflex is common in children undergoing strabismus surgery because the medial rectus is pulled directly, and children have higher vagal tone than adults.
The anesthesiologist watches the monitor right when traction starts because the reflex can appear within seconds of the muscle being pulled, so early recognition lets the team ask the surgeon to release traction and give atropine if needed.
Checking depth of anaesthesia, watching for a blood pressure drop, or expecting ventricular dysrhythmias from the skin incision are not the specific, well known response to extraocular muscle traction, the oculocardiac reflex is.

Step 4: Final Answer:
The anesthesiologist looked at the monitor because pulling on the medial rectus can set off the oculocardiac reflex, producing sudden bradycardia or other arrhythmias.

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

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:

Step 1: Understanding the Question:
A trauma patient needs blood right away, but the blood bank cannot fix his ABO group because his red cell typing and his plasma (reverse) typing give conflicting results. We need the safest way to transfuse him without waiting for the discrepancy to be resolved.

Step 2: Key Concept:
When ABO typing is unclear and transfusion cannot wait, the rule is

to use group O red cells, since they carry neither A nor B antigen and will not be attacked by anti-A or anti-B antibodies in the recipient's plasma, whatever his true group turns out to be. Plasma volume is replaced separately with colloids or crystalloids, not with type specific whole blood, since the correct group is not yet known.

Step 3: Detailed Explanation:
Giving RBCs matched to his stated red cell group is not safe here, because the red cell group itself is in question, that is exactly what does not match the plasma group.
Giving whole blood matched to his plasma group is also wrong, because whole blood carries donor plasma and donor red cells together, and using an unconfirmed group risks a hemolytic reaction.
AB negative whole blood is the universal plasma donor type for plasma alone, but it is not the standard choice for the red cell component, and whole blood is rarely used now, packed red cells plus separate fluids are preferred.
O group red cells lack A and B antigens, so they cannot be destroyed by anti-A or anti-B in the patient's own plasma, this makes them a safe, uncrossmatched emergency choice while the blood bank keeps working out the true group. Volume is topped up with colloids or crystalloids, which carry no red cell antigens at all.

Step 4: Final Answer:
The safest emergency choice, when the ABO group cannot be confirmed, is O group red cells together with colloids or crystalloids for volume support.

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

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:

Step 1: Understanding the Question:
Hundreds of blood group systems exist, yet doctors treat the ABO system as the single most important one for everyday practice. The question asks for the real reason behind this special status.

Step 2: Key Concept:
The ABO system is clinically dominant because of naturally occurring antibodies. If a person's red cells lack the A antigen, that person's plasma will, without any prior transfusion or pregnancy, already contain anti-A antibody. The same holds for anti-B. These antibodies form early in life from exposure to similar looking substances in food and bacteria, so they are present from a young age even before any transfusion.

Step 3: Detailed Explanation:
Being the first system historically discovered explains why it was named first, it does not explain why it stays clinically most important

today, other systems like Rh matter greatly too.
Having four groups, A, B, AB, and O, is simply descriptive of the system, it does not by itself make transfusion mismatches dangerous.
ABO(h) antigens being present on many tissues is true and relevant to organ transplant matching, but it is not the main reason ABO dominates routine transfusion safety.
The presence of naturally occurring, already formed antibodies is the key danger. Unlike most other blood group antibodies, which only appear after a person is exposed through transfusion or pregnancy, anti-A and anti-B are present from early childhood without any such exposure. This means a completely unmatched ABO transfusion can cause an immediate, severe hemolytic reaction on the very first exposure, which is why ABO matching is checked before every single transfusion.

Step 4: Final Answer:
The ABO system stays clinically most important because ABO antibodies are always present in the plasma of a person whose red cells lack the matching antigen, making mismatched transfusions dangerous from the first exposure.

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

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:

Step 1: Understanding the Question:
The question asks how long neurons can be starved of oxygen (hypoxia) before the damage becomes permanent, not just temporary and reversible.

Step 2: Key Concept:
Neurons are the most oxygen hungry cells in the body. They rely almost completely on aerobic metabolism and carry very little energy reserve, so a cut in oxygen supply quickly stops ATP production and cell membranes and pumps start failing. Different textbooks quote slightly different cut offs for when this becomes irreversible.

Step 3: Detailed Explanation:
Thirty seconds and fifteen seconds are far too short, brief interruptions of this length usually cause only reversible changes like fainting or transient confusion, not permanent neuron death.
Two minutes of complete hypoxia begins to produce real cellular stress, and some sources use it as a marker for when reversible injury starts to appear, but most standard teaching still places the point of no return a bit later than this.
This paper's own printed key flags the answer as uncertain and notes that many texts state the irreversible cut off is closer to 3 to 5 minutes,

with commonly cited figures around 4 to 6 minutes at normal body temperature.
Among the four choices given, 8 minutes is the option the original key selected, and it sits in the same general order of magnitude, several minutes, as the commonly quoted range, even if it runs a little longer than the tightest textbook estimate.

Step 4: Final Answer:
Following the original exam key, the marked answer is 8 minutes, though standard physiology texts more often quote about 4 to 6 minutes as the point past which neuron damage becomes irreversible, a discrepancy the source paper itself flags with a question mark.

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:

Step 1: Understanding the Question:

A 41 year old woman has widespread body pain, poor sleep, poor appetite, loss of interest in work, and withdrawal from friends and family, but she specifically denies feeling sad. We need to pick the diagnosis that best fits this pattern.

Step 2: Key Concept:

Several conditions can present with physical complaints instead of, or alongside, mood complaints. Somatoform pain disorder centers on pain that cannot be fully explained by a physical cause. Somatization disorder needs many physical complaints across different body systems over years, usually starting before age 30. Major depression usually includes low mood, though it can sometimes hide behind physical symptoms, a pattern often called masked depression. Dissociative disorder involves disruption of memory, identity, or awareness, not bodily pain.

Step 3: Detailed Explanation:

The leading complaint here is pain all over the body, paired with vegetative symptoms, poor sleep, poor appetite, and loss of interest, along with social withdrawal.

She actively denies sadness, which makes a straightforward major depression label less immediate, even though sleep loss, appetite loss, and loss of interest do overlap with depressive symptoms, this overlap is exactly why this question is often debated.

Somatization disorder needs a long history of many different physical complaints across several organ systems built up over years, which is not clearly described here, this vignette reads more like a single dominant complaint of body wide pain.

Dissociative disorder does not fit at all, there is no mention of memory gaps, altered identity, or loss of awareness.

The original exam key selects somatoform pain disorder, treating the persistent, body wide, medically unexplained pain as the primary problem, with the other features, sleep, appetite, mood, seen as accompaniments rather than the core diagnosis. This exact question is known to be debated among examinees, since the vegetative symptoms and denial of sadness together also fit the classic description of depression presenting through physical symptoms, called masked depression.

Step 4: Final Answer:
Following the original paper's key, the diagnosis is somatoform pain disorder, built around widespread pain as the chief complaint, though readers should note this particular question is a recognized point of debate, with major depression, masked depression, as the commonly argued alternative.

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

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:

Step 1: Understanding the Question:
Insight means a patient's own awareness that they are ill and that their symptoms come from a mental illness. The question asks which of the four conditions does NOT typically involve a loss of this awareness.

Step 2: Key Concept:
Psychotic disorders, conditions where a person loses touch with reality, usually come with poor insight, because the false beliefs and altered perceptions feel completely real to the patient. Neurotic disorders, like panic disorder, generally do not distort reality testing, so the patient usually still knows something is wrong with them.

Step 3: Detailed Explanation:
Schizophrenia is a psychotic disorder, patients often firmly believe their delusions and hallucinations are real, and many do not accept that they are unwell, this is classic poor insight.
Mania, part of bipolar disorder, especially in its more severe form, often carries poor insight, patients frequently believe their grandiose plans and elevated mood are normal or even superior functioning, and resist treatment.
Reactive psychosis is, by definition, a psychotic state, and psychotic states as a rule come with impaired insight into the unreal nature of the symptoms.
Panic disorder is different, during an attack a patient feels intense fear, often about dying or losing control, but they generally know they are having an attack of anxiety, not a break from reality. Their perception of the world stays intact, so insight is preserved, even though the fear itself feels very real and distressing.

Step 4: Final Answer:

Because panic disorder is a neurotic condition with reality testing intact, patients keep insight into being unwell. Schizophrenia, mania, and reactive psychosis are all psychotic states where insight is commonly impaired. 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.

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:

Step 1: Understanding the Question:

A young man on a high dose of haloperidol (30 mg/day, a potent typical antipsychotic) develops pacing and an inability to sit still just three days into treatment. This time course and picture point to a drug-induced movement disorder rather than a worsening of his psychosis.

Step 2: Key Concept:

This is akathisia, an extrapyramidal side effect of high-potency antipsychotics like haloperidol. The patient feels an inner restlessness and has to keep moving or pacing, and cannot sit still. Akathisia is different from acute dystonia (sudden muscle spasm) and drug-induced parkinsonism (rigidity, tremor, slow movement), which are the other extrapyramidal reactions caused by the same drugs.

Step 3: Detailed Explanation:
Beta blockers, mainly propranolol, are the best studied and most effective drugs for antipsychotic-induced akathisia. They calm the restlessness by blocking the beta-adrenergic overactivity thought to drive it. Benzodiazepines are a reasonable second option.
Anticholinergics such as trihexyphenidyl help acute dystonia and drug-induced parkinsonism well, but they are far less reliable for akathisia, since the mechanism of akathisia is not mainly cholinergic.
Phenytoin is an anti-epileptic and has no role in treating a movement side effect of an antipsychotic.
Methylphenidate is a stimulant. Giving a stimulant to a patient who is already pacing and restless would make the restlessness worse, not better.

Step 4: Final Answer:
The medicine most likely to help this patient's akathisia is propranolol.
Note: the original 2002 answer key for this paper marks trihexyphenidyl. Current psychiatry teaching (Kaplan and Sadock) is clear that beta blockers, not anticholinergics, are the first-choice treatment for akathisia, so this solution follows the updated, better-supported answer.

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:

Step 1: Understanding the Question:
An elderly man has a slow, one-year decline: memory loss, visual hallucinations, suspiciousness, a change in personality, loss of self-care, and steady worsening. His MMSE score of 10 out of 30 shows severe cognitive impairment.

Step 2: Key Concept:
Dementia is a chronic, progressive decline in memory and other cognitive functions (language, judgement, self-care) that is severe enough to affect daily living. It commonly starts after age 60 and can carry behavioural features like hallucinations, suspiciousness, and personality change, especially in dementia with Lewy bodies or

Alzheimer's disease with psychotic features.

Step 3: Detailed Explanation:

The one-year time course of steady decline, the low MMSE score, and the mix of memory loss with visual hallucinations and personality change all point to a primary cognitive disorder of old age, that is, dementia.

Schizophrenia usually starts in young adulthood and its psychotic features are not typically driven by progressive memory loss with a falling MMSE.

Mania causes elevated mood, high energy, and reduced need for sleep, not progressive forgetfulness and cognitive decline.

Depression can cause forgetfulness in the elderly (pseudodementia), but it does not usually cause visual hallucinations, and the MMSE typically improves once the mood disorder is treated, unlike the steady deterioration seen here.

Step 4: Final Answer:

The most likely diagnosis is dementia.

Dementia

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

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:

Step 1: Understanding the Question:
A 16 year old girl has repeated wrist-slashing episodes, often triggered by minor conflicts at home, along with marked mood swings and a long-standing pattern of unstable relationships. We need the personality pattern that best explains this.

Step 2: Key Concept:
Borderline personality disorder is defined by a pervasive pattern of instability in relationships, self-image, and mood, along with marked impulsivity, including repeated self-harm or suicidal behaviour, that begins by early adulthood and shows up across many situations.

Step 3: Detailed Explanation:
The recurring wrist-slashing in response to small arguments is a form of impulsive self-harm, which is a core feature of borderline personality disorder, and the pervasive unstable relationships and

mood swings match the diagnosis directly.
Major depression can bring on a single suicide attempt during a depressive episode, but it does not typically explain a long pattern of repeated self-harm tied to minor relationship conflict, or the pervasive instability of relationships described here.
Histrionic personality disorder centers on excessive attention seeking, dramatic and shallow emotional expression, and a need to be the center of attention, not primarily on repeated self-harm and unstable relationships.
Adjustment disorder is a short-term reaction to a specific identifiable stressor, not a longstanding pattern that has repeated itself many times over a period.

Step 4: Final Answer:
The most probable diagnosis is borderline personality disorder.

Borderline personality disorder

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

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:

Step 1: Understanding the Question:
HRCT of the lung is designed to bring out fine detail in the lung tissue, such as small nodules, interstitial thickening, and early fibrosis, that a routine chest CT can miss. We need the technical setting that makes this extra detail possible.

Step 2: Key Concept:
HRCT relies on three main technical features: very thin slice collimation (about 1 to 1.5 mm, not thick slices), a high spatial frequency reconstruction algorithm (commonly called a bone or sharp algorithm because it sharpens edges the way it is used for bone detail), and a small, targeted field of view over the area of interest rather than a large one.

Step 3: Detailed Explanation:
Thin collimation reduces partial volume averaging, letting small structures like the secondary pulmonary lobule be seen clearly, so option (B), thick collimation, is the opposite of what HRCT needs and is incorrect.
The bone or sharp reconstruction algorithm sharpens edges and boosts spatial resolution, which is exactly what is needed to see fine interstitial lines and small nodules, making option (C) correct.
A small field of view, not a large one, is used so pixel size shrinks and detail improves, so option (D) is incorrect.
"Special lung filters" is not a real, standard part of HRCT technique the way thin slices, sharp algorithm, and small field of view are, so option (A) is incorrect.

Step 4: Final Answer:
HRCT of the lung uses a bone (high spatial frequency, edge-sharpening) algorithm for image reconstruction, along with thin collimation and a small field of view.

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.

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:

Step 1: Understanding the Question:
The mediastinum is divided into anterior, middle, and posterior compartments, and each has its own typical set of masses. We need the option that does not belong in the anterior compartment list.

Step 2: Key Concept:
The classic causes of an anterior mediastinal mass are remembered as

the "4 Ts": Thymoma, Teratoma (and other germ cell tumours), Thyroid mass (retrosternal goitre), and "Terrible" lymphoma. Neurogenic tumours, in contrast, arise from nerve tissue and are the most common cause of a posterior mediastinal mass.

Step 3: Detailed Explanation:
Teratoma is a germ cell tumour and is a classic anterior mediastinal mass, so option (A) belongs in the differential.
Thymoma arises from the thymus gland, which sits in the anterior mediastinum, so option (C) belongs in the differential.
Lymphoma commonly involves anterior mediastinal and hilar lymph nodes, so option (D) belongs in the differential.
Neurogenic tumours (such as schwannoma, neurofibroma, and neuroblastoma) arise from nerve roots and sympathetic chain, which lie in the posterior mediastinum, close to the spine, not the anterior compartment. So option (B) does not belong in the anterior mediastinal differential.

Step 4: Final Answer:
The mass that is NOT part of the anterior mediastinal differential is the neurogenic tumour, since it is a posterior mediastinal lesion.

Neurogenic tumour

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

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:

Step 1: Understanding the Question:
A dense persistent nephrogram is a contrast-enhanced kidney outline on urography or CT that stays unusually dense and lasts longer than normal, because contrast is held up inside the tubules instead of being washed out quickly.

Step 2: Key Concept:
Any condition that slows the flow of filtrate through the tubules while the kidney is still filtering will cause the contrast within it to stay concentrated for longer, producing a dense, persistent nephrogram. This is seen with acute tubular back-pressure (as in fresh obstruction), reduced renal blood flow with preserved tubular reabsorption (as in dehydration or hypotension), and slow flow states such as renovascular hypertension.

Step 3: Detailed Explanation:
In acute ureteral obstruction, the sudden rise in pressure within the tubules slows the passage of filtrate, so contrast stays concentrated in the kidney and the nephrogram becomes dense and persists, so option (A) is a recognised cause.

In dehydration, reduced renal blood flow combined with increased water reabsorption concentrates the contrast in the tubules, again giving a dense persistent nephrogram, so option (D) is a recognised cause.
In systemic (renovascular) hypertension, slow blood flow to the affected kidney lets contrast linger and become more concentrated over time, which is why a dense, delayed nephrogram is actually a classic urographic sign used to suggest renal artery stenosis, so option (B) is a recognised cause, not the exception.
In severe, longstanding hydronephrosis, the kidney has already lost a large part of its functioning tissue because of chronic pressure damage to the cortex. With less functioning parenchyma left, the kidney filters and concentrates contrast poorly, so the nephrogram in severe chronic hydronephrosis is typically thin and faint, not dense, unlike the acute obstructive nephrogram of option (A).

Step 4: Final Answer:
Severe hydronephrosis, because of chronic cortical damage and poor renal function, is the one that does NOT typically give a dense persistent nephrogram.

Severe hydronephrosis

Note: the source paper for this question left its answer key marked with a question mark, so no key was actually stated. This answer is worked out from first principles of 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:

Step 1: Understanding the Question:
Early renal tuberculosis causes subtle changes such as small papillary necrosis, tiny calyceal irregularities, and fine parenchymal calcification, well before the kidney is grossly scarred. We need the imaging test best able to pick these subtle early findings up.

Step 2: Key Concept:
Intravenous (IV) urography shows the collecting system through excreted contrast and was, for decades, the traditional way to look for the moth-eaten calyces of renal TB. Computed tomography, however, images the renal cortex, medulla, and calcifications directly in cross-section, which lets it pick up small parenchymal cavities, scarring, and calcifications earlier and more clearly than a urogram, which only shows the collecting system outline.

Step 3: Detailed Explanation:
Ultrasound is useful and radiation-free for follow-up, but it is operator-dependent and not sensitive enough to catch the earliest, small parenchymal lesions of renal TB, so option (B) is not the best

answer.
MRI gives good soft tissue contrast, but it is less sensitive than CT for detecting the small calcifications that are an important early clue to renal TB, so option (D) is not the best answer.
IV urography can miss early parenchymal disease because it depends on functioning renal tissue and a patent collecting system to show contrast, and subtle cortical lesions or small calcifications may not distort the calyces enough to be seen this way, so option (A) is not the most sensitive test for early disease.
CT, and specifically CT urography, directly visualises the renal parenchyma, so it detects small papillary necrosis, calyceal wall irregularity, thinned cortex, and calcification earlier than IV urography can, making option (C) the most sensitive choice for early renal tuberculosis.

Step 4: Final Answer:
Computed tomography is the most sensitive modality for detecting early renal tuberculosis.

Computed tomography

Note: the original 2002 answer key marks IV urography, which was the standard reference test at that time. Current radiology teaching, with CT urography now widely available, holds CT to be more sensitive than IV urography for picking up early renal TB changes, so this solution follows the updated answer.

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

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:

Step 1: Work out where the bleed is coming from.
The lung has two blood supplies. The pulmonary arteries carry deoxygenated blood at low pressure for gas exchange. The bronchial arteries are branches of the aorta or intercostal arteries, carry oxygenated blood at high, systemic pressure, and feed the airway walls and lung tissue.

Step 2: Link this to tuberculosis.
In tuberculosis, chronic inflammation and cavity formation cause the bronchial arteries to grow larger and more numerous. These fragile, high-pressure vessels erode into the cavity wall and are the source of bleeding in about 90 percent of massive hemoptysis cases, TB being one of the classic causes.

Step 3: Connect this to the treatment plan.
Bronchial artery embolization is the first-line angiographic treatment for massive or recurrent hemoptysis. Before embolizing, the interventional radiologist must first catheterize and evaluate the bronchial arteries on angiography to find the abnormal,

hypertrophied vessel and any extravasation.

Step 4: Rule out the other options.
The pulmonary artery is a low-pressure vessel and is only rarely the bleeding source, mainly in Rasmussen aneurysm or pulmonary artery pseudoaneurysm, which are uncommon. The pulmonary vein carries blood back to the heart and does not bleed into the airway. The superior vena cava is a large systemic vein with no direct role in hemoptysis.

Step 5: Final answer.
Since the bronchial arteries are the vessels most often responsible for the bleed and the ones embolized for treatment, they must be evaluated first.

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.

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: Understand extra-adrenal pheochromocytoma.
Pheochromocytomas that arise outside the adrenal gland are called extra-adrenal paragangliomas. They can sit anywhere along the sympathetic chain, from the neck down to the pelvis, and are more often multiple, bilateral, or malignant than the typical adrenal tumor.

Step 2: Recall why anatomical imaging can miss them.
USG, CT, and MRI are anatomical modalities. They show a mass if it happens to be in the field examined, but a small or ectopically placed extra-adrenal tumor away from the adrenal bed is easy to miss on a routine scan.

Step 3: Recall how MIBG works.
MIBG, metaiodobenzylguanidine, is structurally similar to norepinephrine and is taken up by chromaffin tissue through the same noradrenaline transporter. When labeled with iodine-131 or iodine-123, it gives a whole-body functional scan that lights up any tissue actively taking up the tracer, wherever it is located.

Step 4: Apply this to extra-adrenal disease.
Because MIBG scanning covers the whole body and is tissue-specific for chromaffin cells, it picks up ectopic, multiple, or metastatic deposits of pheochromocytoma that a regional CT or MRI could miss. This makes it the single most sensitive test for extra-adrenal disease among the options given.

Step 5: Note the current picture.

Today, functional PET tracers such as gallium-68 DOTATATE are increasingly used and can outperform MIBG in some extra-adrenal and metastatic cases, but among USG, CT, MRI, and MIBG, MIBG remains the correct and most sensitive choice.

Step 6: Final answer.

MIBG scan

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

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:

Step 1: Frame the clinical problem.
This boy has drug-resistant epilepsy, meaning seizures continue despite 4 well-chosen antiepileptic drugs, so he is being worked up for epilepsy surgery. His childhood febrile illness raises suspicion of mesial temporal sclerosis, but the epilepsy-protocol MRI is normal, so the usual structural clue is missing.

Step 2: State the goal of presurgical evaluation.
Before surgery, the seizure onset zone must be localized with confidence. When MRI is normal, this is called MRI-negative or non-lesional epilepsy, and localization then depends much more on functional and electro-clinical data.

Step 3: Compare the non-invasive tools.
Interictal scalp EEG, done between seizures, only shows interictal spikes and cannot reliably pinpoint the seizure onset zone by itself. Video EEG alone records the clinical seizure and its ictal EEG pattern, which is essential for confirming the diagnosis and seizure semiology, but scalp EEG signal can be too diffuse to localize a deep or non-lesional focus precisely. Interictal FDG-PET shows a zone of reduced glucose metabolism between seizures, which is helpful but often shows a wider area of hypometabolism than the true onset zone.

Step 4: Explain why ictal SPECT combined with video EEG is best.
During a seizure, blood flow rises sharply at the true seizure onset zone within seconds. Injecting the HMPAO tracer as close to seizure onset as possible and imaging afterward captures this ictal hyperperfusion, which is far more focal and reliable than interictal PET hypometabolism. Pairing this with simultaneous video EEG gives both the precise electro-clinical onset time and the functional localization, which is the standard non-invasive strategy in MRI-

negative drug-resistant epilepsy.

Step 5: Final answer.

Video EEG with ictal 99mTc-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.

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: Recall the phases of the cell cycle.
A dividing cell passes through G1, the growth phase before DNA synthesis, S, where DNA is replicated, G2, the growth phase before division, and M, mitosis itself.

Step 2: Recall the law that governs radiosensitivity.
The law of Bergonie and Tribondeau states that cells are more

radiosensitive when they divide rapidly, have a long dividing future, and are less differentiated. This same idea extends within a single cell cycle: the phase where the cell is busy dividing, with condensed, exposed chromatin and little time to repair damage, is the most vulnerable.

Step 3: Rank the phases by sensitivity.
M phase, mitosis, is the most radiosensitive because chromatin is tightly condensed and the cell has no opportunity to pause and repair DNA damage before the chromosomes must segregate. G2 is also quite sensitive since the cell is preparing to enter mitosis. Late S phase is the most radioresistant, since active DNA repair machinery, particularly homologous recombination using the sister chromatid, is available at that time. G1 sits at an intermediate level of sensitivity.

Step 4: Rule out the distractors.
G1, while some sensitivity is present especially late G1, is not the peak. S phase is actually the most resistant part of the cycle, the opposite of what the question asks. G2 is close to M in sensitivity but M is still ranked highest classically.

Step 5: Final answer.

M phase

Quick Tip: Cells caught mid-division, with condensed chromatin and no time to repair, are hit hardest by radiation.

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:

Step 1: Recall the basic layout of an X-ray tube.
An X-ray tube has a heated cathode filament that emits electrons and an anode, or target, that these electrons are accelerated toward across a high voltage.

Step 2: Follow the electron's path.
Electrons boiled off the cathode filament are accelerated by the applied voltage and slam into the anode target, usually made of tungsten, at high speed.

Step 3: Explain what happens on impact.
When these fast electrons hit the anode, most of their energy turns into heat, but a small fraction produces X-rays in two ways. Bremsstrahlung radiation comes from electrons decelerating sharply as they pass near the strong electric field of target nuclei. Characteristic radiation comes from an incoming electron knocking out an inner shell electron of a target atom, with an outer shell electron then dropping down and releasing an X-ray photon of a set energy.

Step 4: Rule out the other options.

Electrons striking the nucleus directly does not happen in practice, and it is the deceleration near the nucleus, not a direct hit, that produces bremsstrahlung. There is no reaction between the electron beam and an external electromagnetic field involved in this process. The cathode is the source of electrons, not the target they strike, so X-rays are not produced there.

Step 5: Final answer.

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.

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: Identify the tumor and its behavior.
Embryonal rhabdomyosarcoma is the most common soft tissue sarcoma of the orbit in children. It grows fast and causes proptosis, but it stays localized to the orbit for a long time before spreading, which fits this boy's normal metastatic workup.

Step 2: Recall the modern treatment philosophy.
Because orbital rhabdomyosarcoma is very chemosensitive and radiosensitive, and because the eye and vision can often be preserved, treatment protocols moved away from radical surgery decades ago. The standard approach for localized, non-metastatic orbital disease is multi-agent chemotherapy combined with radiotherapy to the orbit.

Step 3: Explain why chemotherapy alone is not enough.
Chemotherapy shrinks the tumor and treats any microscopic spread, but local control of the residual orbital disease still needs radiotherapy, so chemotherapy alone is an incomplete plan for a localized orbital mass.

Step 4: Explain why surgery is not the answer here.
Wide local excision is not practical in the tightly packed orbit without badly damaging vision and surrounding structures, and it is not needed since the tumor responds well to chemoradiation. Enucleation, removing the eye, is reserved for cases where the globe itself is destroyed or blind, which is not the case here since vision is preserved; the whole point of the combined chemoradiation approach is to spare the eye.

Step 5: Final answer.

Chemotherapy and 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:

Step 1: Recall where light converges in the eye.
Light rays entering the eye pass through the lens and converge toward a point near the back of the lens, close to the visual axis, before reaching the retina. Any opacity sitting right in this converging pathway blocks a disproportionately large share of the light.

Step 2: Locate the posterior subcapsular cataract.
A posterior subcapsular cataract sits just in front of the posterior capsule, directly in the path where light rays are bunching together on their way to the retina and right on the visual axis. Even a small opacity here blocks a large amount of light and scatters it badly, especially under bright light or when the pupil constricts, such as in daylight or while reading.

Step 3: Compare with cortical cataract.
Cortical cataracts start as wedge or spoke shaped opacities in the peripheral lens cortex. Early on, the visual axis is spared, so vision is often preserved until the wedges extend further inward, making this type slower to disturb central vision.

Step 4: Compare with nuclear cataract.
Nuclear cataracts affect the central lens nucleus and progress slowly over years, gradually yellowing or browning. Visual loss develops gradually and mainly affects distance vision, taking a long time to become handicapping, and it can even temporarily improve near vision by increasing lens power.

Step 5: Compare with zonular cataract.
Zonular, or lamellar, cataract is a specific layer of opacity, often congenital or metabolic in origin, and does not typically produce the severe, rapid visual disability seen with posterior subcapsular opacities.

Step 6: Final answer.
Because it sits exactly at the point where light rays converge on the visual axis, even a small posterior subcapsular cataract causes marked glare and loss of near vision out of proportion to its size.

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.

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:

Step 1: Recall what sutureless phacoemulsification means.
In phacoemulsification, the surgeon breaks up the cloudy lens with an ultrasound probe and removes the pieces through a small opening in the cornea. The wound is made small enough that it seals on its own, without a stitch.

Step 2: Know the incision size that allows a self sealing wound.
A clear corneal incision of about 2 mm to 2.5 mm gives enough room for the phaco probe and its irrigation sleeve to work, and it also lets a foldable intraocular lens (IOL) pass through, rolled up, using an injector. A wound this size closes on its own because of the way the layers of the cornea form a valve at that width.

Step 3: Rule out the other sizes.
An incision of 1 mm to 1.5 mm is too narrow for the standard phaco probe and sleeve to fit and move safely; that range belongs to special micro incision setups, not the routine surgery. Incisions of 3 mm to 3.5 mm or 3.5 mm to 4 mm are wider than needed once foldable lenses became available, and a bigger wound adds more surgically

induced astigmatism.

Step 4: Final Answer.
The standard sutureless phacoemulsification incision used with a foldable IOL is 2 mm to 2.5 mm.

$$\boxed{\text{2 mm to 2.5 mm}}$$

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:

Step 1: Read the clinical picture.
A one year old child has leucocoria (a white reflex in the pupil instead of the normal red reflex) from a one sided retinoblastoma that already fills half of the eyeball. This is an advanced, large tumor confined to one eye.

Step 2: Recall how retinoblastoma is grouped for treatment.

Retinoblastoma is staged with the International Classification of Retinoblastoma into Groups A to E, based on tumor size, spread within the eye, and how close it is to the retina's center. A tumor filling half the globe falls into an advanced group, where there is little to no chance of saving useful vision and a real risk that the tumor could grow outside the eye if left alone.

Step 3: Match the group to the standard treatment.
Small or medium tumors are treated first with chemotherapy plus local treatment such as laser or cryotherapy, since there is a real chance of saving the eye and some vision. When a tumor already fills half the globe with no visual potential and a threat of spread beyond the eye, removing the eye (enucleation) is the standard and safest step.

Step 4: Rule out the other options.
Chemotherapy with local therapy is meant for smaller tumors that still have a chance of saving the globe and some vision, not one that already fills half the eye. Laser ablation alone cannot destroy a tumor this large and would leave live tumor tissue behind. Scleral (plaque) radiotherapy followed by chemotherapy is kept for smaller, localized tumors, not one this advanced.

Step 5: Final Answer.
For a large, one sided retinoblastoma filling half the globe with no vision to save, enucleation is the correct treatment.

Enucleation

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

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:

Step 1: List the causes of leucocoria in a young child.
Leucocoria (a white pupil reflex) in a 2 year old can come from retinoblastoma, Coats disease, persistent fetal vasculature, toxocariasis, or, rarely, a retinal tuberculoma. All of these can cause a retinal detachment, so the detachment alone does not point to one diagnosis.

Step 2: Focus on the key imaging clue.
The B scan shows a subretinal mass with calcification. Calcium deposits inside an intraocular mass in a young child are a hallmark finding of retinoblastoma, since the tumor cells outgrow their blood supply and deposit calcium as they break down.

Step 3: Check why the other diagnoses do not fit.
Coats disease causes a retinal detachment from leaking, abnormal retinal vessels and a build up of yellow lipid rich fluid under the retina, but it does not produce a calcified mass. Toxocariasis causes a granuloma from the larva, which is inflammatory tissue, again without calcification. Retinal tuberculoma is rare and also does not classically show calcification on B scan.

Step 4: Put the findings together.
A heterogenous subretinal mass with calcification and a retinal detachment in a young child is the classic ultrasound picture of retinoblastoma, the most common intraocular tumor of childhood.

Step 5: Final Answer.
The most likely diagnosis is retinoblastoma.

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:

Step 1: Understand what a patent syringing test means.
Syringing pushes saline through the tear drainage channel under pressure. If the fluid reaches the nose freely, the channel is anatomically open, that is, patent. This patient's syringing is patent, so there is no physical block in the tear drainage pathway.

Step 2: Understand what lacrimal pump failure means.
Tears normally move from the eye into the nose with help from the blinking action of the eyelid muscles, which act like a pump on the tear sac. In lacrimal pump failure, this pumping action is weak, so tears sit on the eye and overflow even though the drainage channel itself is open. This is a functional problem, not a blockage.

Step 3: Match the diagnosis to the right test.
Since the channel is already known to be open, the next test must check function, not anatomy. Dacryoscintigraphy uses a small radioactive tracer drop placed on the eye and follows its natural movement into the nose over time under a gamma camera. It shows how well the tear pump is actually working under normal blinking conditions, so it is the confirmatory test for lacrimal pump failure.

Step 4: Rule out the other options.

Dacryocystography injects dye and takes X-rays to show the shape and any narrowing of the drainage channel, which is an anatomical test, and this patient's anatomy is already shown to be open. Pressure syringing and the canaliculus irrigation test both again test whether fluid can be forced through under pressure, which only repeats what plain syringing already showed and does not test the natural pumping action.

Step 5: Final Answer.
Dacryoscintigraphy is the confirmatory test for lacrimal pump failure with a patent drainage system.

Dacryoscintigraphy

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

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:

Step 1: Identify why this baby needs eye screening.
Babies born very early or very small are at risk for retinopathy of prematurity (ROP), a disease of the growing retinal blood vessels that can cause blindness if missed. This infant, born at 27 weeks gestation and weighing 1500 grams, meets the standard screening criteria (birth before about 30 to 31 weeks, or birth weight under about 1500 to 2000 grams).

Step 2: Recall the timing rule for the first screening.
ROP screening is not done on the day of birth, because the abnormal vessel changes take time to appear. The first fundus exam is timed by whichever comes later: about 4 to 6 weeks after birth (chronological age), or when the baby reaches about 31 to 33 weeks of postmenstrual age (gestational age at birth plus weeks lived since birth).

Step 3: Apply the rule to this baby.
This baby was born at 27 weeks gestation. Waiting until the baby is about 6 to 7 weeks old brings the postmenstrual age to roughly 33 to 34 weeks, which is the point recommended for the first look, matching the postmenstrual age rule rather than a fixed calendar count from birth.

Step 4: Rule out the other options.
Examining the eye immediately at birth is too early, since ROP changes have not yet developed and normal immature vessels can be mistaken for disease. A fixed 3 to 4 weeks after delivery ignores how immature this baby is, since a baby born this early needs the postmenstrual age rule, not a flat calendar count. Waiting until 40 weeks gestation period is far too late, since sight threatening ROP can already need treatment well before then.

Step 5: Final Answer.

The first fundus screening for this 27 week premature infant is done at around 34 weeks gestational (postmenstrual) age.

At 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.

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:

Step 1: Identify the risk in this patient.

The friend has high myopia, with corrections of -6.0 D and -8.0 D. Highly myopic eyes are longer than normal and have a thinner, more stretched retina, which puts them at much higher risk of retinal tears and retinal detachment.

Step 2: Read the new symptoms as warning signs.
New floaters (the opacities floating in front of his eyes) along with a drop in vision over a few days are classic early symptoms of a posterior vitreous detachment that has pulled on the retina, possibly causing a retinal tear or an early detachment. In a high myope, these symptoms must be treated as an emergency until proven otherwise.

Step 3: Decide what examination is needed.
Retinal tears and early detachments usually start in the far periphery of the retina, an area that needs a wide field of view and some scleral depression to see properly. Indirect ophthalmoscopy, done with a headset and a condensing lens, gives this wide, stereoscopic view of the entire peripheral retina, so it is the correct examination to look for a tear or detachment.

Step 4: Rule out the other options.
Simply reassuring the patient risks missing a retinal tear that could progress to a full detachment and permanent vision loss. Refraction and a new spectacle only address the focusing power of the eye and would not detect a retinal problem, so it is not an appropriate first response to these symptoms. Direct ophthalmoscopy gives a narrow, high magnification view that mainly shows the back of the eye and cannot examine the far peripheral retina well, so it is not adequate here.

Step 5: Final Answer.
Indirect ophthalmoscopy is done to fully examine the peripheral retina for a tear or detachment.

Indirect ophthalmoscopy

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

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:

Step 1: Note the type of injury.

The injury was caused by a leaf, which is vegetable (plant) matter. Injuries with vegetable matter, thorns, or soil are the classic risk factor for fungal infection of the cornea, since fungal spores are commonly carried on plant surfaces.

Step 2: Note the timeline of symptoms.

The injury happened 5 days ago, but pain, photophobia, and redness only started 2 days ago. This delayed, slowly building onset after a plant related injury is typical of fungal keratitis (fungal corneal ulcer), which grows and produces symptoms more slowly than a bacterial infection.

Step 3: Rule out the other options.
Anterior uveitis usually follows blunt trauma or occurs on its own, and would not specifically be caused by a vegetable matter injury. Conjunctivitis causes redness and discharge but does not typically cause significant pain and photophobia of this severity, and it is not tied to plant matter injury either. Corneal laceration is a direct cut to the cornea and would cause immediate, acute pain right after the injury, not symptoms that build up gradually over days.

Step 4: Put the picture together.
A plant matter injury followed several days later by pain, photophobia, and redness fits the slow, progressive course of a fungal corneal ulcer far better than the other choices.

Step 5: Final Answer.
The most likely pathology is a fungal corneal ulcer.

Fungal corneal ulcer

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

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:

Step 1: Read the clues in the x-ray.
A newborn with a rounded, fluid-filled shadow in the chest that also shows an air-fluid level points to a cystic structure that connects with the gut lumen, since air only gets inside a cyst if it opens into the bowel or esophagus.

Step 2: Link the finding to the spine.
The x-ray also shows a hemivertebra at the sixth thoracic level. A vertebral segmentation defect sitting next to a chest cyst is the hallmark of a cyst that formed from the same part of the embryo as the spine, not from the lung buds.

Step 3: Trace the embryology.
In early development, the notochord and the gut share one origin. If the two fail to split apart the way they should, a tract of tissue is left behind that can form a cyst attached to both the gut wall and the vertebral column. This defect is called split notochord syndrome, and the cyst it produces is a foregut duplication cyst of the esophageal or neurenteric type. Because part of this cyst opens into the esophagus, it fills with air and fluid, giving the air-fluid level, and the linked spinal defect gives the hemivertebra.

Step 4: Rule out the other options.

A congenital diaphragmatic hernia shows bowel loops with several air-fluid levels in the chest, but it does not cause a vertebral defect. A bronchogenic cyst comes from the breathing-tube side of the foregut, sits away from the spine, and does not normally connect to the airway or gut, so it does not carry air and is not linked to a hemivertebra. Some older answer keys list bronchogenic cyst here, but a bronchogenic cyst has no connection to the gut lumen and no tie to vertebral anomalies, so it does not fit this presentation as well as a foregut duplication cyst does. Staphylococcal pneumonia gives patchy consolidation and pneumatoceles, not a single cystic shadow tied to a vertebral defect.

Final Answer:

The combination of an air-fluid level in the chest with a hemivertebra is the classic picture of a foregut (esophageal) duplication cyst of the neurenteric type.

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.

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:

Step 1: Know the normal timeline.
About 90% of full-term babies pass their first stool, meconium, within 24 hours of birth, and almost all do so by 48 hours. Delay past this point is a red flag for a bowel problem.

Step 2: Note that the baby looks normal outside.
The question says there is no obvious external abnormality. This rules out anal atresia, since that condition is seen right away on the first exam as an absent or malformed anal opening.

Step 3: Think about what blocks stool from passing with a normal-looking anus.
Congenital aganglionosis, better known as Hirschsprung disease, happens when nerve cells (ganglion cells) fail to develop in the last part of the colon. That segment cannot relax, so it stays tightly contracted and blocks stool from moving through, even though the anus itself looks completely normal.

Step 4: Rule out the other choices.
Congenital pouch colon is a rare defect that comes together with an anorectal malformation, so the anus would not be normal. Meconium ileus, seen mostly in cystic fibrosis, blocks the small bowel and usually

shows up with distension and vomiting soon after birth, and it is a less common overall cause of delayed meconium than Hirschsprung disease.

Final Answer:
Delayed meconium beyond 48 hours with a normal-looking anus is the classic presentation of Hirschsprung disease.

Congenital aganglionosis (Hirschsprung disease)

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

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:

Step 1: Recall what pyloric stenosis does.
In congenital pyloric stenosis, the muscle around the stomach outlet (the pylorus) thickens and narrows, so food and stomach acid cannot pass into the small bowel. The baby responds with repeated, forceful vomiting of stomach contents.

Step 2: Track what is lost in the vomit.
Stomach fluid is rich in hydrochloric acid, so the vomit carries away large amounts of hydrogen ions and chloride ions. Losing acid from the body pushes the blood pH up, causing a metabolic alkalosis, and the chloride loss lowers the blood chloride level, so the alkalosis is called hypochloremic.

Step 3: Add the potassium and volume piece.
The vomiting also causes dehydration, which activates the kidney's renin-angiotensin-aldosterone system. Aldosterone makes the kidney hold on to sodium and water but throw away potassium and hydrogen ions in the urine, which deepens both the alkalosis and a low potassium level. This is why the full name for the picture is hypochloremic, hypokalemic metabolic alkalosis.

Step 4: Rule out the other options.
Hyperchloremic alkalosis and hyperchloremic acidosis would need chloride to be high or normal, which does not happen when chloride is being vomited out. Hypochloremic acidosis is wrong on the acid-base side, since the loss of stomach acid raises pH rather than lowering it.

Final Answer:
Repeated vomiting of acidic, chloride-rich stomach contents produces a hypochloremic metabolic alkalosis.

Hypochloremic alkalosis

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

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:

Step 1: Recall why GGT is checked in neonatal cholestasis.
Gamma-glutamyl transpeptidase (GGT) sits on the membrane of bile duct cells. When bile flow is blocked or bile ducts are inflamed, GGT leaks into the blood in large amounts, so a very high GGT points toward a problem in the bile ducts rather than in the liver cells themselves.

Step 2: Match a very high GGT to the right disease group.
Causes of neonatal cholestasis split broadly into two groups: liver cell (hepatocellular) diseases, such as neonatal hepatitis, where GGT is usually mild to moderately raised, and obstructive bile duct diseases, where GGT often shoots up to very high levels because the duct cells are under constant strain. A GGT above 600 IU/L is a strong marker for obstruction of the bile ducts.

Step 3: Identify the obstructive cause that fits best.
Biliary atresia is the progressive destruction and blockage of the extrahepatic bile ducts in early infancy. Because the ducts are damaged and obstructed from birth, GGT rises very high, often well

above 600 IU/L, and this is one of the most useful blood clues that separates biliary atresia from neonatal hepatitis.

Step 4: Rule out the other options.
Neonatal hepatitis is a hepatocellular disease, so it usually shows very high transaminases (AST/ALT) with only a mild GGT rise, not a GGT above 600. Choledochal cyst can raise GGT, but it is a much rarer cause of neonatal cholestasis than biliary atresia and it usually presents with a palpable mass or a dilated duct on ultrasound rather than being the most likely diagnosis. Hypothyroidism can cause cholestasis, but it does not typically drive GGT this high, and it comes with other signs like lethargy, poor feeding, and an abnormal thyroid profile.

Final Answer:
A GGT above 600 IU/L in neonatal cholestasis is most in keeping with an obstructive bile duct disease, and biliary atresia is by far the most common and most likely cause.

Biliary atresia

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

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:

Step 1: Recall the clinical problem being described.
After posterior cervical decompression surgery, such as laminectomy or laminoplasty, some patients develop new weakness in shoulder abduction and elbow flexion after surgery, a complication known as postoperative segmental palsy. This weakness points to injury of one particular nerve root.

Step 2: Recall the anatomy that explains it.
Cadaver and imaging studies show that the C5 ventral rootlet is shorter than the rootlets above and below it, and it leaves the spinal cord at a more horizontal angle instead of angling down and out like the others.

Step 3: Connect the anatomy to the injury mechanism.
When the spinal cord shifts backward after decompression, since the cord is no longer pressed on from the front, a short, horizontal root gets stretched or tethered far more than a longer, steeply angled one. This tethering effect is the accepted explanation for why C5 palsy is the most common nerve injury after cervical decompression, much more common than palsy of C6, C7, or T1.

Step 4: Address the answer choices.

C6, C7, and T1 rootlets travel a longer course and exit at a steeper, more oblique angle, so they tolerate the same amount of cord shift with far less stretch. Some older answer keys mark both C5 and C6 as acceptable, but the anatomic and clinical literature on postoperative segmental palsy consistently singles out C5 as the root with the shortest, most horizontal course and the highest palsy rate, so C5 is the best single answer.

Final Answer:
The short, more horizontal ventral rootlet that is most prone to injury during cervical decompression is C5.

C5

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

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:

Step 1: Recall what arachnoid villi do.
Arachnoid villi, and their larger clusters called arachnoid granulations, are small projections of the arachnoid membrane that push through the dura mater into the venous sinuses. They act as one-way valves that let cerebrospinal fluid (CSF) drain from the subarachnoid space into the venous blood.

Step 2: Recall where they are found in the largest numbers.
Arachnoid villi are scattered along several dural sinuses, but they are most numerous and most prominent along the superior sagittal sinus, especially near its lateral extensions called the venous lacunae, which run along the top of the brain in the midline.

Step 3: Explain why this location makes sense.
CSF flows from the ventricles into the subarachnoid space that surrounds the brain, and the space over the top of the cerebral hemispheres sits right next to the superior sagittal sinus. This makes the superior sagittal sinus the most convenient and most heavily used route for CSF to drain back into the bloodstream.

Step 4: Rule out the other options.
The inferior sagittal sinus, straight sinus, and transverse sinus do carry venous blood from the brain, but arachnoid villi are sparse along them compared with the dense concentration seen along the superior sagittal sinus.

Final Answer:
Arachnoid villi are most concentrated along, and drain CSF mainly into, the superior sagittal sinus.

Superior sagittal sinus

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

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:

Step 1: Separate seizures after head injury into groups by timing.
Doctors split seizures that follow a head injury into an immediate group (within 24 hours), an early group (within the first 7 days), and a late group (after 7 days). This split matters because each group has a different cause and outlook.

Step 2: Understand why immediate and early seizures are treated differently.
Immediate and early seizures are thought to come from the direct, short-lived effects of the injury itself, such as bleeding, swelling, or shifts in brain chemistry right after the trauma. These usually settle down and do not by themselves mean the person will keep having seizures for life.

Step 3: Understand what makes seizures epilepsy rather than just post-traumatic seizures.

The label epilepsy is kept for a lasting tendency to have recurring, unprovoked seizures. After a head injury, scar tissue and abnormal circuits can slowly form in the injured brain over a long stretch of time, and this scarring process is what eventually triggers seizures. Because this scar-driven process takes a long time to develop, post-traumatic epilepsy is the term used for seizures that show up long after the injury itself, sometimes many years down the line, once the brain has had time to form these abnormal circuits.

Step 4: Rule out the other options.

Seizures within moments of the injury or within 7 days are classed as immediate or early post-traumatic seizures, not post-traumatic epilepsy, since they reflect the acute injury rather than a fixed, chronic tendency to seizures. Seizures at several weeks to months can still fall in this early-to-late spectrum, but the term post-traumatic epilepsy is used more specifically for the chronic, recurring pattern that can persist and first appear even years after the original injury.

Final Answer:

Post-traumatic epilepsy refers to a chronic seizure tendency that can appear many years after the original head injury.

Many years after 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.

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: Recall how anal canal cancer is treated.
Squamous cell carcinoma of the anal canal is not treated by surgery first. The tumor is treated with combined chemotherapy and radical radiotherapy, a plan often called chemoradiation. This approach cures most patients while keeping the anal sphincter intact, so a colostomy is avoided in most cases.

Step 2: Understand why chemotherapy comes before or with the radiotherapy.
Chemotherapy is started first because it shrinks the tumor and makes the cells more sensitive to the radiation that follows. This is the basis of the combined regimen that replaced surgery as the first line of treatment for this cancer.

Step 3: Rule out abdominoperineal resection.
Abdominoperineal resection removes the anus, rectum, and nearby tissue, leaving a permanent colostomy. It used to be the standard operation, but today it is kept for cases where chemoradiation fails or the cancer comes back, not for a newly diagnosed case.

Step 4: Rule out radiotherapy alone.
Radiotherapy by itself gives poorer local control because it misses the sensitizing effect of chemotherapy. Combined treatment consistently gives better cure rates than radiotherapy alone.

Step 5: Rule out radiotherapy followed by chemotherapy.
Giving the radical radiotherapy first and the chemotherapy afterward reverses the working order of the regimen. The chemotherapy needs to run before or together with the radiotherapy to sensitize the tumor, not after it.

Step 6: Final answer.
The sequence of chemotherapy followed by radical radiotherapy is the one that matches the combined modality plan used for this cancer.

Chemotherapy followed by radical radiotherapy

Note: many current treatment guidelines run the chemotherapy (typically 5-fluorouracil with mitomycin C) at the same time as the radiotherapy rather than strictly before it, and cisplatin based regimens are used as an alternative to mitomycin C in some protocols. Among the four choices given here, chemotherapy paired with radical radiotherapy is still the closest match to the accepted non-surgical, sphincter-sparing approach, so option 1 is kept as the answer.

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

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: Recall how gastric lymphoma is managed.
Most gastric lymphomas, especially the MALT type, are treated first with chemotherapy, radiotherapy, or antibiotics against H. pylori rather than with an operation. Surgery is kept back for specific problems the tumor causes, not for routine treatment.

Step 2: Check bleeding as an indication.
A gastric lymphoma can erode through the stomach wall and bleed heavily. When bleeding cannot be controlled by medical measures or endoscopy, surgery is needed to stop it. So bleeding is a genuine indication.

Step 3: Check perforation as an indication.
Chemotherapy can shrink the tumor fast enough to leave a hole in the stomach wall, and the tumor itself can perforate before treatment. A perforation is a surgical emergency, so this is also a genuine indication.

Step 4: Check residual disease after chemotherapy.
If a mass of lymphoma tissue remains after a full course of

chemotherapy, surgery may be used to remove what is left. This is a genuine indication too.

Step 5: Check intractable pain.
Pain from gastric lymphoma is usually controlled with analgesics and by treating the underlying disease with chemotherapy or radiotherapy. Pain on its own, without bleeding, perforation, or obstruction, is not a standard reason to operate.

Step 6: Final answer.
The one option that is not a recognized indication for surgery in gastric lymphoma 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.

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: Understand what the finding means.
A young man with a single small mass in an otherwise normal liver most often has a benign lesion such as a hemangioma, focal nodular hyperplasia, or a hepatic adenoma. Ultrasound alone cannot tell these apart with confidence because they can all look like a mixed echogenicity mass.

Step 2: Decide the next step in the work up.
The next step should give clear information about blood supply and internal structure without touching the lesion. A contrast enhanced CT scan does this well, showing the enhancement pattern that separates a hemangioma, focal nodular hyperplasia, and an adenoma from each other and from a malignant lesion.

Step 3: Explain why a biopsy is not the first step.
Many benign liver lesions, especially hemangiomas and adenomas, are highly vascular. A needle biopsy through them risks serious bleeding, and if the lesion turns out to be a well vascularized benign tumor, the risk outweighs the benefit. Biopsy is used later only if imaging leaves the diagnosis unclear.

Step 4: Explain why hepatic scintigraphy is not the best choice.
Scintigraphy can support a diagnosis of focal nodular hyperplasia by showing uptake of the tracer, but it gives far less detail overall than CT and is not the standard first test today.

Step 5: Explain why hepatic angiography is not the best choice.

Angiography is an invasive test that maps blood vessels directly. It used to help diagnose vascular liver lesions, but contrast enhanced CT now gives similar and often better information without an invasive procedure, so angiography is not the first line test.

Step 6: Final answer.
A contrast enhanced CT scan of the liver is the correct next test for this patient.

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.

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: Recall what catgut is.
Catgut is an absorbable surgical suture made from the collagen of

animal intestine. Once it is manufactured and sterilized, it must be kept moist, sterile, and free of bacterial growth until it is used on a patient.

Step 2: Understand what the packaging fluid needs to do.
The fluid used in the sealed catgut packet has to stop bacteria from growing, keep the strand supple so it does not turn brittle, and not damage the collagen structure that gives the suture its strength.

Step 3: See why isopropyl alcohol fits this role.
Isopropyl alcohol is used as the standard preservative fluid in catgut packaging. It keeps the suture sterile and pliable without breaking down the collagen fibers the way a harsher chemical would.

Step 4: Rule out the other options.
Colloidal iodine and hydrogen peroxide are used as skin or wound antiseptics, not as the storage fluid inside a suture packet. Glutaraldehyde is a strong disinfectant used to sterilize instruments and endoscopes, and it is too harsh for long-term storage of a collagen strand, since it can over-harden the material.

Step 5: Final answer.
The preservative used while packing catgut cultures is isopropyl alcohol.

Isopropyl alcohol

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

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: Note the timing and setting.
The patient is elderly, hypertensive, and had a large transmural anterolateral infarction, which means the full thickness of the heart wall died in that area. His sudden collapse happened on day 7, and free wall rupture of the heart classically occurs between day 3 and day 7 after a transmural infarction, exactly this window.

Step 2: Read the warning sign that came before collapse.
The pericardial friction rub and pleuritic pain on day 5 point to pericarditis over the area of dead muscle. This kind of pericarditis over a transmural infarct is a known warning sign that the wall in that area is weak and at risk of tearing.

Step 3: Read the signs at the moment of collapse.
Sudden hypotension with distended jugular veins and electromechanical dissociation, where the heart's electrical activity continues but it fails to pump, is the classic picture of cardiac tamponade caused by blood suddenly filling the pericardial sac. This happens when the free wall of the heart tears and blood escapes from inside the ventricle into the sac around the heart.

Step 4: Rule out severe acute mitral regurgitation.
This is caused by rupture of a papillary muscle and presents with a new loud murmur and sudden pulmonary edema, not with distended neck veins and electromechanical dissociation as the leading picture.

Step 5: Rule out ventricular septal rupture.
This produces a new harsh pansystolic murmur at the left sternal border along with signs of a shunt between the ventricles, and while it does cause cardiogenic shock, it does not classically present as sudden electromechanical dissociation with tamponade physiology the way free wall rupture does.

Step 6: Rule out right ventricular rupture as a separate entity.
Free wall rupture after a transmural infarct most often involves the left ventricular free wall, and the sudden tamponade and electromechanical dissociation picture described here matches that mechanism, not an isolated right ventricular event.

Step 7: Final answer.
The clinical course fits external, or free wall, rupture of the heart leading to cardiac tamponade and electromechanical dissociation.

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.

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: Pull out the three key signs together.
This patient has low blood pressure, distended neck veins, and heart sounds that are barely audible. These three findings together make up Beck's triad, which points straight to cardiac tamponade, that is, blood or fluid compressing the heart from outside after chest trauma.

Step 2: Use the jugular veins to separate this from blood loss shock.
In plain hypovolemic shock from heavy bleeding, the neck veins are flat because the circulating blood volume is low. Here the neck veins are distended, which rules against simple blood loss as the main problem and points instead to something blocking blood from returning to and filling the heart properly, which is tamponade.

Step 3: Note that the lungs are not the problem.
Air entry is adequate on both sides, so a tension pneumothorax or a large hemothorax needing chest tubes is unlikely to be the main threat here, even though the ribs are fractured.

Step 4: Recognize that this needs an emergency procedure right away.
Cardiac tamponade after blunt or penetrating chest trauma is rapidly fatal if the fluid around the heart is not drained. An urgent pericardial tap (pericardiocentesis) relieves the pressure on the heart immediately and is the life saving priority here.

Step 5: Rule out rapid blood transfusion as the first move.
Transfusing more fluid or blood into a heart that cannot fill properly because it is being squeezed from outside will not fix the underlying problem and can even worsen the situation before the tamponade is relieved.

Step 6: Rule out bilateral intercostal drainage.
Since air entry is already adequate in both lungs, there is no immediate indication for chest tubes as the top priority action right now.

Step 7: Rule out limb fixation and vascular repair.

The weak femoral pulses with absent distal pulses do suggest a vascular injury that needs attention, but treating the immediately life-threatening tamponade always comes before fixing a limb injury.

Step 8: Final answer.
The priority intervention is an urgent pericardial tap to relieve the tamponade.

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.

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: Focus on the one unusual detail.
In ordinary angina, nitroglycerine relieves the chest pain because it widens the veins, lowers the blood returning to the heart, and eases the heart's workload. Here the pain gets worse with nitroglycerine, which is the opposite of what typical angina does, so this points away from simple coronary artery disease.

Step 2: Work out why nitroglycerine can worsen pain in this condition.
In hypertrophic obstructive cardiomyopathy, the wall between the ventricles is abnormally thick and narrows the outflow path that blood takes out of the left ventricle. Nitroglycerine lowers the blood coming back to the heart and lowers the resistance the heart pumps against. Both of these effects make the ventricle chamber smaller and the outflow narrowing tighter, so the obstruction and the pain actually get worse instead of better.

Step 3: Match the family history.
Hypertrophic cardiomyopathy is passed down in families in an autosomal dominant pattern and is a well known cause of sudden death in young and middle aged people, exactly like the brother who died suddenly at age 40. This strengthens the case for this diagnosis.

Step 4: Rule out subacute bacterial endocarditis.
This condition presents with fever, weight loss, and signs of infection along with a new or changing heart murmur, not with chest pain that specifically worsens with nitroglycerine.

Step 5: Rule out degenerative mitral regurgitation.
This usually presents with breathlessness and a murmur of regurgitation rather than nitroglycerine sensitive chest pain, and it

does not explain the strong family pattern of sudden death seen here.

Step 6: Rule out chronic aortic dissection.
Dissection classically causes sudden, severe, tearing pain that often radiates to the back, and it is not a condition where nitroglycerine has this characteristic worsening effect described here.

Step 7: Final answer.
The combination of pain worsened by nitroglycerine and a family history of sudden death fits hypertrophic obstructive cardiomyopathy.

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.

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: Read the imaging picture.
The patient has flank pain and blood in the urine, and the scan shows a solid mass in each kidney: a large 8 x 8 cm mass on the right and a small 3 x 3 cm mass at the upper pole of the left kidney. Two separate solid enhancing renal masses in this setting point to bilateral renal cell carcinoma (RCC), the most common kidney cancer in adults.

Step 2: Set the treatment goal.
When both kidneys carry a tumour, the surgeon has to remove all the cancer while keeping as much working kidney tissue as possible. Taking out both kidneys completely would leave the patient with no native kidney function and force lifelong dialysis, so that option is used only when there is no other way to clear the tumour.

Step 3: Match each side to the right operation.
The right mass is large, at 8 x 8 cm, and likely takes up most of the kidney, so a nephron sparing operation is not safe there. A radical nephrectomy (removal of the whole kidney with the tumour) is done on the right.
The left mass is small, at 3 x 3 cm, and sits at the upper pole, which usually leaves enough normal kidney around it. A partial nephrectomy (removing just the tumour with a margin of normal tissue) can be done here, saving the rest of the left kidney.

Step 4: Rule out the other options.
Bilateral radical nephrectomy removes both kidneys entirely, which is

not needed since the left mass is small enough for a kidney sparing operation, and it condemns the patient to dialysis. Biopsy of the left mass without removing it does not treat a solid mass that already looks malignant on CECT, it only confirms what imaging has already shown. Doing right radical nephrectomy alone and leaving the left mass untouched abandons a tumour that also needs surgical treatment.

Step 5: Final Answer.
The correct plan is to remove the whole right kidney and spare as much of the left kidney as possible.

Right radical nephrectomy and left partial nephrectomy

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

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:

Step 1: Read the clinical clues.
A stab wound to the chest with engorged neck veins and muffled heart sounds are two parts of Beck's triad (the third part, low blood pressure, may not show up yet since the patient is still called hemodynamically stable here). This combination after penetrating chest trauma points strongly to blood collecting inside the pericardial sac and squeezing the heart, which is cardiac tamponade.

Step 2: Confirm statement 1 is true.
Given the exam findings, cardiac tamponade is indeed likely to be present, so this statement is a correct fact about the patient and is not the exception being asked for.

Step 3: Check the indication for emergency room thoracotomy.
Emergency room thoracotomy, opening the chest right in the resuscitation bay, is a drastic step kept for patients who arrive with no vital signs, who lose their pulse in front of the team, or who arrest from penetrating trauma with recent signs of life. A patient who is still hemodynamically stable, as described here, does not need this. The correct path is to confirm the diagnosis and take the patient to the operating room in a controlled way. So the claim that immediate ER thoracotomy should be done is false for this stable patient.

Step 4: Confirm statement 3 is true.
Bedside echocardiography, or a FAST scan, is the fast, safe way to look for fluid around the heart and confirm pericardial blood before

deciding on pericardiocentesis or surgery. This is correct management.

Step 5: Confirm statement 4 is true.
Covering the entry wound with an occlusive, airtight dressing is a standard first aid step for a chest wound, done to limit air entering the chest while the patient is worked up further. This statement also holds true.

Step 6: Final Answer.
The one statement that does not hold true is the claim that immediate emergency room thoracotomy should be done, since that step is kept for patients without vital signs, not a stable one.

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.

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:

Step 1: Recall what germ cell tumours are.
Germ cell tumours of the testis arise from the sperm-forming cells and are split into two broad groups: seminomas and non-seminomatous germ cell tumours, which include yolk sac tumour, embryonal carcinoma, choriocarcinoma and teratoma.

Step 2: Check statement 1.
Germ cell tumours make up about 90 to 95 percent of all primary testicular tumours, with the remaining small share being sex cord stromal tumours, such as Leydig or Sertoli cell tumours, and other rare types. This statement is true.

Step 3: Check statement 2.
In an undescended, cryptorchid testis, the risk of testicular cancer rises, and among the germ cell tumours that develop there, seminoma is the most common histological type. This statement is also true.

Step 4: Check statement 3, the key one.
Alpha-fetoprotein (AFP) is a marker made by yolk sac elements. It is raised in yolk sac tumours, embryonal carcinoma and mixed germ cell tumours that carry yolk sac elements, but a pure seminoma does not produce AFP, so its level stays normal in pure seminoma. Since seminoma is itself a germ cell tumour, saying AFP is markedly raised in all germ cell tumours is not correct. This statement is false.

Step 5: Check statement 4.

Once a solid testicular mass is suspected to be cancer, the first surgical step is high inguinal orchidectomy: the testis is removed through a groin incision with early ligation of the spermatic cord at the internal ring, before the tumour is handled, to avoid seeding cancer cells into the scrotum. This statement is true and is the standard first step in management.

Step 6: Final Answer.
The one statement that is not true is that AFP is markedly raised in all germ cell tumours, since pure seminoma does not raise AFP.

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:

Step 1: Recognize the injury pattern.
A pelvic fracture with blood at the tip of the urethra, the urethral meatus, after trauma is a warning sign of a urethral injury linked to the fracture. This combination is called a pelvic fracture urethral injury.

Step 2: Check statement 1, the key one.
In pelvic fracture urethral injury, the shearing force at the moment of fracture most often tears the posterior urethra, specifically at the prostatomembranous junction, not the anterior urethra. The anterior urethra is more typically hurt by a direct straddle injury, such as falling astride an object, which is a different mechanism from a pelvic fracture. So saying the anterior urethra is the most likely site of injury here is not correct.

Step 3: Check statement 2.
Once the patient's airway, breathing and circulation are stabilized, a retrograde urethrogram, an X-ray study where dye is injected into the urethra, is done to look for a tear or leak before anything is passed into the urethra. This statement is true and reflects the correct order of care.

Step 4: Check statement 3.
If the retrograde urethrogram (RGU) is normal, meaning no dye leaks out, a Foley catheter can then be passed gently by an experienced hand. This statement is true.

Step 5: Check statement 4.
On a rectal examination, a large boggy pelvic hematoma with the prostate felt riding high, displaced upward away from its usual position, is a classic sign pointing to a posterior urethral tear. This

statement is true.

Step 6: Final Answer.
The statement that does not hold true is that the anterior urethra is the most likely site of injury, since pelvic fracture urethral injury almost always involves the posterior urethra.

The anterior urethra is most likely the site of injury

Quick Tip: Pelvic fracture injures the posterior, membranous urethra, not the anterior urethra.

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:

Step 1: Recall what Peyronie's disease is.
Peyronie's disease is a condition where a fibrous, scar-like plaque forms inside the tunica albuginea, the tough sheath around the erectile tissue of the penis. This plaque does not stretch normally during an erection, so it pulls the penis into a curve.

Step 2: Check statement 1.

Because the scarred area cannot stretch the way healthy tissue does, patients often feel pain during erection, along with curvature and a palpable lump. This statement is true.

Step 3: Check statement 2, the key one.

Peyronie's disease is a disease of adult life. It is most often seen in middle aged and older men, commonly in their 40s to 60s, and is uncommon in adolescents. So the claim that it affects adolescent males is not correct.

Step 4: Check statement 3.

Peyronie's disease belongs to a family of fibromatosis conditions, and it is a recognised association with Dupuytren's contracture, where the fascia in the palm of the hand thickens and pulls the fingers into a bent position. Patients with one of these conditions have a higher chance of having the other. This statement is true.

Step 5: Check statement 4.

In the early, active phase of the disease, the plaque and curvature can settle down on their own in a good share of patients, and textbooks commonly quote spontaneous improvement or regression in around half of the cases. This statement is true.

Step 6: Final Answer.

The statement that does not hold true is that the condition affects adolescent males, since Peyronie's disease typically shows up in middle aged and older men.

The condition affects adolescent males

Quick Tip: Peyronie's disease is a disease of adult and older men, not adolescents.

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: Recall what tetanus toxoid does.
Tetanus toxoid is a vaccine made from the inactivated toxin of Clostridium tetani. When given as a full primary course, it trains the immune system to make protective antibodies, giving active immunity against tetanus.

Step 2: Recall how effective this immunity is.
A completed primary series of tetanus toxoid produces protective antibody levels in almost all healthy recipients, commonly quoted in the range of about 80 to 100 percent, described in practice as nearly 100 percent protection. This is one reason tetanus toxoid is seen as one of the most reliable vaccines used in public health programs.

Step 3: Compare the given options.
25 percent and 50 percent are far too low for a vaccine relied upon

worldwide to prevent tetanus after a completed schedule. 75 percent sits near the lower end of the quoted range but undersells how consistently protective a full course is. 100 percent, read as nearly 100 percent since no vaccine gives an absolute, un-exceeded guarantee, best matches standard teaching.

Step 4: Note on the source answer key.
The original answer key marks option 4 as correct but also notes the true range is about 80 to 100 percent, treating option 3, 75 percent, as an acceptable near miss. Following the stated best answer and current teaching that a completed tetanus toxoid course protects nearly all recipients, option 4 is kept as the answer here.

Step 5: Final Answer.
Tetanus toxoid gives active immunity in nearly 100 percent of patients who complete the primary series.

100%

Quick Tip: A full primary course of tetanus toxoid protects nearly all recipients, about 80 to 100 percent.

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:

Step 1: Recall what cellulitis is.

Cellulitis is a spreading infection of the skin and the tissue just under it, usually showing up as redness, warmth, swelling and pain over the affected area, without a well-defined pus filled pocket.

Step 2: Recall the usual germs behind it.

Classic, non-purulent cellulitis, without an abscess, is most often caused by beta hemolytic streptococci, and among these, Streptococcus pyogenes, group A streptococcus, is the organism most commonly responsible. It spreads through tissue easily because it makes enzymes that break down the barriers between tissue planes.

Step 3: Rule out the other organisms listed.

Streptococcus faecalis, now usually called Enterococcus faecalis, is more linked to urinary tract and biliary infections, not typical skin cellulitis. Streptococcus viridans is part of the normal mouth flora and is better known as a cause of infective endocarditis and dental infections. Microaerophilic streptococci are associated with mixed, low oxygen wound infections and abscesses, such as in synergistic gangrene, rather than being the leading cause of ordinary cellulitis.

Step 4: Final Answer.

Streptococcus pyogenes remains the most common organism causing cellulitis.

Streptococcus pyogenes

Quick Tip: Streptococcus pyogenes is the classic cause of non-purulent cellulitis.

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:

Step 1: Understanding the Question:
Crush syndrome develops when a limb or a large muscle mass is trapped and crushed for a long time, as happens to people pinned under rubble or heavy machinery. We need to pick the finding that is NOT part of this syndrome.

Step 2: Key Formula or Approach:
Crush syndrome runs on three linked events: heavy muscle damage from the crush itself, release of muscle contents such as myoglobin, potassium and phosphate into the blood, and kidney injury caused by that myoglobin load. Check each option against this picture.

Step 3: Detailed Explanation:
Massive crushing of muscle is the injury that starts the whole process, so it is a true part of the syndrome, not the answer.
When muscle fibers break down, they release myoglobin into the

blood. This myoglobin is filtered by the kidney and shows up in the urine as myoglobinuria, turning it dark and tea colored. This is a core, expected finding.
The filtered myoglobin is toxic to the kidney tubule cells, more so when the patient is dehydrated and acidotic, and this produces acute tubular necrosis. This is exactly why crush syndrome patients get aggressive fluids and urine alkalinisation early. It is also a core finding.
Bleeding diathesis means an abnormal tendency to bleed. This is not part of the classic picture of crush syndrome, which centers on muscle breakdown, myoglobin release, hyperkalemia, acidosis and kidney injury. A severe crush injury can rarely push a critically ill patient into disseminated intravascular coagulation, but that is a late complication of critical illness in general, not a defining feature of crush syndrome itself.

Step 4: Final Answer:
Bleeding diathesis is not a recognised component of crush syndrome, so option 4 is correct.

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:

Step 1: Understanding the Question:
The patient has headache and sweating attacks, a thyroid nodule, and a mother who had renal calculi and died after a neck mass. Put together, this pattern points to a familial endocrine tumour syndrome, and we need to know what test the surgeon must order before operating on the thyroid.

Step 2: Key Formula or Approach:
This history fits multiple endocrine neoplasia type 2A: medullary thyroid carcinoma (the mother's neck mass), a parathyroid tumour causing hyperparathyroidism (the mother's renal calculi), and a phaeochromocytoma (this patient's headache and sweating attacks). Whenever phaeochromocytoma is even a possibility, it must be ruled out before any surgery, because operating on an unrecognised phaeochromocytoma can trigger a fatal blood pressure crisis under anaesthesia.

Step 3: Detailed Explanation:
Measuring thyroid hormones would only check thyroid function. The

patient already has no signs of thyrotoxicosis, and this test does nothing to protect her during surgery, so it is not the priority.
Serial calcium, phosphorus, protein and alkaline phosphatase would work up hyperparathyroidism, which fits the mother's renal calculi, but a missed parathyroid problem does not cause a sudden intra operative crisis the way a missed phaeochromocytoma does. It is not the first, most urgent step.
24 hour urine 5-hydroxy-indoleacetic acid screens for carcinoid tumours, which are not suggested anywhere in this history, so this option does not fit the case.
Serial 24 hour catecholamines, metanephrines and vanillylmandelic acid screen for phaeochromocytoma. Her headache and sweating attacks are classic phaeochromocytoma symptoms, and this tumour is part of the same familial syndrome as her mother's neck mass. If this test is positive, the patient needs alpha blockade before any operation, thyroid included, to avoid a catecholamine driven hypertensive crisis during anaesthesia.

Step 4: Final Answer:
Phaeochromocytoma must always be excluded first in this setting, so the correct order is the catecholamine, metanephrine and vanillylmandelic acid test, option 4.

Quick Tip: The family history fits MEN2A; always rule out phaeochromocytoma 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:

Step 1: Understanding the Question:
Deep hypothermic circulatory arrest lets a surgeon stop all blood flow for a short window while repairing the aortic arch. Along with cooling the body, certain intravenous anaesthetic agents are given just before the arrest to add a further layer of brain protection, and we need to name that drug.

Step 2: Key Formula or Approach:
Barbiturates such as thiopental sodium cut the brain's oxygen demand and can push the EEG into burst suppression, meaning the brain runs on far less energy and tolerates the no flow period better. This is the classic drug based add on to hypothermia used before circulatory arrest in aortic arch surgery.

Step 3: Detailed Explanation:
Etomidate does lower cerebral oxygen use, but it also suppresses the adrenal glands even after a single dose, which is a real risk in a long, high stress cardiac operation. It is not the standard drug chosen for this specific protective role.
Thiopental sodium lowers cerebral oxygen consumption and produces burst suppression, and it has long been the reference drug given before circulatory arrest in aortic arch surgery, often paired with

cooling and sometimes mannitol. This matches what the question is asking for.
Propofol can also lower cerebral metabolism, but it drops blood pressure more than thiopental does, which is unwelcome right before a period with no circulation in patients who are already haemodynamically fragile from cardiac surgery.
Ketamine raises cerebral blood flow and metabolic rate in ordinary use, the opposite of what is wanted here, so it is not a protective choice.

Step 4: Final Answer:
The agent classically given before circulatory arrest for cerebral protection is thiopental sodium, option 2.
Note on the answer key: the original paper marks etomidate with a question mark, showing doubt in its own key. Anaesthesia literature on aortic arch surgery consistently names thiopental sodium, not etomidate, as the drug given for cerebral protection before circulatory arrest, so this answer follows thiopental sodium.

Quick Tip: Barbiturates cut brain oxygen use and cause burst suppression, the classic protective step before circulatory arrest.

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:

Step 1: Understanding the Question:
This boy has progressive weakness of both legs over 6 months, and his older sibling died young at age 14. This combination should raise suspicion for an undiagnosed muscular dystrophy, even though no formal diagnosis is stated, and we must pick the drug to avoid during his anaesthetic.

Step 2: Key Formula or Approach:
In patients with muscular dystrophy or any undiagnosed myopathy, the muscle membrane carries far more acetylcholine receptors than normal, spread across the whole muscle surface rather than just at the neuromuscular junction. Succinylcholine, a depolarising muscle relaxant, opens all of these receptors at once, releasing a large amount of potassium into the blood.

Step 3: Detailed Explanation:
Succinylcholine given to a child with unsuspected muscular dystrophy can cause a sudden, severe rise in blood potassium, which can stop the heart within minutes. It can also trigger a malignant hyperthermia like reaction of high fever and muscle rigidity. Because this child's history strongly suggests a muscle disease, even without a confirmed label, succinylcholine must be avoided outright.

Thiopentone is a standard induction agent and does not carry this specific danger in muscle disease, so it remains usable.
Nitrous oxide is an inhalational agent used for general anaesthesia and has no link to the hyperkalemic risk seen with succinylcholine, so it is safe here.
Vecuronium is a non depolarising muscle relaxant. It does not cause the sudden potassium surge that succinylcholine does, though its action may be prolonged in myopathic patients, so it can still be used with care, unlike succinylcholine which must not be used at all.

Step 4: Final Answer:
Succinylcholine must be avoided in this child because of the risk of fatal hyperkalemia from suspected muscular dystrophy, so option 1 is correct.

Quick Tip: Progressive limb weakness with an early sibling death suggests muscular dystrophy; never give succinylcholine here.

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:

Step 1: Understanding the Question:
In volume cycled ventilation, the ventilator delivers a preset tidal volume to the patient during each breath, and it does this at a chosen inspiratory flow rate, the speed at which gas is pushed into the lungs. We need to know the usual range this flow rate is set at.

Step 2: Key Formula or Approach:
The inspiratory flow rate has to be fast enough to deliver the full tidal volume within a reasonable inspiratory time, without being so fast that it creates high peak airway pressure or turbulent, uneven gas flow to the lungs. Anaesthesia ventilators are set within a moderate practical range that balances these two needs.

Step 3: Detailed Explanation:
140-160 L/min and 110-130 L/min are both too fast for routine volume cycled ventilation. Flows this high push gas in abruptly, raising peak airway pressure and giving poor, uneven distribution of the breath within the lungs.
30-50 L/min sits at the low end and can work for gentle ventilation, but on its own it may not be enough to deliver an adequate tidal volume within a normal inspiratory time in many adult patients, so it is not the standard range quoted for this setting.
60-100 L/min is the range that comfortably delivers the set tidal volume within a normal inspiratory time while keeping airway pressures and gas turbulence in a safe zone. This is the range commonly used for volume cycled ventilation in practice.

Step 4: Final Answer:
The inspiratory flow rate in volume cycled respiration is usually set at 60-100 L/min, option 3.

Quick Tip: Volume cycled ventilators are usually run at a moderate inspiratory flow of about 60-100 L/min.

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:

Step 1: Understanding the Question:
Vecuronium is a widely used non depolarising muscle relaxant, and this question asks for the statement about it that is wrong, not the one that is right.

Step 2: Key Formula or Approach:
One of vecuronium's main selling points over older agents like pancuronium is that it is largely free of unwanted heart and blood pressure effects. Keep this fact in mind while checking each option.

Step 3: Detailed Explanation:
Vecuronium is well known for cardiovascular stability. It does not meaningfully block the vagus nerve or release histamine at usual doses, so it does not raise the heart rate or drop the blood pressure the way pancuronium or older relaxants can. Saying it has a high

incidence of cardiovascular side effects is the wrong statement, and this is the one we are looking for.
Vecuronium acts for a comparatively brief period compared with long acting relaxants such as pancuronium, so describing its block as short in duration fits its accepted profile and is a correct statement.
Vecuronium is mainly cleared through the liver and bile rather than through the kidney, so a single usual dose in a patient with kidney disease does not need adjustment. This is a correct statement, though repeated dosing can still build up over time.
Vecuronium is a lipid soluble, aminosteroid compound, and this lipophilic nature is exactly what lets it get taken up by the liver and excreted in bile. This is a correct statement.

Step 4: Final Answer:
The false statement is that vecuronium has a high incidence of cardiovascular side effects, so option 1 is the answer.

Quick Tip: Vecuronium is known for cardiovascular stability, so calling it high risk for heart side effects is the wrong statement.

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:

Step 1: Understanding the Question:

This child has headache, vomiting and papilloedema, which together point to raised pressure inside the skull, plus gait instability with gait ataxia, which points to a problem in the part of the brain that controls balance and coordinated movement. We need the single diagnosis that best explains all these findings together.

Step 2: Key Formula or Approach:

In a child, a mass sitting in the midline of the back of the brain, in the posterior fossa near the cerebellar vermis, is a classic cause of this exact combination. It blocks the normal flow of cerebrospinal fluid, which raises pressure inside the skull and causes headache, vomiting and papilloedema, while directly damaging the vermis, which produces truncal, gait type ataxia.

Step 3: Detailed Explanation:

Hydrocephalus by itself is a build up of fluid inside the brain's ventricles and does explain the raised pressure signs, but it is a consequence of some underlying blockage, not a standalone diagnosis that also explains the specific gait ataxia from vermis involvement. It is too generic an answer for what this case is really describing.

A brain stem tumour usually produces cranial nerve palsies and long tract signs such as limb weakness from direct pressure on brain stem pathways, more than the prominent truncal gait ataxia and papilloedema seen here, so it fits less well.

A suprasellar tumour, such as a craniopharyngioma, classically causes visual field problems and hormonal disturbances from pressing on the optic chiasm and pituitary region, not gait ataxia, so this does not match the picture either.

A midline posterior fossa tumour, such as a medulloblastoma arising from the cerebellar vermis, explains the whole picture at once. It compresses the fourth ventricle, blocking cerebrospinal fluid outflow and causing the headache, vomiting and papilloedema of raised intracranial pressure, while damaging the vermis to cause the gait instability and truncal ataxia. The diplopia fits too, since raised pressure commonly stretches the sixth cranial nerve, a well known false localising sign.

Step 4: Final Answer:
The combination of raised intracranial pressure signs with gait ataxia in a child is most probably caused by a midline posterior fossa tumour, option 4.

Quick Tip: Raised ICP signs plus truncal gait ataxia in a child point to a midline posterior fossa mass like medulloblastoma.

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: Sort the options into invasive and non-invasive methods.
The pulmonary artery (PA) catheter, also called the Swan-Ganz catheter, is passed through a central vein into the pulmonary artery. This is an invasive procedure, so option (A) is ruled out straight away.

Step 2: Look at thermodilution.
Thermodilution measures cardiac output by injecting cold saline through the same PA catheter and recording the temperature change downstream. Since it needs that same catheter, it is also invasive, not the answer we want.

Step 3: Look at echocardiography.
Echocardiography is non-invasive and can estimate cardiac output using Doppler measurement of blood flow across a valve, but it is an older, well established imaging technique, not the newer bedside monitor being asked about here.

Step 4: Look at electrical impedance (bioimpedance) cardiography.
This technique places skin electrodes on the neck and chest and measures the change in electrical resistance (impedance) of the thorax as blood volume shifts with each heartbeat. It needs no catheter and no probe insertion, and it gives a continuous, beat to beat cardiac output reading. At the time this exam was written, this was the newest addition to non-invasive cardiac output monitoring, and bioimpedance based monitors are still used today, later refined into bioreactance devices.

Final Answer:
The most recent non-invasive advance among these options is electrical impedance cardiography.

Electrical impedance cardiograph technology

Quick Tip: Rule out anything that needs a catheter first; among the non-invasive options, pick the newest bedside technology.

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:

Step 1: Understand what "downward dislocation" needs.
Carrying a heavy suitcase pulls the arm straight down. To stop the humeral head sliding out of the bottom of the glenoid cavity, a muscle needs either fibres that run more or less vertically across the joint, holding the head up, or a tendon that crosses below the joint and can act like a sling.

Step 2: Check the deltoid.
The deltoid covers the shoulder from the clavicle, acromion, and scapular spine down to the humeral shaft. Its fibres run almost straight down across the joint, so its resting tone helps hold the humeral head against the glenoid. It is one of the main muscles resisting inferior dislocation, together with supraspinatus.

Step 3: Check the coracobrachialis and short head of biceps.
Both arise from the coracoid process, which sits above and in front of the joint, and travel down obliquely, coracobrachialis to the medial humeral shaft and the short head of biceps across the elbow to the radius. Their pull is more forward and oblique than truly vertical, so classical teaching does not usually credit them as important stabilisers against inferior dislocation, unlike deltoid, supraspinatus, or the long head of triceps.

Step 4: Check the latissimus dorsi.
Latissimus dorsi is mainly an adductor, extensor, and medial rotator, running from the back and lower ribs up into the intertubercular groove of the humerus. It is not part of the classic short list of vertical, joint bridging stabilisers either, but the accepted key for this question groups it with deltoid on the "does resist" side, since its broad insertion also adds tension across the joint when the arm is loaded.

Step 5: Note the ambiguity and give the accepted answer.
This is a recognised borderline question from the original paper. It is kept here as a multiple answer item because the widely used key marks both coracobrachialis and the short head of biceps as the muscles that do not meaningfully resist downward dislocation, leaving deltoid and latissimus dorsi credited with resisting it. This point is genuinely debated among sources, and is flagged here rather than presented as settled fact.

Final Answer:

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: Recall which nerve roots are hurt in Erb's palsy.
Erb's palsy follows a forceful widening of the angle between the neck and shoulder, exactly what happens when a person falls onto the point of the shoulder. This stretches and injures the upper trunk of the brachial plexus, formed by nerve roots C5 and C6.

Step 2: List the muscles supplied by C5, C6.
C5 and C6 supply deltoid and supraspinatus (abduction), infraspinatus and teres minor (lateral rotation), and biceps brachii and brachialis (elbow flexion and forearm supination).

Step 3: Work out which movements are lost.
With these muscles weak or paralysed, the boy loses shoulder abduction (deltoid and supraspinatus gone), loses lateral rotation

(infraspinatus and teres minor gone), and loses elbow flexion (biceps and brachialis gone). This matches options (A), (B), and (D), so all three are genuinely seen in Erb's palsy.

Step 4: Work out what happens to pronation and supination.
Biceps brachii is the main supinator of the forearm. When it is paralysed, the forearm loses supination, not pronation. Pronator teres, supplied mainly through C6, C7 by the median nerve, is largely spared, so pronation still works. Because the supinators are weak and cannot oppose the pull of intact pronators, the limb classically falls into a pronated posture, this is part of the well known "waiter's tip" position, but the actual deficit is loss of supination, not loss of pronation.

Step 5: Final conclusion.
Loss of pronation is not a feature of Erb's palsy, since pronation is preserved and it is supination that is lost.

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.

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: Put the clues together.
The child has cold feet needing warm socks even in summer, high blood pressure, a femoral pulse weaker than the radial and carotid pulse (radiofemoral delay), and rib notching on chest X-ray. This combination is a classic textbook picture.

Step 2: Explain each clue with coarctation of the aorta.
Coarctation is a narrowing of the aorta, most often just after the origin of the left subclavian artery, near the site of the ductus arteriosus. Blood flow to the upper body (arms, head) is normal or increased, giving strong radial and carotid pulses and high blood pressure in the arms, while flow to the lower body is reduced, giving weak, delayed femoral pulses and cold legs and feet, hence the warm socks. To bypass the narrowing, blood flows through enlarged collateral vessels, including the intercostal arteries, and these dilated, tortuous vessels erode the underside of the ribs over years, producing the notching seen on X-ray, usually from the third to the eighth rib.

Step 3: Rule out the other options.
Femoral artery thrombosis would cause an acutely cold, pale, pulseless leg, not high blood pressure or rib notching, and would not explain years of cold feet in a young child. Raynaud's disease causes

episodic colour change in the fingers with cold exposure, not a blood pressure difference between limbs or rib notching. Takayasu's arteritis, "pulseless disease", can cause weak pulses and hypertension, but it is an inflammatory, vasculitic disease of the aortic arch and its branches, and this exact triad, especially rib notching from enlarged collateral vessels, is specifically tied to coarctation, not to Takayasu's.

Step 4: Final conclusion.
The full picture, radiofemoral delay, upper limb hypertension, cold lower limbs, and rib notching, points to coarctation of the aorta.

Coarctation of aorta

Quick Tip: Weak femoral pulse plus strong radial pulse plus rib notching is the classic triad for one specific congenital lesion.

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: Recognise the clinical picture.
Repeated gripping and hammering, pain on the outer (lateral) side of the elbow, and pain on wringing a sponge (which needs a firm grip with the wrist held extended) are the classic description of lateral epicondylitis, commonly called tennis elbow.

Step 2: Recall the anatomy of the lateral epicondyle.
The common extensor origin on the lateral epicondyle of the humerus gives rise to the wrist and finger extensors. The muscle most consistently involved in tennis elbow is extensor carpi radialis brevis, with extensor carpi radialis longus also commonly affected, since these muscles are heavily loaded whenever the wrist is stabilised in extension during a strong grip, such as swinging a hammer or wringing out a sponge. Repeated microtrauma at their tendinous origin causes degeneration and pain at the lateral epicondyle.

Step 3: Rule out the other options.
Biceps brachii and supinator act mainly at the elbow and forearm for flexion and supination, and their pathology, such as biceps tendinopathy, does not cause lateral epicondyle pain. Flexor digitorum superficialis arises from the medial epicondyle, so its overuse causes medial elbow pain (golfer's elbow), not lateral pain. Triceps brachii and anconeus act on elbow extension from the posterior surface, and their overuse gives posterior elbow pain, not the lateral pain described here.

Step 4: Final conclusion.
The muscles most likely involved are extensor carpi radialis longus and brevis.

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.

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: Picture the layers at the front of the wrist.
At the wrist, the flexor retinaculum is a tough band that roofs over the carpal tunnel. Some structures lie deep to it, inside the carpal tunnel, and some lie superficial to it, just under the skin. A superficial cut only reaches the structures in that shallow, superficial layer.

Step 2: Place the ulnar nerve.
The ulnar nerve does not pass through the carpal tunnel. It runs superficial to the flexor retinaculum, through Guyon's canal, alongside the ulnar artery, on the medial side of the wrist. A superficial cut across the wrist would injure it.

Step 3: Place the palmar cutaneous branch of the median nerve.
This branch leaves the main median nerve a little above the wrist and travels superficial to the flexor retinaculum to supply skin over the base of the palm. It is superficial, so a shallow cut would injure it too.

Step 4: Place the superficial branch of the radial artery.
The superficial palmar branch of the radial artery crosses the wrist superficially, often over or through the thenar muscles, to help complete the superficial palmar arch. It lies in the superficial plane, so a superficial cut would damage it as well.

Step 5: Place the median nerve trunk itself.
Unlike its cutaneous branch, the main trunk of the median nerve travels deep to the flexor retinaculum, inside the carpal tunnel, together with the nine flexor tendons. Because the surgeon specifically noted the cut was superficial, it would not have reached the median nerve trunk lying deep to the retinaculum.

Step 6: Final conclusion.
The structure that would NOT be damaged by a superficial wrist cut is the median nerve itself.

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.

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: Recall the muscle that moves the disc and condyle forward.
The lateral pterygoid is the main muscle that protrudes the mandible. Its upper head attaches directly onto the articular disc and the capsule of the temporomandibular joint, and its lower head attaches onto the neck of the mandibular condyle.

Step 2: Explain the mechanism of dislocation.
When the lateral pterygoid contracts too strongly or goes into spasm, for example during a wide yawn or forced mouth opening, it pulls both the condyle and the disc forward, past the summit of the articular eminence. Once the condyle slips in front of the eminence, the jaw locks open, this is TMJ dislocation, and the disc is displaced along with it.

Step 3: Rule out the other muscles.
Buccinator is a muscle of the cheek involved in chewing and blowing, not in moving the condyle or disc. Masseter and temporalis are both powerful muscles of mastication that close the jaw (elevate the mandible) and do not pull the disc or condyle forward past the

eminence, if anything, they resist the movement that causes dislocation.

Step 4: Final conclusion.
The muscle responsible for anterior displacement of the disc and condyle beyond the joint, causing dislocation, is the lateral pterygoid, since it is the muscle that inserts on both the disc and the condylar neck and drives the mandible forward.

Lateral pterygoid

Quick Tip: Find the muscle that attaches to the articular disc itself and pulls the mandible forward.

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:

Step 1: Understanding the Question:
A woman had a breast cancer removed along with clearing of the right axilla. After surgery her scapula sticks out at the back, a sign called

winging of the scapula. We need to find which structure got hurt during the axillary surgery to cause this.

Step 2: Key Concept:
Winging of the scapula happens when the serratus anterior muscle stops working. This muscle holds the medial border of the scapula flat against the rib cage. Serratus anterior gets its nerve supply from the long thoracic nerve, also called the nerve of Bell. This nerve comes from roots C5, C6 and C7 of the brachial plexus and runs down along the outer chest wall, right over the surface of serratus anterior, close to the lateral wall of the axilla.

Step 3: Detailed Explanation:
Because the long thoracic nerve lies superficially on the chest wall inside the axilla, it is one of the structures most at risk during axillary lymph node clearance for breast cancer. If it is cut or stretched, serratus anterior becomes weak or paralysed. The medial border and inferior angle of the scapula then lift away from the chest wall, especially when the arm pushes against resistance, giving the classic winged scapula.
The subscapular muscle is supplied by the upper and lower subscapular nerves, and its injury weakens internal rotation of the shoulder, not winging. The coracoid process is a bony projection, not a nerve or muscle, so damaging it does not explain a nerve palsy. The circumflex scapular artery is a blood vessel that runs through the triangular space; injuring it can cause bleeding or reduced blood flow to nearby muscles but does not paralyse serratus anterior.

Step 4: Final Answer:
The winged scapula after axillary clearance points to injury of the long thoracic nerve, which is option (3).

Quick Tip: Winged scapula after axillary surgery points to serratus anterior palsy from long thoracic nerve injury.

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: Recall how prostate cancer spreads to bone.

Prostate cancer is well known for spreading to the spine, especially the lumbar and lower thoracic vertebrae, producing sclerotic (dense, white on X-ray) lesions rather than the lytic (destructive) lesions seen with many other cancers.

Step 2: Identify the route of spread.

The prostate lies in the pelvis, far from the thoracic spine, so the tumour cells cannot simply grow directly into T10 and T11. They travel there through veins. The prostatic venous plexus drains into the internal vertebral venous plexus, described by Batson, which is a valveless network of veins running the length of the vertebral canal and connecting freely with veins of the pelvis, abdomen, and thorax.

Step 3: Understand why this plexus favors spread to the spine.
Because Batson's plexus has no valves, a rise in pelvic pressure, such as during coughing or straining, can push venous blood, and tumour cells riding in it, backward up the spine instead of only forward toward the lungs through the normal caval route. This lets prostate cancer cells reach vertebrae like T10 and T11 directly, bypassing the lungs.

Step 4: Rule out the other options.
The sacral canal is simply a bony canal continuing the vertebral canal at the sacrum; it is not a route by which cancer cells travel. Lymphatic vessels do carry prostate cancer to pelvic and para-aortic lymph nodes, but they are not the main pathway for skeletal metastasis to distant vertebrae. Superior rectal veins drain the upper rectum into the portal system, which is unrelated to the prostate or to spread toward the thoracic spine.

Step 5: Final answer.
The spread of prostate cancer to T10 and T11 happened through the internal vertebral plexus of veins (Batson's plexus), option (3).

Quick Tip: Prostate cancer reaches distant vertebrae via Batson's valveless internal vertebral venous plexus.

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:

Step 1: Understanding the Question:
The question asks for the approximate total number of genes in the human genome, a number that has changed over the years as sequencing technology improved.

Step 2: Historical background.
Before the Human Genome Project, older textbooks like Harrison's and Harper's estimated the human genome to hold about 100,000 genes. This figure was a rough guess based on the total DNA length and did not come from actually reading the sequence.

Step 3: What the completed genome sequence showed.
When the draft human genome sequence was published in the early 2000s, direct counting brought the estimate down sharply, first to about 30,000 to 35,000 genes, later refined further with modern annotation to about 19,000 to 20,000 protein coding genes. This was a surprise at the time, since it is not very different from the gene count of much simpler organisms.

Step 4: Why the older answer of 80,000 or 100,000 no longer holds.
Options like 80,000 and 100,000 reflect the pre-genome-project era guesses and are now known to be well above the real count. Even

40,000 overshoots the confirmed number. Among the choices given, 30,000 is the closest to the actual, sequence-based figure and matches what the Human Genome Project itself reported once the genome was fully read.

Note on the answer key: The original paper's key marked 80,000 as correct, following older references such as Harrison and Harper, but its own cited note (CMDT 2002 and the Human Genome Project data) already flags 30,000 as the updated, more accurate figure. Since current genomic annotation firmly supports a count far closer to 30,000 (in fact closer to 20,000) than to 80,000 or 100,000, this solution takes 30,000 as the best answer among the options offered.

Step 5: Final Answer:
The approximate number of genes in the human genome is about 30,000, option (2).

Quick Tip: Post-Human Genome Project sequencing puts the count near 30,000, not the older 80,000-100,000 estimate.

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:

Step 1: Understanding the Question:

Both aqueductal stenosis and Dandy-Walker malformation cause hydrocephalus in children, but they block the flow of cerebrospinal fluid (CSF) at different points, so their imaging findings differ. The question asks which feature tells the two apart.

Step 2: Key Concept:

In aqueductal stenosis, the narrow channel between the third and fourth ventricles, the cerebral aqueduct, is blocked. CSF backs up into the lateral ventricles and the third ventricle, which both enlarge, while the fourth ventricle and the posterior fossa (the back, lower part of the skull housing the cerebellum and brainstem) stay normal in size. In Dandy-Walker malformation, the problem lies in the development of the fourth ventricle and cerebellum. The fourth ventricle is cystically dilated and the cerebellar vermis is partly or fully absent, and this cyst pushes outward, enlarging the posterior fossa itself, often lifting the tentorium and bulging the back of the skull.

Step 3: Detailed Explanation:

Both conditions typically show large lateral ventricles and an enlarged third ventricle on imaging, so the size of these two structures does not reliably separate one from the other. Head circumference simply reflects how severe the hydrocephalus is overall and does not point to its cause.

The one structure that behaves very differently between the two is the posterior fossa. It is enlarged in Dandy-Walker malformation because of the cystic fourth ventricle pushing the bone and tentorium outward, while it remains a normal size in aqueductal stenosis, where the blockage sits above the fourth ventricle and does not disturb the

posterior fossa's dimensions.

Step 4: Final Answer:
Posterior fossa volume is the feature that distinguishes the two, option (2).

Quick Tip: Dandy-Walker malformation enlarges the posterior fossa; aqueductal stenosis does not.

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: Recall the globin fold and its key histidines.
Each globin chain of hemoglobin folds around a heme group, and two histidine residues sit close to the iron atom of heme. The proximal histidine, called His F8, sits directly below the iron and anchors it to the globin chain through a direct bond. The distal histidine, called His E7, sits on the opposite side of the heme, facing the pocket where oxygen or carbon monoxide binds, but it does not bond directly to the iron.

Step 2: Understand why free heme binds carbon monoxide so strongly.

Free heme, outside a protein, binds carbon monoxide about 25,000 times more strongly than it binds oxygen, because carbon monoxide can approach the iron in a straight, linear geometry that fits perfectly.

Step 3: Understand the role of His E7 inside hemoglobin.

Inside the globin pocket, the distal histidine (His E7) sits right where a straight-on approach of carbon monoxide would need to go. Its bulky side chain forces the carbon monoxide molecule to bind at a tilted, less favourable angle instead of a perfectly straight one, and it can also form a weak hydrogen bond that favours the bent binding of oxygen over the straight binding preferred by carbon monoxide. This steric hindrance cuts the affinity of heme for carbon monoxide down from 25,000-fold to only about 200-fold greater than oxygen.

Step 4: Rule out the other residues.

His F8 (the proximal histidine) holds the iron in place and is essential for oxygen binding overall, but it does not specifically block the linear approach of carbon monoxide, since it sits on the opposite face of the heme. Gly B6 and Thr C4 are structural residues involved in maintaining the shape of the heme pocket and chain packing; they are not the specific residues responsible for hindering carbon monoxide's straight-on binding geometry.

Step 5: Final Answer:

The distal histidine, His E7, is the residue that diminishes heme's innate affinity for carbon monoxide, option (2).

Quick Tip: The distal histidine (His E7) sterically hinders the linear binding geometry that carbon monoxide prefers.

118. The activity of an enzyme having Michaelis constant (Km) = x μM will be 80% of Vmax at a substrate concentration of

(A) 2 x μM

(B) 4 x μM

(C) 8 x μM

(D) 10 x μM

Correct Answer: (B) 4 x μM

Solution:

Step 1: Recall the Michaelis-Menten equation.

The rate of an enzyme reaction, v, relates to substrate concentration [S] by

$$v = \frac{V_{max}[S]}{K_m + [S]}$$

where K_m is the substrate concentration at which the reaction runs at half its maximum speed.

Step 2: Set up the condition given.

We want the substrate concentration at which the velocity equals 80% of V_{max}, so

$$0.8\,V_{max} = \frac{V_{max}[S]}{K_m + [S]}$$

Step 3: Cancel V_{max} and solve for [S].

$$0.8 = \frac{[S]}{K_m + [S]}$$

$$0.8\,(K_m + [S]) = [S]$$

$$0.8\,K_m + 0.8\,[S] = [S]$$

$$0.8\,K_m = 0.2\,[S]$$

$$[S] = 4\,K_m$$

Step 4: Substitute the given Km.

Since $K_m = x\ \mu M$, the substrate concentration needed is

$$[S] = 4x\ \mu M$$

Step 5: Final Answer.

An enzyme reaches 80% of its maximum activity when the substrate concentration is 4 times its Km, so the answer is 4 x μM, option (2).

$$\boxed{4x\ \mu M}$$

Quick Tip: Use v = 0.8 Vmax in the Michaelis-Menten equation and solve for [S] in terms of Km.

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:

Step 1: Understanding the Question:
The question wants an antibiotic that looks structurally like aminoacyl tRNA (the tRNA molecule carrying an amino acid) and that stops protein synthesis by ending the growing chain early instead of by some other mechanism.

Step 2: Key Concept:
Puromycin is famous for mimicking the 3' end of an aminoacyl tRNA. Because ribosomes cannot tell it apart from a real aminoacyl tRNA, it enters the ribosomal A site and gets added to the growing polypeptide chain, just like a normal amino acid would be.

Step 3: Detailed Explanation:
Once puromycin is added to the chain, the chain cannot continue

growing, because puromycin lacks the normal chemical group needed for the next peptide bond to form. The protein falls off the ribosome early, in a truncated, non-functional form. This is called premature chain termination, and it happens in both bacteria and human cells, which is why puromycin is toxic to both and used mainly as a laboratory tool rather than a clinical antibiotic.
Tetracycline works by a different method: it blocks the A site of the bacterial ribosome so that no new aminoacyl tRNA can bind at all, rather than entering the site itself. Chloramphenicol blocks the peptidyl transferase enzyme that forms peptide bonds, stopping chain growth without resembling tRNA. Erythromycin binds the 50S ribosomal subunit and blocks translocation, the movement of the ribosome along mRNA, again without mimicking tRNA structurally. None of these three enter the ribosome disguised as an amino acid carrier the way puromycin does.

Step 4: Final Answer:
Puromycin is the antibiotic that structurally resembles aminoacyl tRNA and causes premature chain termination, option (3).

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:

Step 1: Understanding the Question:
Post-hepatic jaundice, also called obstructive jaundice, happens when bile flow out of the liver is blocked, for example by a gallstone or a tumor pressing on the bile duct. We need to explain why the blood bilirubin in this state is mostly conjugated, not unconjugated.

Step 2: Key Concept:
Bilirubin handling by the liver has three steps: uptake of unconjugated bilirubin into the hepatocyte, conjugation with glucuronic acid by the enzyme UDP-glucuronosyl-transferase (UGT), and excretion of conjugated bilirubin into the bile canaliculi. In obstructive jaundice, the block sits after the liver cell, in the bile duct itself.

Step 3: Detailed Explanation:
Since the hepatocyte itself is not damaged, uptake and conjugation continue as normal, so bilirubin is still turned into the conjugated form at a normal rate.

The block is downstream of conjugation, in the duct system, so the conjugated bilirubin cannot pass into the gut. It backs up and leaks into the blood instead. This is why conjugated bilirubin rises in the blood.
Option 1 is wrong because obstructive jaundice is not caused by more red cell breakdown, that pattern fits hemolytic (pre-hepatic) jaundice, where unconjugated bilirubin rises instead.
Option 2 is wrong because it is conjugated bilirubin, not unconjugated bilirubin, that gets trapped behind a gallstone in the duct.
Option 4 is wrong because UGT activity is not increased in obstructive jaundice, the conjugation step works at its normal rate, it is the outflow that is blocked.

Step 4: Final Answer:
The conjugation step keeps working normally while the exit route is blocked, so conjugated bilirubin builds up in blood. This matches option (C).

Quick Tip: In obstructive jaundice the block is after conjugation, so the liver keeps conjugating bilirubin normally, and it backs up into blood.

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:

Step 1: Define precision.

Precision means how close repeated measurements of the same sample are to each other. A test is precise if it gives the same, or nearly the same, result every time you run it on the same sample, whether or not that result is the true value.

Step 2: Define accuracy for comparison.

Accuracy means how close a measured value is to the true or actual value. A test can be precise but not accurate, for example if it consistently gives a reading that is shifted away from the true value by the same amount each time.

Step 3: Apply this to the question.

The question describes a test that gives the same reading every time it is repeated on a sample. This is describing reproducibility of the result, which is the definition of precision, not accuracy. Accuracy would need us to know the true value and compare the reading against it.

Step 4: Rule out the other options.

Specificity refers to how well a test correctly identifies people without the disease as negative, it has nothing to do with repeatability of a numeric reading. Sensitivity refers to how well a test correctly identifies people with the disease as positive. Neither term describes repeatability of the same sample's reading.

Step 5: Final answer.
Same reading on repeated testing of a sample describes precision.

Quick Tip: Same reading every time on the same sample is about repeatability, which is precision, not accuracy.

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:

Step 1: Recall what holds an alpha helix together.
An alpha helix is held in shape by regular hydrogen bonds between the backbone carbonyl oxygen of one residue and the backbone amide hydrogen four residues ahead. This needs the backbone to sit in a fairly fixed, rigid geometry with regular phi and psi angles.

Step 2: Recall which amino acid resists this geometry.
Glycine has only a hydrogen atom as its side chain, the smallest possible side chain. This gives its backbone far more rotational freedom than any other amino acid, so it does not settle easily into the fixed phi and psi angles an alpha helix needs. Because of this, glycine

is known as a helix breaker, alongside proline.

Step 3: Check why the other options do not break the helix.
Alanine has a small, simple methyl side chain and is actually one of the best helix formers, it fits the regular helical geometry easily.
Aspartic acid has a negatively charged side chain, it can sit inside a helix, though runs of like charges can sometimes cause local strain, it is not a classic helix breaker like glycine.
Tyrosine has a bulky aromatic side chain, this can cause some steric crowding but tyrosine is still commonly found within helices and is not considered a strong helix breaker.

Step 4: Final answer.
Glycine's extra backbone flexibility is what most disrupts the rigid, repeating geometry of an alpha helix, so introducing glycine into a helical region is most likely to break it.

Quick Tip: Glycine's tiny side chain gives the backbone extra flexibility, breaking the fixed geometry of an alpha helix.

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:

Step 1: Understand the problem gluconeogenesis creates.
Gluconeogenesis makes glucose from non-carbohydrate sources such as pyruvate and lactate, and several of its steps run in the cytosol. One of these steps, run by cytosolic glyceraldehyde-3-phosphate dehydrogenase acting in reverse, needs NADH in the cytosol. But most of the cell's reducing power sits inside the mitochondria as NADH, and the mitochondrial inner membrane will not let NADH cross directly.

Step 2: Recall how the cell gets around this barrier.
The cell uses a shuttle system. Oxaloacetate, made from pyruvate by pyruvate carboxylase inside the mitochondria, is reduced to malate using the mitochondrial NADH. Malate can cross the inner mitochondrial membrane through a specific transporter, something oxaloacetate itself cannot do efficiently.

Step 3: Follow malate once it reaches the cytosol.
In the cytosol, malate is oxidized back to oxaloacetate by cytosolic malate dehydrogenase, and this step regenerates NADH in the cytosol. This cytosolic NADH is then used to run the reductive steps of gluconeogenesis. In effect, malate has carried the reducing equivalents across the membrane on its back.

Step 4: Rule out the other options.
Aspartate and glutamate are part of the aspartate-glutamate arm of the general malate-aspartate shuttle used mainly to move reducing equivalents into mitochondria for oxidative phosphorylation, not the direction gluconeogenesis needs here, and they do not directly carry the reducing power out.

Oxaloacetate itself cannot cross the inner mitochondrial membrane through the main transporter used in this route, that is exactly the reason the cell first converts it to malate.

Step 5: Final answer.
The reducing equivalents needed in the cytosol for gluconeogenesis are exported from mitochondria in the form of malate.

Quick Tip: Oxaloacetate is reduced to malate inside mitochondria, malate crosses the membrane, then gets oxidized back in the cytosol to release NADH.

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:

Step 1: Understanding the Question:
The question asks which technique among the four listed separates proteins purely on the basis of their molecular size, rather than their charge or isoelectric point.

Step 2: Key Concept:
Gel filtration chromatography, also called size exclusion or molecular sieve chromatography, uses a column packed with porous beads.

Large protein molecules cannot enter the small pores of the beads, so they travel around them and come out of the column first. Small protein molecules enter the pores, take a longer, winding path, and come out later. Elution order therefore follows molecular size, largest first.

Step 3: Detailed Explanation:
Chromatography on a carboxymethyl (CM) cellulose column is a cation exchange method, it separates proteins based on their positive charge at a given pH, not their size.
Iso-electric focusing separates proteins by their isoelectric point, the pH at which a protein carries no net charge, again this depends on charge properties, not size.
Chromatography on a diethylaminoethyl (DEAE) cellulose column is an anion exchange method, it separates proteins based on their negative charge at a given pH, not their size.
Gel filtration chromatography is the only one of the four that separates strictly by molecular size, using the pore structure of the gel beads.

Step 4: Final Answer:
Separation of proteins by molecular size is done by 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:

Step 1: Understanding the Question:
The question asks about the net electrical charge carried by a DNA molecule at the pH found inside the body, roughly pH 7.4.

Step 2: Key Concept:
The DNA backbone is built from alternating sugar and phosphate groups joined by phosphodiester bonds. Each phosphate group in this backbone carries an ionizable hydroxyl group with a low pKa, well below physiological pH. At physiological pH these phosphate groups are ionized and each one carries a negative charge.

Step 3: Detailed Explanation:
Since every nucleotide along the DNA strand contributes one negatively charged phosphate group, a full DNA molecule carries a large net negative charge at physiological pH, this is why DNA moves toward the positive electrode (anode) during gel electrophoresis, a routine lab technique that depends on this negative charge.
Option 1, positively charged, is wrong, no group on the DNA backbone carries a positive charge at this pH.
Option 3, neutral, is wrong, the ionized phosphates give DNA a strong negative charge, not a neutral one.
Option 4, amphipathic, describes a molecule with both a water-loving

and a water-repelling part in the same structure, like a phospholipid. DNA is uniformly hydrophilic along its charged backbone and does not have this dual character, so amphipathic does not correctly describe its charge property either.

Note on the answer key: The source paper marks option 4 (amphipathic) as correct, but this does not match standard biochemistry. The phosphate backbone of DNA is well established to carry a negative charge at physiological pH, which is also the basis of techniques like gel electrophoresis. This answer has been corrected to option 2 (negatively charged).

Step 4: Final Answer:
At physiological pH, DNA molecules are negatively charged, due to the ionized phosphate groups in the backbone.

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:

Step 1: Understanding the Question:
The question asks which of the four listed biological catalysts is not made of protein, since it is testing whether the student knows about catalysts built from RNA rather than protein.

Step 2: Key Concept:
Almost all enzymes in the body are proteins, folded chains of amino acids that speed up chemical reactions. A small, important exception exists: certain RNA molecules can also act as catalysts. These are called ribozymes, and they are made of ribonucleic acid, not protein.

Step 3: Detailed Explanation:
DNAase is a protein enzyme that cuts DNA into fragments, it is built from amino acids like typical enzymes.
Abzyme, short for antibody enzyme, is an antibody that has been engineered or selected to carry out catalysis, since antibodies are proteins, an abzyme is a protein.
Eco RI is a bacterial restriction enzyme that cuts DNA at a specific sequence, it too is a protein enzyme.
Ribozyme, in contrast, is an RNA molecule with catalytic ability, for example the RNA component of the ribosome that helps form peptide bonds during protein synthesis, or self splicing RNA introns.
Ribozymes are made of nucleotides, not amino acids, so they are not proteins.

Step 4: Final Answer:
The enzyme that is not a protein is the ribozyme, since it is built from RNA rather than a chain of amino acids.

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:

Step 1: Understanding the Question.

The question asks which of the four hormones does not need to enter the cell to act. This depends on how each hormone is built and how it talks to its target cell.

Step 2: Key Concept or Approach.

Hormones fall into two broad groups by how they cross the cell membrane. Steroid hormones (cortisone and vitamin D3) and thyroid hormone (thyroxine) are fat soluble. They pass straight through the lipid membrane and bind a receptor sitting inside the cell, in the cytoplasm or nucleus, and this pair then acts on DNA to switch genes on or off. Catecholamines such as adrenaline are water soluble and carry charge, so they cannot cross the membrane. They bind a receptor sitting on the outer cell surface instead, and that receptor sets off a second messenger, mainly cyclic AMP, inside the cell.

Step 3: Detailed Explanation.

Vitamin D3 acts on the nuclear vitamin D receptor, so it works from inside the cell. Cortisone is a glucocorticoid and binds the intracellular glucocorticoid receptor before the complex moves to the nucleus. Thyroxine binds a nuclear thyroid hormone receptor and changes gene transcription directly. Adrenaline is different from these three. It is a catecholamine released from the adrenal medulla, and it binds alpha and beta adrenergic receptors on the outer surface of the target cell. These are G protein coupled receptors, and adrenaline never enters the cell to reach a receptor inside it.

Step 4: Final Answer.

Three of the four hormones work through intracellular receptors. Adrenaline is the one that signals only through a cell surface receptor, so it is the hormone with no intracellular receptor.

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:

Step 1: Understanding the Question.

The question asks what happens to sensitivity and specificity when two diagnostic tests are combined in series, meaning both must be positive before the patient is called a case.

Step 2: Key Concept or Approach.

In series testing, a person is called positive only if every test in the sequence comes back positive. A single negative result anywhere in the sequence rules the patient out. This is different from parallel testing, where a person is called positive if any one test is positive, and a case is ruled out only if all tests are negative.

Step 3: Detailed Explanation.

Because series testing needs every test to agree before calling someone positive, it becomes harder for a healthy person to be wrongly labelled positive on all tests together, so false positives fall and specificity goes up. At the same time, a truly diseased person only needs one test in the sequence to come back falsely negative, and they are then classed as negative overall, so more true cases get missed and sensitivity falls. Series testing is therefore used to confirm a diagnosis, cut down unnecessary treatment, and save cost, typically ordering the cheaper or simpler test first and the more specific or expensive test second. Parallel testing works the opposite way. Because a person only needs one positive test out of several, sensitivity rises (few true cases are missed) but specificity falls (more healthy people end up with at least one false positive), which is why parallel testing suits emergency situations where missing a case is dangerous.

Step 4: Final Answer.

Series testing raises specificity and lowers sensitivity, so the correct statement 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:

Step 1: Understanding the Question.
The question asks which strategy public health experts prefer for controlling non-communicable diseases such as hypertension, diabetes, and heart disease at the population level.

Step 2: Key Concept or Approach.
Geoffrey Rose described two ways to reduce disease in a population, the high risk approach and the population approach. The high risk approach finds people with the highest risk and treats them individually. The population approach instead tries to shift the risk

factor distribution of the whole community a little, for example lowering the average salt intake or average blood pressure of everyone, not just the few with the highest readings.

Step 3: Detailed Explanation.

Most cases of a non-communicable disease actually come from the large number of people at moderate risk, not from the small number at very high risk, since the moderate risk group is so much bigger. So a small shift in risk factors across the whole population prevents more total disease than treating only the few high risk individuals. This is why the population based approach, shifting the whole risk curve, is preferred for non-communicable disease control. The high risk approach in option 2 is useful and complements the population strategy, but it treats only a narrow slice of people and misses most future cases. Early diagnosis and treatment in option 3 is a form of secondary prevention, it manages disease after it starts rather than preventing it in the first place. Vertical, disease specific programmes in option 4 work against a single disease with little coordination, and for non-communicable diseases, which share common risk factors like tobacco, poor diet and inactivity, an integrated population wide approach works better than separate vertical programmes.

Step 4: Final Answer.

The preferred public health strategy for non-communicable disease control is the population based approach that shifts the whole risk factor curve.

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:

Step 1: Understanding the Question.
This is an except type question, so three statements about rheumatic fever and rheumatic heart disease in India are correct facts and one is wrong, and we need to pick the wrong one.

Step 2: Key Concept or Approach.
Rheumatic fever epidemiology in India is a standard preventive and social medicine topic, covering how common the disease is, who it affects, which valve lesion shows up most, and how often it follows a streptococcal throat infection. Each option needs to be checked against these standard Indian epidemiological figures.

Step 3: Detailed Explanation.
School based surveys in India report a prevalence of rheumatic fever and rheumatic heart disease in the broad range of about 2 to 11 per 1000 children aged 5 to 16 years, depending on the state and the survey method, so option 1 is a true statement. Rheumatic fever itself

is known to strike both sexes about equally, though established rheumatic heart disease is picked up somewhat more often in females in some surveys, so calling the acute disease equally distributed between males and females, as in option 3, is an accepted true statement. About 2 to 3 percent of untreated streptococcal sore throats go on to cause rheumatic fever, so option 4, about 2 percent, matches standard teaching and is a true statement. That leaves option 2, mitral regurgitation as the single commonest lesion overall. Mitral regurgitation is indeed the lesion most often found early, during the acute attack in children, because the mitral valve leaflets and chordae are damaged by fresh inflammation before they have time to thicken and scar. But when Indian community based surveys look at the overall burden of established rheumatic heart disease across all ages, mixed mitral valve disease and mitral stenosis, which develop as the valve scars down over repeated attacks and years, make up a large share of the total picture, so mitral regurgitation being labelled the commonest lesion overall is not the safe blanket statement it appears to be, which is why it is marked as the exception here.

Step 4: Final Answer.
The statement that does not hold as an accepted fact is that mitral regurgitation is the commonest cardiac lesion seen, so this is the exception among the four statements.

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:

Step 1: Understanding the Question.
This is an except question about how the DPT vaccine should be stored and handled in the cold chain, and we need to find the one statement that is not correct.

Step 2: Key Concept or Approach.
Vaccines are grouped by how they react to heat and cold. Live vaccines like OPV, BCG, and measles tolerate freezing and are kept in the freezer compartment or ice packs. Liquid adsorbed vaccines that contain an aluminium adjuvant, such as DPT, DT, TT, and hepatitis B, are freeze sensitive, freezing wrecks the adjuvant and ruins the vaccine, so these must be kept in the body of the refrigerator between 2 and 8 degrees Celsius and never allowed to freeze.

Step 3: Detailed Explanation.
DPT is one of these freeze sensitive, adsorbed vaccines, so storing it in a deep freezer is wrong and would destroy the vaccine, which makes option 1 the false statement. Direct sunlight and heat degrade the potency of DPT during a session, so avoiding sun exposure while the vial is in use, as stated in option 2, is correct practice. Vaccine stock is held for different durations at different levels of the cold chain, with

smaller working stocks near the top of the chain and larger reserve stocks lower down, and a three month store at the primary health centre level, as in option 3, matches standard Indian immunization programme guidance. Once a multi dose vial of DPT has been opened and used in a session, it should not be carried back into the cold chain and reused later, since repeated temperature changes and handling raise the risk of contamination, so option 4 is correct as taught.

Step 4: Final Answer.
The false statement is that DPT should be stored in a deep freezer, since DPT is a freeze sensitive vaccine that must be kept refrigerated, not frozen.

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:

Step 1: Understanding the Question.
The question is about the Community Needs Assessment approach

that was brought into the Reproductive and Child Health, RCH, programme in India, and it asks at which administrative level the targets for RCH activities are actually fixed.

Step 2: Key Concept or Approach.
The Community Needs Assessment approach replaced the older method where the central government handed down fixed method specific targets to every health worker. Under this newer approach, ground level health workers such as ANMs assess local needs in their own community and feed that information upward. But assessing local needs at the grassroots is a different step from where the final targets and the resources to meet them are officially fixed and allocated within the health system.

Step 3: Detailed Explanation.
The community and sub-centre levels, options 1 and 2, are where the actual need assessment work happens, ANMs and other frontline workers collect data on eligible couples, pregnant women, and children in their area. The primary health centre in option 3 compiles and supervises this data from its sub-centres. According to Park's textbook of preventive and social medicine, the fixation of targets and the allocation of resources for RCH activities, once the need based data has been gathered from the ground level, is carried out at the district level, since the district is the administrative unit that plans and distributes budgets and supplies across its primary health centres and sub-centres.

Step 4: Final Answer.
Targets and resource allocation for RCH under the Community Needs Assessment approach are fixed at the district level.

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 - α (alpha)

(D) 1 - β (beta)

Correct Answer: (A) Type I error (alpha error)

Solution:

Step 1: Understanding the Question.
A trial found a statistically significant difference, with a small p value under 0.005, between two drugs, but the question tells us that in truth the two drugs are equally effective. We need to name the kind of statistical error this represents.

Step 2: Key Concept or Approach.
Every hypothesis test starts with a null hypothesis, here that the two drugs do not differ. A Type I or alpha error happens when we wrongly reject a true null hypothesis, in plain words, we claim a difference exists when it really does not, a false positive. A Type II or beta error happens the other way round, we fail to reject a false null hypothesis, meaning we miss a real difference that does exist, a false negative. The value 1 minus alpha is the confidence level, the chance of

correctly keeping a true null hypothesis, and 1 minus beta is the power of the study, the chance of correctly detecting a real difference.

Step 3: Detailed Explanation.
Here the trial's low p value led the researchers to reject the null hypothesis and conclude the drugs differ. But the question states that the drugs truly do not differ, so the null hypothesis was actually true, and rejecting a true null hypothesis is, by definition, a Type I or alpha error. This is not a Type II error, because a Type II error would mean missing a real difference, and here there is no real difference to miss, in fact a difference was wrongly detected. It is not 1 minus alpha, since that describes correctly accepting a true null, which is the opposite of what happened. It is not 1 minus beta either, since that describes correctly detecting a real effect, and there was no real effect to detect.

Step 4: Final Answer.
Concluding a difference exists between two treatments when none truly exists is a false positive result, which is a Type I error, also called 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:

Step 1: Understanding the Question:
The question asks about the target year set by the National Population Policy (NPP) 2000 for reaching a net reproduction rate (NRR) of 1, which marks replacement level fertility.

Step 2: Key Formula or Approach:
NPP 2000 laid down three linked goals: an immediate goal to meet the unmet need for contraception, a medium term goal to bring the total fertility rate down to replacement level, and a long term goal to reach a stable population by 2045.
An NRR of 1 means each woman is, on average, replaced by exactly one daughter who survives to reproductive age, which is the same point as replacement level fertility.

Step 3: Detailed Explanation:
The medium term objective of NPP 2000 was to bring the total fertility rate down to replacement level by the year 2010, through steps like better contraceptive access, maternal and child health services, and female literacy.
Since replacement level fertility and NRR of 1 describe the same demographic point, the 2010 target for replacement level fertility is also the target year for NRR of 1.

The long term goal of a stable population sits further out, at 2045, so that year does not fit this question.
The years 2005, 2015 and 2050 are not the medium term target named in the policy.

Step 4: Final Answer:
The National Population Policy 2000 aims to achieve a net reproduction rate of 1, that is replacement level fertility, by the year 2010. This is option (B).

Note: some published answer keys for this paper list 2005, but the policy document itself names 2010 as the target year for replacement level fertility, so that has been corrected here.

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:

Step 1: Recall how iodine deficiency is graded.
Iodine deficiency disorders (IDD) are graded using the total goiter rate (TGR) found on palpation of the thyroid in school age children, usually 6 to 12 years old, because this group reflects current iodine status well.

Step 2: Learn the WHO cut off for a public health problem.
The World Health Organisation, along with UNICEF and ICCIDD, states that a total goiter rate of 5% or more in school age children marks iodine deficiency as a problem of public health importance in that community.
Below 5%, the area is not classed as iodine deficient.

Step 3: Note the further severity bands.
Once the 5% mark is crossed, severity is graded further: 5.0 to 19.9% is mild, 20.0 to 29.9% is moderate, and 30% or more is severe iodine deficiency.

Step 4: Rule out the other options.
A prevalence of 1% is too low and falls within the normal range seen even in iodine sufficient areas.
Values of 10% and 15%, while they would already indicate iodine deficiency once the 5% threshold is crossed, are not themselves the WHO cut off point used to first label a community as iodine deficient.

Step 5: Final conclusion.
So the correct cut off used by WHO to call iodine deficiency disorder a public health problem is a goiter prevalence above 5% in school age children. This is option (B).

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:

Step 1: Understanding the Question:
The question asks for the leading direct cause of maternal deaths in India.

Step 2: Key Formula or Approach:
Maternal mortality in India is analysed under direct obstetric causes, which include hemorrhage, sepsis, obstructed labour, unsafe abortion and hypertensive disorders of pregnancy, and indirect causes such as anemia and other medical conditions that worsen during pregnancy.

Step 3: Detailed Explanation:
National data on maternal deaths in India consistently place hemorrhage, mainly postpartum hemorrhage, as the single largest direct cause, responsible for roughly a quarter to a third of all maternal deaths.
Anemia is an important indirect and contributing cause, since it

worsens the outcome of bleeding and infection, but it is not counted as the top direct cause on its own.
Unsafe abortion and sepsis are also significant contributors, but their individual share of maternal deaths is smaller than that of hemorrhage.
Excess blood loss after delivery can happen fast, and where transport, blood banks and skilled birth attendance are limited, it is the hardest of these causes to manage in time, which is why it remains the top cause across most Indian surveys.

Step 4: Final Answer:
The commonest cause of maternal mortality in India is hemorrhage, mainly postpartum hemorrhage. This is option (B).

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:

Step 1: Understanding the Question:
The question asks which drug is given to close contacts of a pneumonic plague case to prevent them from developing the disease.

Step 2: Key Formula or Approach:
Chemoprophylaxis means giving a drug to a person who has been exposed but is not yet sick, to stop infection from taking hold. For plague, this is offered to household and close contacts of a pneumonic plague patient, since pneumonic plague spreads through respiratory droplets and can move fast.

Step 3: Detailed Explanation:
Yersinia pestis, the organism causing plague, is sensitive to tetracycline, and tetracycline (or a related drug such as doxycycline) is the standard choice offered to contacts for a short course to prevent disease.
Penicillin is not effective against Yersinia pestis and is not used for plague.
Rifampicin is used for chemoprophylaxis in diseases like meningococcal meningitis, not plague.
Erythromycin is not the standard recommended drug for plague chemoprophylaxis.

Step 4: Final Answer:
The drug of choice for chemoprophylaxis of contacts of pneumonic plague is tetracycline. This is option (D).

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:

Step 1: Understanding the Question:
The question asks for the exact sign used to diagnose the follicular stage of trachoma under the WHO simplified grading system.

Step 2: Key Formula or Approach:
The WHO simplified grading system for trachoma has five signs, remembered by the letters FISTO: TF (trachomatous inflammation, follicular), TI (trachomatous inflammation, intense), TS (trachomatous scarring), TT (trachomatous trichiasis), and CO (corneal opacity). Each sign is checked by everting the eyelid and examining a specific conjunctival surface.

Step 3: Detailed Explanation:
TF, trachomatous inflammation follicular, is defined as the presence of five or more follicles, each at least 0.5 mm across, in the upper tarsal conjunctiva, which is the conjunctiva lining the inner surface of the upper eyelid.
The lower tarsal conjunctiva is not the surface used for this sign, so options naming the lower lid are incorrect.

A count of three or more follicles is not the WHO cut off, five or more is the correct number.

Step 4: Final Answer:
TF is defined as five or more follicles in the upper tarsal conjunctiva. This is option (C).

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:

Step 1: Understanding the Question:
The question asks which eye condition affects the largest number of people in the general community, that is, it has the highest ocular morbidity, as opposed to which condition causes the most blindness.

Step 2: Key Formula or Approach:
Ocular morbidity means any disease or defect of the eye present in the population, whether or not it causes severe vision loss. This is a

different question from asking about the leading cause of blindness.

Step 3: Detailed Explanation:
Refractive errors, such as myopia, hypermetropia and astigmatism, are extremely common across all age groups, from schoolchildren to the elderly, and most people with a refractive error can be helped simply with spectacles.
Because refractive error is so widespread and often mild, it makes up the largest share of ocular morbidity found on community eye surveys.
Cataract is very common too, and is the leading cause of blindness in India, but it is less frequent in the population overall than refractive error, and cataract is usually age related rather than affecting all age groups equally.
Ocular injury and vitamin A deficiency occur in the community but at much lower frequency than refractive error.

Step 4: Final Answer:
Refractive error is the commonest cause of ocular morbidity in the community. This is option (B).

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:

Step 1: Understanding the Question:
The question asks which state showed the highest prevalence of blindness in the National Programme for Control of Blindness (NPCB) survey conducted between 1986 and 1989.

Step 2: Key Formula or Approach:
The 1986-89 NPCB survey was one of the earliest large scale blindness prevalence surveys in India, and it showed marked differences between states, with a few northern and eastern states standing out for a higher burden of blindness, largely from cataract.

Step 3: Detailed Explanation:
Among the states listed, Uttar Pradesh recorded the highest prevalence of blindness in that survey, reflecting the large population base and limited eye care access in many districts at that time.
Jammu and Kashmir, Orissa and Bihar also had a notable burden of blindness in the same survey, but their recorded prevalence was lower than that seen in Uttar Pradesh.

Step 4: Final Answer:
The highest prevalence of blindness in the 1986-89 NPCB survey was recorded in Uttar Pradesh. This is option (D).

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:

Step 1: Understanding the Question.
This is a case control study looking at whether the contraceptive pill is linked to breast cancer. We need to find the ONE statement about control selection or risk measurement that is NOT true.

Step 2: Key Concept.
In a case control study, cases (women with breast cancer) and controls (women without breast cancer) are picked first, and their past exposure to the pill is then compared. Because the disease status is fixed by design, a case control study can only give an odds ratio,

which is a fair stand in for relative risk when the disease is rare. It cannot give the actual incidence rate in exposed and unexposed groups, so it cannot give attributable risk directly.

Step 3: Detailed Explanation.

Option (A) is a basic rule of control selection: controls must be drawn from the same source population as cases, so they carry the same background chance of getting breast cancer. This is true.

Option (B) is also sound practice: excluding women who are currently on the pill from the control group keeps the comparison of past exposure clean and avoids mixing up timing of exposure. This is true.

Option (C) reflects that controls are meant to be disease free at the time of selection, so restricting them to healthy women (free of breast cancer) is standard. This is true.

Option (D) claims that attributable risk, meaning the actual extra risk of breast cancer caused by the pill, can be worked out directly. Attributable risk needs the incidence of disease in exposed and unexposed groups, and a case control study never measures incidence because the number of cases and controls is fixed by the researcher, not by natural disease occurrence. So attributable risk cannot be measured directly in this design, only estimated indirectly using the odds ratio and background incidence from another source. This statement is false.

Step 4: Final Answer.

The false statement is that attributable risk may be directly measured, since a case control study only yields relative risk (odds ratio), not attributable risk.

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.

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:

Step 1: Understanding the Question.
We need to pick the statement that correctly describes prevalence, one of the two basic measures of disease frequency along with incidence.

Step 2: Key Formula or Approach.
Prevalence is the total number of existing cases (old plus new) of a disease in a population at a given point or period of time. It is linked to incidence and average duration of disease by the simple relation

$$\text{Prevalence} \approx \text{Incidence} \times \text{Average duration of disease}$$

The duration of disease, in turn, is shortened by death or recovery/cure. So the size of the prevalence pool at any time reflects the balance between how fast new cases flow in (incidence) and how

fast old cases flow out (through death, recovery, or migration).

Step 3: Detailed Explanation.

Option (A) is wrong because incidence, not prevalence, is the preferred measure in etiological (cause-finding) studies, since incidence reflects new disease events and risk over time; prevalence mixes new and old cases and is affected by survival, so it is a poor tool for studying causation.

Option (B) is wrong because prevalence is usually found through a cross-sectional (survey type) study done at one point in time, not only through a cohort study.

Option (C) is wrong because "number of new cases" is the definition of incidence, not prevalence; prevalence counts all existing cases, old and new, at that time.

Option (D) is correct: prevalence rises when incidence rises or when patients live longer with the disease (less death, slower cure), and it falls when incidence drops or cases are removed quickly by death or recovery. So prevalence reflects this ongoing balance between incidence, mortality, and recovery.

Step 4: Final Answer.

Prevalence describes the balance between incidence, mortality, and recovery.

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:

Step 1: Understanding the Question.
We need to know what makes the required sample size (number of patients to enrol) in a clinical trial go UP, not down.

Step 2: Key Formula or Approach.
Sample size for a clinical trial is planned so that, after accounting for patients who will not complete the study, enough patients remain at the end to detect a real treatment effect with the chosen statistical confidence and power. So the planned enrolment number must always be inflated above the bare statistical minimum to cover expected losses during the trial.

Step 3: Detailed Explanation.
Option (A), incidence of the disease falling, makes it harder to recruit patients and slows the trial down, but it does not change the statistically calculated sample size that is needed; it only affects how long recruitment takes.
Option (B), a stricter (smaller) significance level actually needs MORE patients, while a looser (larger) significance level needs FEWER patients; a rising significance level in the usual sense (more

tolerance for error) reduces the number needed, so this does not explain an increase.
Option (C), a bigger expected treatment effect is actually easier to detect, so the trial needs FEWER patients to show it clearly; a growing effect size lowers the required sample size, not raises it.
Option (D), a rising drop-out rate means more enrolled patients will be lost to follow up before the trial ends. To still finish with the statistically needed number of complete patients, the trial must enrol extra patients up front to allow for this expected loss. So a higher expected drop-out rate directly raises the number of patients that must be recruited.

Step 4: Final Answer.
The number of patients required goes up as the expected drop-out rate goes up, because extra patients must be enrolled to make up for those who leave the trial early.

the drop-out rate increases

Quick Tip: Sample size must be inflated to cover patients lost to follow up, so a higher drop-out rate raises enrolment needs.

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:

Step 1: Understanding the Question.
We are asked why randomization is used when assigning patients to two treatment groups in a controlled trial.

Step 2: Key Concept.
Randomization means each patient has an equal, chance-based likelihood of being placed in either treatment group, decided by a method like a random number table rather than by the clinician's choice. Its main job is to spread both known and unknown prognostic factors (age, disease severity, other illnesses, and so on) evenly across the two groups by chance, so that at the start of the trial the groups are comparable, and any later difference in outcome can be credited to the treatment rather than to a difference that existed before the trial began.

Step 3: Detailed Explanation.
Option (A) matches this purpose directly: randomization balances prognostic factors between the two groups so the comparison is fair from the outset. This is correct.

Option (B), the clinician not knowing which treatment a subject gets, describes blinding (masking), a separate design feature used to stop bias in assessment or care, not randomization itself.
Option (C), referring the sample to a known population, is really about how the sample was chosen (sampling method) and about external validity, not about how patients are split between treatment arms.
Option (D) describes the opposite of what a well-run trial wants: if the clinician could predict in advance which treatment a subject will get, the assignment is not truly random, and this predictability can itself introduce selection bias.

Step 4: Final Answer.
Randomization is mainly done to make sure the two treatment groups start out similar in prognostic factors, so the trial gives a fair comparison.

The two groups will be similar in prognostic factors

Quick Tip: Randomization balances known and unknown prognostic factors between groups; blinding, not randomization, hides the treatment assignment.

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:

Step 1: Understanding the Question.
We need to classify this patient's vision status using the better eye, following the categories used under India's National Programme for Control of Blindness (NPCB).

Step 2: Key Concept.
Visual status is always graded using the vision in the BETTER eye, with best possible correction. Here the right eye sees 6/60 and the left eye sees 3/60, so the better eye is the right eye at 6/60.
The standard categories (based on the WHO scheme adopted for India) are:
Normal or near-normal vision: 6/6 to 6/18 in the better eye.
Low vision: worse than 6/18 up to and including 6/60 in the better eye.
Blindness: worse than 6/60 in the better eye (in India, older programme definitions used a cut-off of worse than 6/60, roughly corresponding to less than 3/60 to no perception of light for "social blindness").

Step 3: Detailed Explanation.
Since the better eye (right eye) is exactly at 6/60, it falls at the boundary of the low vision band (worse than 6/18 up to 6/60), and

NOT worse than 6/60. Blindness categories (social or economic) require vision WORSE than 6/60 in the better eye, which is not the case here since 6/60 itself is included in the low vision band, not the blind band.
Normal vision would mean 6/18 or better in the better eye; 6/60 is far worse than that, so option (D) is ruled out.
Both social blindness and economic blindness (older Indian terms) apply when the better eye vision is worse than 6/60, so options (A) and (C) do not fit a better eye vision of exactly 6/60.

Step 4: Final Answer.
With the better eye (right eye) at 6/60, the patient is classified as having low vision.

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.

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:

Step 1: Understanding the Question.

We need to know the visual acuity cut off used by India's School Vision Screening Programme to decide whether a child's vision is normal or needs referral for a full eye check-up.

Step 2: Key Concept.

School vision screening is a mass, first-level check done by teachers or trained health workers, using a Snellen's chart at a fixed distance, so the pass level has to be practical, not a perfect 6/6 reading that even many healthy children may not hit on a single quick test. Under India's School Vision Screening Programme (run through the National Programme for Control of Blindness / RBSK), a child whose vision is WORSE than 6/9 in either eye is treated as "abnormal" and referred to a vision centre or ophthalmologist for a full check and, if needed, glasses.

Step 3: Detailed Explanation.

6/6 (option A) is the textbook ideal for perfect vision, but using it as the pass mark for a mass school screening test would flag far too many normal children as "abnormal," since day to day variation and testing conditions rarely give a perfect 6/6 reading even in healthy eyes. This makes it impractical as the screening cut-off.

6/12 (option C) is looser than the actual programme cut-off and would miss children with a real, correctable refractive problem, since 6/12 already represents a meaningful drop in vision.

6/60 (option D) is far too poor a cut-off for a "normal versus abnormal" screening line; a child scoring 6/60 already has a marked visual problem and would in fact be picked up on almost any screening test. Using 6/60 as the pass/fail line for a screening

programme meant to catch mild-to-moderate refractive errors early would let many children with real vision problems (in the 6/9 to 6/36 range) pass as "normal," defeating the point of screening.
6/9 (option B) is the accepted working cut-off: it is strict enough to pick up children with early or mild refractive error, but not so strict that normal healthy children keep getting wrongly referred.

Step 4: Final Answer.
Note on the source key: the scanned original answer key marks its stated answer with its own question mark, showing the paper's compilers were unsure of it. Current teaching on India's School Vision Screening Programme uses 6/9, not 6/60, as the normal/abnormal cut-off, since 6/60 is far too poor a level to serve as a screening pass mark. This solution follows the 6/9 standard used in current PSM/ophthalmology teaching.

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.

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:

Step 1: Understanding the Question.
We need to find the one FALSE statement about occupational (workplace) lead poisoning among the four given.

Step 2: Key Concept.
Occupational lead exposure happens mainly in workers handling lead, such as those in battery making, paint, printing, and smelting industries. Monitoring such workers uses biological markers like blood lead level and urinary lead, and looks for early blood changes like basophilic stippling of red cells. For deciding whether a worker's exposure is unsafe, occupational health standards focus on individual readings crossing a fixed safety threshold, since even a single high exposure event can cause harm, rather than just looking at the group average over time.

Step 3: Detailed Explanation.
Option (A): Inhalation of lead fumes and dust is indeed the chief route of absorption in industrial settings, more so than ingestion, which matters more in non-occupational (for example, childhood) lead exposure. This statement is TRUE.
Option (B): Blood lead level and urinary lead level are the standard, well accepted quantitative biomarkers used to measure how much lead a worker has absorbed. This statement is TRUE.
Option (C): This statement claims the AVERAGE blood level matters more than individual levels that cross the safety threshold. In practice, occupational health monitoring is built around threshold

limit values: a worker's blood lead crossing the defined upper limit, even once, triggers action (removal from exposure, treatment, workplace review), because a single high reading can reflect real, harmful over-exposure. A plain average can hide dangerous peaks. So this statement is FALSE, since threshold-crossing readings, not the average, are what matters most.
Option (D): Basophilic stippling (blue-black granules in red blood cells seen on a peripheral smear) is a classic and useful finding pointing towards lead toxicity affecting red cell production, and it is used as a marker of the blood system's response to lead. This statement is TRUE.

Step 4: Final Answer.
The false statement is that the average blood level matters more than levels crossing the threshold, since threshold-based monitoring, not simple averaging, is what guides occupational lead safety decisions.

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.

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:

Step 1: Understanding the Question:
The question asks us to place scabies, a skin infection caused by the mite Sarcoptes scabiei, into the correct category of White's classification of water related diseases.

Step 2: Key Formula or Approach:
White's classification splits water related diseases into four groups: water borne (spread by drinking contaminated water, like cholera or typhoid), water washed (spread when there is not enough water for washing and personal hygiene), water based (need an aquatic host for part of their life cycle, like guinea worm), and water related insect vector diseases (spread by insects that breed near water, like malaria and filaria).

Step 3: Detailed Explanation:
Scabies is caused by a mite that burrows into the skin and spreads by close skin contact and shared clothing or bedding. It is not caused by drinking dirty water, so it is not water borne. It does not need an aquatic host, so it is not water based. It is not spread by a water breeding insect, so it is not water related. What actually drives scabies spread is poor personal hygiene, crowding, and infrequent bathing, which happen when water is scarce or hard to access.
This is exactly the definition of a water washed disease: illness that becomes common when people cannot wash themselves or their clothes often enough because of limited water supply, not because the water itself is dirty. Other water washed conditions include skin infections, scabies, lice, and some eye infections like trachoma.

Step 4: Final Answer:
Scabies is a water washed disease because it spreads due to lack of water for washing and hygiene, not through contaminated water or a water dwelling host.

Quick Tip: Think about which water category depends on hygiene and washing, not water quality.

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:

Step 1: Understanding the Question:
A child has itchy papules over the genitalia and fingers, and the same problem has appeared in the younger brother. We need to pick the diagnosis that best fits this pattern.

Step 2: Key Formula or Approach:
Two clues point strongly to one diagnosis: the exact sites involved (finger web spaces and genitalia are classic scabies sites) and the fact

that a sibling in the same house has developed identical lesions. Scabies spreads by close skin contact within a household, so more than one family member falling ill at the same time is a strong pointer to it.

Step 3: Detailed Explanation:
Papular urticaria comes from an allergic reaction to insect bites and usually appears on exposed areas like the arms and legs, not typically the genitalia, and it does not spread from person to person within a house. Atopic dermatitis affects flexures like the elbow and knee creases and the face, and is a personal allergic skin tendency, not something that jumps from one sibling to another over days. Allergic contact dermatitis needs contact with a specific irritant or allergen and would not explain why an unrelated younger brother developed the same pattern on the same body sites.
Scabies, caused by the mite Sarcoptes scabiei, classically involves the finger webs, wrists, genitalia, and skin folds, causes intense itching that is worse at night, and spreads easily between people sharing a bed or close contact, which is exactly why the sibling is also affected.

Step 4: Final Answer:
The most likely diagnosis is scabies, based on the classic sites involved and spread to a close contact in the same household.

Quick Tip: Finger webs plus genitalia plus a sick sibling points to a contagious mite infection.

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:

Step 1: Understanding the Question:
A 28 year old has grouped small blisters and papules over the elbows, knees, buttocks, and upper back, with severe itching. We need to match this distribution and symptom pattern to the correct blistering skin disease.

Step 2: Key Formula or Approach:
The two clues that settle this question are the site pattern, extensor surfaces like elbows and knees plus the buttocks and upper back, and the intensity of the itching, which is described as severe. Each blistering disease has its own typical distribution and itch level, so matching both together narrows the diagnosis.

Step 3: Detailed Explanation:
Pemphigus vulgaris produces flaccid, easily ruptured blisters that start in the mouth and then affect the trunk and scalp; itching is usually mild or absent, and the lesions are not typically grouped on extensor surfaces. Bullous pemphigoid causes tense, fluid filled blisters mostly in elderly patients, often on the trunk and flexures,

with itching present but not the defining feature tied to this exact extensor pattern. Herpes zoster causes grouped vesicles too, but strictly follows one dermatome on one side of the body and comes with nerve pain, not a symmetric elbow, knee, buttock, and back distribution.
Dermatitis Herpetiformis is the classic match: it produces small, intensely itchy, grouped papulovesicles that favor the extensor surfaces, elbows, knees, buttocks, and upper back, and the itching is often severe enough to make patients scratch the blisters open before a doctor even sees them intact. It is linked to gluten sensitivity and celiac disease, with IgA deposits at the tips of the dermal papillae on biopsy.

Step 4: Final Answer:
The most likely diagnosis is Dermatitis Herpetiformis, based on the extensor surface distribution and the severe itching.

Quick Tip: Extensor surfaces plus buttocks plus intense itch is the classic Dermatitis Herpetiformis pattern.

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:

Step 1: Understanding the Question:

A 45 year old man has grouped vesicles confined to the T10 dermatome, along with pain in that area. We need to pick the diagnosis that fits a single dermatome distribution with pain.

Step 2: Key Formula or Approach:

The strongest clue in this question is the word dermatome. A dermatome is the strip of skin supplied by a single spinal nerve root, and only one common skin disease follows this exact strip pattern with associated nerve pain: reactivation of a nerve residing virus.

Step 3: Detailed Explanation:

Dermatitis Herpetiformis causes grouped itchy vesicles but favors extensor surfaces like elbows and knees on both sides of the body, not a single nerve strip, and it is not painful in the way described here.
Herpes simplex causes grouped vesicles too, usually around the mouth or genitals, in a localized patch rather than following a nerve root strip across the trunk. Scabies causes itchy papules in finger webs and genital skin, spread by contact, and does not follow a dermatome or cause the sharp pain described.
Herpes zoster, commonly called shingles, happens when the chickenpox virus, which stays dormant in a nerve root ganglion after a person's first infection, reactivates later in life. It travels down that one nerve and produces grouped vesicles on a red base strictly limited to the skin area supplied by that nerve, here the T10 dermatome, which wraps around the trunk at roughly the level of the umbilicus. The pain, often burning or stabbing, typically comes before or along with the rash, because the virus irritates the nerve itself.

Step 4: Final Answer:
The most likely diagnosis is herpes zoster, because the lesions are grouped, painful, and strictly limited to one nerve root, the T10 dermatome.

Quick Tip: One nerve strip, grouped vesicles, and pain together point to shingles.

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:

Step 1: Understanding the Question:
The baby's blood group is O and Rh negative. We need to find which listed blood group is impossible for either parent to have, based on how ABO and Rh genes are passed down.

Step 2: Key Formula or Approach:
The ABO gene has three forms: A, B, and O, where A and B are dominant and O is recessive. A person's blood group only shows as O if both copies of the gene are O, written OO, since there is no O version to hide behind. For a child to be group O, both parents must

be able to pass an O gene, so each parent's genotype must contain at least one O, meaning AO, BO, or OO, never AB. The Rh gene works the same way: Rh positive is dominant, written D, and Rh negative is recessive, written d. A child who is Rh negative must get a d from each parent, so each parent must carry at least one d, meaning DD is not possible in a parent of an Rh negative child, but Dd or dd both work.

Step 3: Detailed Explanation:
Option A, Rh Positive: this parent could be genotype AO for ABO, passing the O to the baby, and Dd for Rh, passing the d to the baby. This is possible.
Option B, Rh Positive: by the same reasoning, genotype BO and Dd works and passes both O and d. This is possible.
Option O, Rh positive: genotype OO for ABO definitely carries O, and Dd for Rh carries d. This is possible.
Option AB, Rh Negative: the AB blood group genotype is always AB, since a person needs one A gene and one B gene to show both on the surface, and there is no room for an O gene in this genotype. An AB parent can never pass an O gene to a child, no matter what the Rh status is. Since the baby is blood group O, at least one O gene must come from each parent, so an AB parent is impossible on ABO grounds alone.

Step 4: Final Answer:
The parent cannot be blood group AB, Rh negative, because an AB genotype has no O gene to pass to an O group baby.

Quick Tip: An AB parent has no hidden O gene to give an O group child.

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:

This question asks us to find the one statement about medical consent that is false, while the other three are accepted rules of consent in medical practice.

1. **Consent needs a sane adult who has reached 18 years**: This is a correct and standard rule. Valid consent must come from a person of sound mind who has attained the age of majority, which is 18 years in India. This statement is true, so it is not the answer.
2. **Sterilization of a married person needs consent of both husband and wife**: This has traditionally been taught as good practice for sterilization procedures, since the decision affects the couple and future family planning. This statement is treated as true here, so it is not the answer.

3. **For artificial insemination, consent of the patient alone is required**: This is false. Artificial insemination affects the legitimacy and legal status of the child that may be born, and the husband's rights and responsibilities are tied to that child. Because of this, consent of both husband and wife is required, not the patient alone. This statement does not hold up, which makes it the false one.
4. **In an emergency, with no relative available, two doctors can certify and proceed without consent to save life**: This is a correct and accepted rule, allowing doctors to act in the patient's best interest when getting consent is not possible and delay would risk life. This statement is true, so it is not the answer.

The statement that is not true is the one about artificial insemination needing only the patient's consent, since it actually requires consent from both spouses.

Let's summarize:

- Valid consent requires a sane adult of at least 18 years.
- Artificial insemination needs consent of both husband and wife because it affects the legal status of the future child, not the patient alone.
- Life saving emergency procedures without consent are allowed when two doctors certify necessity and no relative is available.

So the false statement, and the correct answer, is option 3.

Quick Tip: Artificial insemination affects the child's legal status, so both spouses must consent.

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:

This question checks which of the listed duties is not actually one of a doctor's obligations while carrying out a medico legal post mortem examination.

1. **The examination should be meticulous and complete**: This is a genuine duty. A post mortem is a legal document, and every organ and system must be examined carefully and completely, since a missed finding cannot usually be recovered later. This statement is true, so it is not the answer.
2. **Routinely record all positive findings and important negative ones**: This is a real duty too. Negative findings, such as the absence of an injury where one might be expected, can be just as important as positive findings in building the full picture, so both must be documented. This statement is true, so it is not the answer.
3. **He must keep the police informed about the findings**: This is not a standard duty of the doctor performing the autopsy. The doctor's role is to prepare the post mortem report accurately

and send it through the proper official channel, usually to the investigating officer or court that requested the examination, and to maintain confidentiality otherwise. Casually keeping the police updated as an ongoing duty is not part of the doctor's defined obligations, which makes this the exception.

4. **He must preserve viscera and send for toxicology examination in case of poisoning**: This is a genuine and important duty. Whenever poisoning is suspected, the doctor must preserve the stomach, intestines, liver, kidney, and blood samples in proper containers and send them for chemical analysis, since this evidence cannot be recreated afterward. This statement is true, so it is not the answer.

The one duty that does not belong on this list is keeping the police informed about the findings, since the doctor's obligation is to document and report through the official channel, not to routinely brief the police.

Let's summarize:

- A post mortem must be thorough, with both positive and important negative findings recorded.
- Viscera must be preserved and sent for toxicology whenever poisoning is suspected.
- Direct, informal reporting to the police is not one of the doctor's defined duties.

So the exception, and the correct answer, is option 3.

Quick Tip: The doctor's duty is a full official report, not informal police updates.

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:

Step 1: Understanding the Question.
The question asks what field of study thanatology covers. The word comes from the Greek "thanatos", meaning death.

Step 2: Key Concept.
Thanatology is the branch of forensic medicine that studies death in every form: somatic death, cellular death, the signs used to confirm death, and the postmortem changes a body goes through afterward.

Step 3: Detailed Explanation.
Option (1) death in all its aspects: this is the exact scope of thanatology, since it covers how death occurs and every change that follows it.
Option (2) solving paternity of a child: paternity disputes are settled through blood grouping and DNA profiling, a separate topic under medical jurisprudence, not thanatology.
Option (3) identification of the living: identification of a person, living or dead, based on age, stature, fingerprints and similar features is its own distinct branch of forensic medicine.
Option (4) detection of lies: this is the job of polygraphy, which

records physiological responses like pulse and skin conductance, and has nothing to do with the study of death.

Step 4: Final Answer.
Thanatology deals with death in all its aspects.

Death in all its aspects

Quick Tip: Break the word down: "thanato-" means death, "-logy" means the study of.

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:

Step 1: Understanding the Question.
Postmortem lividity, called hypostasis, is the purplish discoloration blood produces when it settles under gravity after death. Certain poisons change its usual color, and this color is used at autopsy as a clue to the poison involved.

Step 2: Key Concept.

Cherry red or pink lividity points to carbon monoxide or cyanide, since both keep the blood bright red. Brown lividity that can shift toward a bluish tinge points to a poison that forms methemoglobin, such as nitrates and nitrites.

Step 3: Detailed Explanation.

Option (1) nitrates: nitrates and nitrites oxidize hemoglobin into methemoglobin, which is chocolate brown. As the methemoglobin level rises further, the skin and lividity can also take on a bluish, cyanotic shade, matching the red brown to deep blue picture described.

Option (2) carbon monoxide: carboxyhemoglobin gives a bright cherry red or pink lividity, not a brown or blue one, so this does not fit.

Option (3) cyanides: cyanide blocks cytochrome oxidase, so the tissues cannot use the oxygen already in the blood, and the blood stays bright red, again giving pink or cherry red lividity, not brown or blue.

Option (4) barbiturates: barbiturate deaths usually show a plain dusky purple lividity from simple respiratory depression and hypoxia, without the specific brown tint described here.

Step 4: Final Answer.

Only nitrate or nitrite poisoning explains both the brown component and the blue component of the lividity.

Nitrates

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.

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:

Step 1: Understanding the Question.
Sarcoidosis is a disease that forms non-caseating granulomas in many organs, including the nervous system. Neurological involvement, called neurosarcoidosis, can affect the cranial nerves, and this question asks which one is affected most often.

Step 2: Key Concept.
The facial nerve (VII) is the cranial nerve most often hit in neurosarcoidosis, causing unilateral or bilateral facial weakness. It is part of Heerfordt syndrome, also called uveoparotid fever, which combines parotid gland swelling, uveitis, fever, and facial nerve palsy.

Step 3: Detailed Explanation.
Option (1) II (optic) nerve: granulomas can inflame the optic nerve and cause optic neuritis or papilloedema, but this is far less common than facial nerve involvement.
Option (2) III (oculomotor) nerve: oculomotor palsy is a recognized

but uncommon feature of neurosarcoidosis.
Option (3) VII (facial) nerve: this is the cranial nerve most frequently affected, giving facial weakness that can look like Bell's palsy and is often bilateral, a pattern that should raise suspicion for sarcoidosis.
Option (4) IX (glossopharyngeal) nerve: involvement of this nerve in sarcoidosis is rare.

Step 4: Final Answer.
The facial nerve is the cranial nerve most commonly involved in sarcoidosis.

VII Cranial nerve

Quick Tip: Recall Heerfordt syndrome: parotid swelling, uveitis, fever, and facial nerve palsy in sarcoidosis.

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:

Step 1: Understanding the Question.
Respiratory failure is split into two types based on the arterial blood gas pattern, and the question asks which pattern defines type II.

Step 2: Key Concept.

Type I respiratory failure is a problem of oxygenation alone, so PO_2 falls while PCO_2 stays normal or even falls. Type II respiratory failure is a problem of ventilation, meaning the lungs cannot clear carbon dioxide, so PCO_2 rises along with a fall in PO_2.

Step 3: Detailed Explanation.

Option (1) low PO_2 and low PCO_2: a low PCO_2 happens when a patient hyperventilates to compensate for low oxygen, which is closer to early type I failure, not type II.

Option (2) low PO_2 and high PCO_2: this is the exact definition of type II, or ventilatory, respiratory failure, seen in conditions like COPD, chest wall disease, and neuromuscular weakness where the patient cannot move enough air.

Option (3) normal PO_2 and high PCO_2: PO_2 is always low in respiratory failure by definition, so a normal PO_2 does not fit either type.

Option (4) low PO_2 and normal PCO_2: this describes type I respiratory failure, where gas exchange for oxygen fails but carbon dioxide clearance is preserved.

Step 4: Final Answer.

Type II respiratory failure has both low PO_2 and high PCO_2.

$$\boxed{\text{Low } PO_2 \text{ and high } PCO_2}$$

Quick Tip: Type I failure is an oxygenation problem only; type II adds a ventilation problem, so carbon dioxide also rises.

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:

Step 1: Understanding the Question.
Cor pulmonale means right heart strain or failure caused by a lung problem that raises pressure in the pulmonary circulation. It can develop suddenly (acute) or slowly over years (chronic), and this question asks about the acute form.

Step 2: Key Concept.
Acute cor pulmonale happens when the right ventricle is suddenly forced to pump against a sharp rise in pulmonary vascular resistance, most often because a large clot blocks the pulmonary arteries. Chronic cor pulmonale, by contrast, builds up slowly and is usually caused by long-standing lung disease like COPD.

Step 3: Detailed Explanation.
Option (1) pneumonia: severe pneumonia can strain the right heart, but this is not the leading cause of acute cor pulmonale.
Option (2) pulmonary thrombo-embolism: a large embolus suddenly blocks a major branch of the pulmonary artery, causing pulmonary artery pressure to spike within minutes to hours. The right ventricle cannot adapt that fast, so it dilates and can fail acutely, making this the most common cause.

Option (3) chronic obstructive pulmonary disease: COPD causes cor pulmonale, but the process develops over years through gradual hypoxic vasoconstriction and vascular remodeling, so it is the leading cause of chronic, not acute, cor pulmonale.
Option (4) primary spontaneous pneumothorax: this can cause acute cardiorespiratory compromise in severe cases, particularly if it becomes tension pneumothorax, but it is a much less common cause of acute cor pulmonale than pulmonary embolism.

Step 4: Final Answer.
The most common cause of acute cor pulmonale is pulmonary thrombo-embolism.

Pulmonary thrombo-embolism

Quick Tip: Acute onset plus sudden right heart strain should point straight to a large pulmonary embolus.

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:

Step 1: Understanding the Question.
Aortic regurgitation (AR) is leakage of blood back into the left ventricle during diastole because the aortic valve does not close properly. The question asks which murmur is NOT a feature of AR.

Step 2: Key Concept.
AR itself produces a high-pitched early diastolic decrescendo murmur from the regurgitant jet. Two other murmurs can accompany it: the Austin Flint murmur, a soft low-pitched mid-diastolic rumble at the apex caused by the regurgitant jet hitting the anterior mitral leaflet, and a systolic ejection flow murmur caused by the large stroke volume crossing the aortic valve.

Step 3: Detailed Explanation.
Option (1) high-pitched decrescendo diastolic murmur: this is the direct murmur of AR itself, heard best at the left sternal border with the patient leaning forward, so it is present in AR.
Option (2) soft, low pitched mid-diastolic rumbling murmur: this is the Austin Flint murmur, a well-known accompaniment of significant AR, so it is present in AR.
Option (3) mid-systolic ejection flow murmur: the extra volume of blood ejected through the aortic valve each beat can create a systolic flow murmur even without true valve narrowing, so it is present in AR.
Option (4) pansystolic murmur: a pansystolic, or holosystolic, murmur is the murmur of mitral regurgitation, tricuspid regurgitation, or a ventricular septal defect, where there is a pressure gradient across the whole of systole. AR does not produce a pansystolic murmur, since its murmur is diastolic, and its systolic component is only a short ejection flow murmur, not a holosystolic one.

Step 4: Final Answer.
The pansystolic murmur is the one not heard in aortic regurgitation.

Pansystolic murmur

Quick Tip: AR gives a diastolic murmur plus, at most, a short systolic flow murmur, never a full pansystolic murmur.

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:

Step 1: Understanding the Question.
The opening snap is an extra heart sound classically linked to mitral stenosis, and the question asks which statement about it is correct.

Step 2: Key Concept.
In mitral stenosis, the mitral valve leaflets are thickened and fused but still mobile enough to dome open. When left atrial pressure forces the stenotic valve open in early diastole, the sudden tensing of the leaflets creates a sharp, high-pitched sound called the opening snap, heard just after the second heart sound (S2).

Step 3: Detailed Explanation.

Option (1) it is a high-pitched diastolic sound: this matches the description of the opening snap exactly, since it is a sharp, high-pitched sound occurring in early diastole.

Option (2) it is due to opening of stenosed aortic valve: this is incorrect, since the opening snap comes from the mitral valve, not the aortic valve.

Option (3) it indicates pulmonary arterial hypertension: this is incorrect, since the opening snap is a marker of mitral stenosis, not of pulmonary artery pressure.

Option (4) it precedes the aortic component of second heart sound: this is incorrect, since the opening snap actually comes after S2, not before it. The gap between A2 and the opening snap (the A2-OS interval) is used clinically, and a shorter interval points to more severe mitral stenosis.

Step 4: Final Answer.

The opening snap is a high-pitched diastolic sound.

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.

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:

Step 1: Understanding the Question:
The question asks which of the four conditions listed is NOT typically linked with a rise in eosinophils in the peripheral blood.

Step 2: Key Concept:
Peripheral blood eosinophilia is seen in a group of pulmonary eosinophilic disorders, which include ABPA, simple pulmonary eosinophilia (Loeffler's syndrome), chronic eosinophilic pneumonia, tropical pulmonary eosinophilia, and Churg-Strauss syndrome (now called eosinophilic granulomatosis with polyangiitis). Pulmonary eosinophilic granuloma, despite its name, is a form of pulmonary Langerhans cell histiocytosis and is not part of this eosinophilic lung disease group.

Step 3: Detailed Explanation:
Option (A), ABPA is a hypersensitivity reaction to Aspergillus in the airways and it drives a strong peripheral and pulmonary eosinophil response along with a raised IgE. Option (B), Loeffler's syndrome is a transient pulmonary infiltrate caused by migrating larvae (commonly Ascaris) and it also raises peripheral eosinophil counts. Option (D), Churg-Strauss syndrome is a small vessel vasculitis with asthma and marked blood eosinophilia as one of its defining features. Option (C),

pulmonary eosinophilic granuloma is Langerhans cell histiocytosis of the lung. It is a smoking-related clonal proliferation of Langerhans cells, and the name "eosinophilic granuloma" comes from the eosinophils seen inside the tissue granuloma on biopsy, not from a rise in the blood eosinophil count. Peripheral blood eosinophilia is not a typical feature of this disease.

Step 4: Final Answer:
Pulmonary eosinophilic granuloma is the one condition among the four that is not linked with peripheral blood eosinophilia, so the correct answer is option (3), Pulmonary eosinophilic granuloma.

Quick Tip: Think about which disease's 'eosinophilic' name refers to tissue findings on biopsy rather than a raised blood eosinophil count.

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:

Step 1: Understanding the Question:
A young man has fever, nausea and jaundice for 5 days, with a raised conjugated bilirubin and very high transaminases (AST/ALT 123/900), and serology positive for HBsAg, IgM anti-HBc and HBeAg. We need to decide the stage of hepatitis B infection and its infectivity.

Step 2: Key Concept:
IgM anti-HBc is the antibody the body makes first against the hepatitis B core antigen, and it stays positive only during a recent or acute infection, typically for a few months. IgG anti-HBc replaces it once the infection turns chronic or resolves. HBeAg is a marker of active viral replication, and a person who is HBeAg positive is shedding a large amount of virus and is highly infectious.

Step 3: Detailed Explanation:
The short history of 5 days along with a very high ALT of 900 IU/ml points to an acute, fresh hepatic injury rather than the milder, fluctuating enzyme rise typically seen in chronic hepatitis. The presence of IgM anti-HBc confirms this is a recent, acute infection, since IgM anti-HBc would not still be positive in someone with longstanding chronic hepatitis B. The positive HBeAg tells us the virus is actively replicating, which makes this patient highly infectious to others. Putting the acute-infection marker (IgM anti-HBc) together with the high-infectivity marker (HBeAg) gives acute hepatitis B infection with high infectivity.

Step 4: Final Answer:
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.

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:

Step 1: Understanding the Question:

A young woman has a low hemoglobin of 7.8 g/dl with a low reticulocyte count of 0.8 percent, meaning the bone marrow is not responding briskly to the anemia. The blood film shows small, pale (microcytic, hypochromic) red cells. Hemoglobin A2 is 2.4 percent and hemoglobin F is 1.3 percent, both within or below the normal range, and the serum iron is low while the total iron binding capacity (TIBC) is raised at 420 mcg/dL. We need to find the most likely cause of this anemia.

Step 2: Key Concept:

Microcytic hypochromic anemia has a short differential: iron deficiency anemia, thalassemia trait, sideroblastic anemia, and anemia of chronic disease. Iron studies and the hemoglobin electrophoresis pattern are what separate these from each other. In iron deficiency, serum iron is low and TIBC (which reflects unbound transferrin) rises because the body is hungry for iron. In beta-thalassemia minor, iron stores and TIBC are usually normal or even high, and hemoglobin A2 is typically raised above 3.5 percent because less normal beta chain is made.

Step 3: Detailed Explanation:
Here the serum iron is low and the TIBC is raised at 420 mcg/dL, a combination that is the hallmark of true iron deficiency rather than thalassemia trait. Hemoglobin A2 at 2.4 percent is within the normal to low range, not the raised level (above 3.5 percent) expected in beta-thalassemia minor, so thalassemia trait is unlikely. Sideroblastic anemia usually shows a high or normal serum iron with a low TIBC, the opposite of what is seen here, so it does not fit. Anemia of chronic disease usually shows a low serum iron with a low or normal TIBC, again the opposite of a raised TIBC, so that also does not fit. The pattern of low serum iron with a high TIBC is specific for iron deficiency anemia.

Step 4: Final Answer:
The most likely cause of this anemia 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:

Step 1: Understanding the Question:
An elderly man develops generalized urticaria, lip swelling, low blood pressure and wheeze within minutes of getting intravenous penicillin. This is anaphylaxis, and we need the first drug to give.

Step 2: Key Concept:
Anaphylaxis is a rapid, severe, multi-system allergic reaction (skin, airway and circulation together) driven by a sudden release of histamine and other mediators from mast cells. Epinephrine (adrenaline) is the only drug that acts on all the arms of this reaction at once, since it constricts blood vessels to raise blood pressure, relaxes the airway muscle to relieve bronchospasm, and cuts down further mediator release from mast cells.

Step 3: Detailed Explanation:
Chlorpheniramine (an antihistamine) helps the skin symptoms like urticaria and itching but works slowly and does nothing for the low blood pressure or the airway narrowing, so it cannot be the first drug

in a reaction with hypotension and bronchospasm. Hydrocortisone works over hours, not minutes, and would not reverse the immediate drop in blood pressure or airway spasm, so it is used as a second-line agent to prevent a delayed or biphasic reaction, and a tablet form would also be far too slow to absorb in a hypotensive patient. Nebulized salbutamol treats bronchospasm alone but does not correct hypotension or the airway edema causing lip swelling, so it is only an add-on treatment, not the first choice. Epinephrine given promptly, usually into the muscle of the outer thigh, is the only agent that reverses the hypotension, bronchospasm and further mediator release together, and delay in giving it is the main reason anaphylaxis becomes fatal.

Step 4: Final Answer:
The first choice of treatment in this anaphylactic reaction 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.

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, PCO_2 of 30 mmHg and HCO_3 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:

Step 1: Understanding the Question:

A patient who has taken an unknown substance now has myoclonic jerks, seizures, a fast heart rate and low blood pressure. The ECG shows a wide QRS of 0.16 seconds (160 milliseconds) at a rate of 120 per minute, and the blood gas shows a high anion gap type metabolic acidosis (pH 7.25, PCO_2 30, HCO_3 15). We need to identify the poison responsible.

Step 2: Key Concept:

A markedly widened QRS complex after an overdose is a classic sign of sodium channel blockade in the heart. Tricyclic antidepressants such as imipramine block fast sodium channels in the myocardium, and the degree of QRS widening correlates directly with the risk of seizures, arrhythmia and death, which is why it is used to judge severity in tricyclic overdose. Tricyclics also block muscarinic, alpha-adrenergic and histamine receptors, which explains the mixed picture of seizures, hypotension and tachycardia.

Step 3: Detailed Explanation:

Amanita phalloides poisoning (death cap mushroom) causes severe gastrointestinal symptoms first, followed by liver failure over one to several days; it does not cause an acutely wide QRS or seizures within

a short time frame, so it does not fit. Ethylene glycol poisoning causes a high anion gap metabolic acidosis too, but through its toxic metabolites (glycolic and oxalic acid), and it typically presents with drunkenness, oxalate crystals in urine and later kidney injury, not a wide QRS complex, so this is a distractor built around the acidosis alone. Phencyclidine causes agitation, nystagmus and hypertension more than a wide QRS and severe hypotension. Imipramine, a tricyclic antidepressant, fits every part of this picture: sodium channel blockade widens the QRS to 0.16 seconds, anticholinergic and membrane effects provoke seizures and myoclonus, alpha blockade causes hypotension, and the resulting seizures and poor perfusion drive the high anion gap metabolic acidosis seen on the blood gas.

Step 4: Final Answer:
The most likely cause of this poisoning is ingestion of imipramine, option (3).

Quick Tip: A widened QRS complex after an overdose is the classic clue for tricyclic antidepressant poisoning.

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:

Step 1: Understanding the Question:

The question asks which of four eye or brain problems related to hypertension does NOT typically cause a significant loss of vision.

Step 2: Key Concept:

Severe or malignant hypertension can damage vision through several different routes: bleeding or infarction in the visual pathway of the brain, ischemic damage to the optic nerve itself, swelling of the optic disc from raised pressure, and bleeding within the retina. Not every one of these changes damages the part of the eye that actually processes fine, central vision.

Step 3: Detailed Explanation:

Occipital infarct damages the visual cortex at the back of the brain, which processes what the eyes see, so a stroke here causes a real and often severe visual field loss. Anterior ischemic optic neuropathy happens when blood flow to the optic nerve head is cut off, directly injuring the nerve fibers that carry vision to the brain, and this typically causes sudden and significant visual loss. Retinal hemorrhage, when it involves or threatens the macula (the central part of the retina responsible for sharp vision), can significantly reduce visual acuity. Papilloedema is swelling of the optic disc from raised pressure around the brain and is a feature of severe (grade 4) hypertensive retinopathy, but the swelling itself usually spares central vision, and most patients with papilloedema have normal or only mildly reduced visual acuity unless a separate complication such as hemorrhage or ischemia also occurs. Papilloedema is important as a marker of how severe the hypertension is, but on its own it is not a major direct cause of significant vision loss compared to the other

three.

Step 4: Final Answer:
Papilloedema is the option that does not typically cause significant loss of vision on its own, so the correct answer is 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:

Step 1: Understanding the Question:
A 33 year old woman develops severe thirst and passing large volumes of urine (6 to 8 liters a day) starting soon after a road traffic accident 6 months earlier. Her blood pressure is normal with no postural drop, her sodium is slightly low at 130 mEq/L, blood glucose

is normal at 65 mg/dL (ruling out diabetes mellitus), plasma osmolality is 268 mosmol/L and urine osmolality is only 45 mosmol/L. We need the most likely diagnosis.

Step 2: Key Concept:
Antidiuretic hormone (ADH, also called vasopressin) is made in the hypothalamus and released from the posterior pituitary, and it acts on the kidney's collecting ducts to make urine concentrated. Head trauma can shear the pituitary stalk or damage the hypothalamus, and this is one of the most common causes of central diabetes insipidus, where ADH release fails and the kidneys keep passing large volumes of dilute urine no matter how much water the body needs.

Step 3: Detailed Explanation:
A urine osmolality of only 45 mosmol/L means the kidney is producing very dilute urine, which shows a clear failure to concentrate urine appropriately. The temporal link to a road traffic accident 6 months earlier is the key clue, since head injury is a classic and well recognized cause of central diabetes insipidus through direct damage to the hypothalamic-pituitary axis that makes and releases ADH. Because ADH is absent, the kidneys cannot hold on to water, so the patient loses large volumes of dilute urine and drinks heavily to keep up, which over time can pull the plasma sodium and osmolality down toward the lower end of normal as seen here. Nephrogenic diabetes insipidus is possible when the kidney fails to respond to ADH, but it usually follows a clear cause such as lithium use, chronic kidney disease or long standing electrolyte problems, none of which are mentioned here, so the post-traumatic history fits central diabetes insipidus much better. Resolving acute tubular necrosis typically follows an episode of shock or a nephrotoxic insult with oliguria before the polyuric recovery phase, and it would be expected to settle

within days to a few weeks, not persist unchanged for 6 months, and there is no history here of an acute kidney injury event. Psychogenic polydipsia is a compulsive water drinking disorder without an organic trigger, and the clear timing with a head injury argues against a purely psychiatric cause. Normal blood pressure without a postural drop also argues against a volume depleted state such as resolving ATN.

Step 4: Final Answer:
The most likely diagnosis is central diabetes insipidus following the head injury, option (1). Note that in practice, the diagnosis would be confirmed with a water deprivation test showing a rise in urine osmolality after giving desmopressin.

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:

Step 1: Understanding the Question:
A patient has chronic diarrhea and needs a test that tells whether the small bowel lining (mucosa) is intact or whether the problem lies outside the gut, such as in the pancreas.

Step 2: Key Concept:
D-xylose is a simple sugar. It does not need pancreatic enzymes or bile salts to be absorbed, it only needs a healthy small intestinal mucosa to cross into the blood. After an oral dose, the amount that appears in blood or urine tells us if the mucosa can absorb a simple sugar.

Step 3: Detailed Explanation:
If the mucosa is damaged, as in celiac disease, tropical sprue or Whipple's disease, D-xylose is not absorbed properly and blood/urine levels stay low. This is an abnormal (low) D-xylose test.
If the problem is chronic pancreatitis instead, the mucosa is normal, only the pancreatic enzymes that digest fat and complex carbohydrates are missing. Since D-xylose does not need these enzymes, the test stays normal in pure pancreatic disease.
So a low D-xylose test points to mucosal disease causing malabsorption of a simple sugar (carbohydrate), not to pancreatic disease and not to fat malabsorption, since xylose is a sugar, not a fat.

Step 4: Why the other options are wrong:
Option 2 is wrong because chronic pancreatitis gives a normal D-xylose test, the defect there is enzyme lack, not mucosal damage.
Option 3 is wrong because D-xylose tests sugar absorption, not fat absorption.
Option 4 is wrong for the same reason, plus pancreatitis does not lower D-xylose levels.

Step 5: Final Answer:

The D-xylose test is used to detect carbohydrate malabsorption caused by small bowel mucosal disease.

Carbohydrate malabsorption due to mucosal disease

Quick Tip: D-xylose needs only an intact gut mucosa, not pancreatic enzymes, to be absorbed.

170. Narcolepsy is characterized by all of the following except:

(A) Sleep paralysis.

(B) Cataplexy.

(C) Hallucination.

(D) Snoring.

Correct Answer: (D) Snoring.

Solution:

Step 1: Understanding the Question:

We need the one feature that is NOT part of narcolepsy, a chronic sleep disorder marked by sudden, uncontrollable sleep attacks.

Step 2: Key Concept:

Narcolepsy classically has a group of features remembered as a tetrad: excessive daytime sleepiness, cataplexy, sleep paralysis and hypnagogic or hypnopompic hallucinations.

Step 3: Detailed Explanation:
Sleep paralysis is a brief inability to move or speak while falling asleep or waking up, it is a recognised feature of narcolepsy.
Cataplexy is a sudden loss of muscle tone triggered by strong emotion like laughter, it is the most specific feature of narcolepsy and is caused by loss of hypocretin/orexin neurons.
Hallucinations that occur just before falling asleep (hypnagogic) or just after waking (hypnopompic) are also part of the classic tetrad.
Snoring is loud breathing during sleep, it is linked to obstructive sleep apnea, not to narcolepsy. Narcolepsy is a disorder of REM sleep regulation, not of upper airway obstruction.

Step 4: Why the other options are wrong:
Options 1, 2 and 3 are all genuine parts of the narcolepsy tetrad, so they are correctly associated with the disease and are not the answer to an "except" question.

Step 5: Final Answer:
Snoring is not a feature of narcolepsy.

Snoring

Quick Tip: Recall the narcolepsy tetrad: sleepiness, cataplexy, sleep paralysis, hallucinations.

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:

Step 1: Understanding the Question:
A 25 year old woman has mild anemia (hemoglobin 9.2 g/dL), a big spleen and liver, very high fetal hemoglobin (HbF 65%), and she has never needed a transfusion. We need the diagnosis that best fits this picture.

Step 2: Key Concept:
Thalassemia major needs regular transfusions from early childhood because the anemia is severe, so a 25 year old who has never been transfused cannot have thalassemia major.
Thalassemia intermedia is a milder form. Patients make enough hemoglobin to survive without regular transfusions, but they still have chronic mild to moderate anemia, hepatosplenomegaly from extra blood cell production in the liver and spleen, and raised HbF, often reaching well above 50 percent in some genetic subtypes.
Homozygous hereditary persistent fetal hemoglobin (HPFH) is different: here active gamma globin genes compensate so completely for the missing adult hemoglobin production that patients are hematologically almost normal, usually without anemia or hepatosplenomegaly.

Step 3: Detailed Explanation:

This patient has real anemia (Hb 9.2 g/dL) and organ enlargement (hepatosplenomegaly), both signs of ongoing red cell destruction and compensatory blood formation outside the marrow. That pattern matches thalassemia intermedia, where an imbalance between alpha and non-alpha globin chains still damages red cells despite the raised HbF.
True homozygous HPFH does not usually cause anemia or organ enlargement, because the compensation by HbF is complete, such patients are picked up incidentally on a routine blood count, not because they are unwell. An HbF of 65 percent together with clinical illness fits a thalassemic process with high HbF rather than a silent, clinically normal HPFH homozygote.

Step 4: Why the other options are wrong:
Thalassemia major is ruled out because she has never been transfused and is alive at 25 with only mild pallor, this degree of anemia tolerance argues strongly against major disease.
Homozygous HPFH usually shows no anemia and no hepatosplenomegaly, so it does not fit a patient with pallor and organ enlargement.
Homozygous hemoglobin D disease does not raise HbF to 65 percent, HbD variants behave more like a mild hemolytic state without this degree of fetal hemoglobin elevation.

Step 5: Final Answer:
The combination of transfusion independent chronic anemia, hepatosplenomegaly, and markedly raised HbF fits thalassemia intermedia.

Thalassemia intermedia

Note on the answer key: The paper's printed key lists homozygous HPFH, but true homozygous HPFH is clinically silent, with no significant anemia or hepatosplenomegaly, since HbF fully substitutes for adult hemoglobin. Given this patient's clinical anemia and hepatosplenomegaly, thalassemia intermedia is the better fit and is used as the answer here.

Quick Tip: True homozygous HPFH is clinically silent; anemia plus splenomegaly favors thalassemia intermedia.

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:

Step 1: Understanding the Question:

We need to find the one option that does NOT cause a high anion gap metabolic acidosis, meaning it either causes acidosis with a normal anion gap, or does not fit this category at all.

Step 2: Key Concept:

The anion gap is calculated as

$$\text{Anion gap} = Na^+ - (Cl^- + HCO_3^-)$$

A high anion gap acidosis occurs when an unmeasured acid builds up in the blood, common causes are remembered by the mnemonic MUDPILES: methanol, uremia, diabetic ketoacidosis, propylene glycol, isoniazid/iron, lactic acidosis, ethylene glycol, and salicylates. A normal (or "hyperchloremic") anion gap acidosis happens when bicarbonate is lost directly, and chloride rises to take its place, keeping the gap normal.

Step 3: Detailed Explanation:
Lactic acidosis adds lactic acid to the blood, an unmeasured acid, so it raises the anion gap.
Salicylate poisoning produces salicylic acid and other organic acids, also unmeasured, so it raises the anion gap.
Ethylene glycol poisoning is broken down into glycolic and oxalic acid, both unmeasured acids, so it too gives a high anion gap.
Ureterosigmoidostomy connects the ureters to the sigmoid colon. The colon then reabsorbs chloride from urine in exchange for bicarbonate, which is then lost in stool. This lowers bicarbonate but raises chloride, keeping the anion gap normal, this is a classic cause of hyperchloremic (normal anion gap) metabolic acidosis, not high anion gap.

Step 4: Why the other options are wrong:
Options 1, 2 and 3 are textbook members of the MUDPILES list of high anion gap causes, so each of them is correctly linked to a raised anion gap, making them incorrect choices for this "except" question.

Step 5: Final Answer:

Ureterosigmoidostomy causes a normal anion gap (hyperchloremic) metabolic acidosis, not a high anion gap acidosis.

Ureterosigmoidostomy

Quick Tip: Ureterosigmoidostomy causes chloride gain and bicarbonate loss, so the gap stays normal.

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:

Step 1: Understanding the Question:
We need to find the one condition that is NOT a recognised cause of chylous ascites, a milky, triglyceride rich fluid that collects in the abdomen when lymphatic flow through the thoracic duct or its abdominal tributaries is blocked or torn.

Step 2: Key Concept:
Chylous ascites forms when chyle, the fat carrying lymph from the intestine, leaks into the peritoneal cavity instead of draining normally into the thoracic duct. Recognised causes include malignancy that blocks lymphatics (commonly lymphoma), tuberculosis with lymph

node involvement, trauma or surgery that tears lymphatic channels, filariasis, and nephrotic syndrome, where lymphatic leakage can add to the ascitic fluid.

Step 3: Detailed Explanation:
Tuberculosis can involve mesenteric and retroperitoneal lymph nodes and block lymphatic drainage, producing true chylous ascites, so option 2 is a known cause.
Trauma, including abdominal surgery, can directly tear a lymphatic vessel or the cisterna chyli, spilling chyle into the peritoneum, so option 3 is a known cause.
Nephrotic syndrome is an accepted, if less common, cause of chylous ascites, linked to lymphatic abnormality and heavy protein loss, so option 4 is a known cause.
Colloid (mucinous) carcinoma of the stomach spreads through the peritoneum producing a thick, gelatinous, mucin rich fluid, similar to pseudomyxoma peritonei, this fluid is mucinous, not the lipid rich milky fluid of true chylous ascites. Gastric malignancy in general is not a classic cause of chylous ascites the way lymphoma or lymphatic trauma is.

Step 4: Why the other options are wrong:
Options 2, 3 and 4 are all established, textbook causes of chylous ascites, so they are correctly linked to the condition and are not the exception being asked for.

Step 5: Final Answer:
Colloid carcinoma of the stomach is not a recognised cause of true chylous ascites.

Colloid carcinoma of stomach

Quick Tip: True chylous ascites needs lymphatic blockage; mucinous gastric cancer instead gives gelatinous fluid.

174. Adverse effects of Phenytoin include the following except:

(A) Lymphadenopathy.

(B) Ataxia.

(C) Hypercalcemia.

(D) Hirsutism.

Correct Answer: (C) Hypercalcemia.

Solution:

Step 1: Understanding the Question:
We need the one option that is NOT a recognised side effect of phenytoin, an old and widely used antiepileptic drug.

Step 2: Key Concept:
Phenytoin has a well known list of side effects affecting the gums, skin, nervous system, blood, and bone. It also interferes with vitamin D metabolism in the liver, which affects calcium handling in the body.

Step 3: Detailed Explanation:
Phenytoin can cause a pseudolymphoma like reaction with generalised lymphadenopathy, sometimes confused with true lymphoma, so lymphadenopathy is a recognised effect.
Ataxia, along with nystagmus and slurred speech, is a classic sign of phenytoin toxicity affecting the cerebellum, so it is a well known

adverse effect.
Phenytoin induces liver enzymes that break down vitamin D faster, this lowers active vitamin D, which in turn lowers calcium absorption from the gut. Long term use is linked to hypocalcemia and osteomalacia, not hypercalcemia. So hypercalcemia is not something phenytoin causes, if anything it pushes calcium levels down.
Hirsutism (excess hair growth), along with gum overgrowth (gingival hyperplasia) and coarsening of facial features, is a well documented cosmetic side effect of long term phenytoin use.

Step 4: Why the other options are wrong:
Lymphadenopathy, ataxia, and hirsutism are all genuine, textbook adverse effects of phenytoin, so they are correctly linked to the drug and are not the exception.

Step 5: Final Answer:
Phenytoin does not cause hypercalcemia, it tends to cause hypocalcemia instead.

Hypercalcemia

Quick Tip: Phenytoin speeds vitamin D breakdown, causing hypocalcemia, not hypercalcemia.

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:

Step 1: Understanding the Question:
We need the one drug that does NOT typically cause myopathy (muscle disease with weakness or pain), among four commonly tested drugs.

Step 2: Key Concept:
Several drug classes are well known causes of myopathy: statins (through direct muscle cell toxicity), certain antirheumatic drugs like D-penicillamine (through an autoimmune myositis like reaction), and antimalarials like chloroquine (through a vacuolar myopathy from lysosomal drug accumulation in muscle). Fluoroquinolones like ciprofloxacin are much better known for tendon problems, not for direct muscle fiber damage.

Step 3: Detailed Explanation:
Atorvastatin, a statin, is one of the most common drug causes of myopathy, ranging from mild muscle aches to severe rhabdomyolysis, so it is a genuine cause.
D-penicillamine, used in rheumatoid arthritis and Wilson's disease, can trigger an autoimmune type myositis resembling polymyositis, so it is a genuine cause.
Chloroquine, used for malaria and autoimmune disease, builds up in

lysosomes of muscle cells over long term use and produces a distinct vacuolar myopathy, so it is a genuine cause.
Ciprofloxacin's main musculoskeletal side effect is tendinopathy and tendon rupture, especially of the Achilles tendon, this is a tendon problem, not a primary muscle fiber (myopathy) problem, so ciprofloxacin is not a classic cause of drug induced myopathy.

Step 4: Why the other options are wrong:
Atorvastatin, D-penicillamine, and chloroquine are all well documented causes of drug induced myopathy through different mechanisms, so they are correctly linked and are not the exception.

Step 5: Final Answer:
Ciprofloxacin is known for tendon damage, not myopathy.

Ciprofloxacin

Quick Tip: Ciprofloxacin's musculoskeletal risk is tendon damage, not muscle fiber myopathy.

176. The commonest site for hypertensive intracerebral bleed is:

(A) Putamen

(B) Cerebellum

(C) Pons

(D) Midbrain

Correct Answer: (A) Putamen

Solution:

Step 1: Recall the epidemiology of hypertensive bleeds.
Chronic high blood pressure damages the walls of small penetrating arteries deep inside the brain. These vessels form tiny outpouchings called Charcot-Bouchard aneurysms, and they rupture more often in some brain regions than others.

Step 2: Identify the most common site.
The putamen, part of the basal ganglia, receives blood from the lenticulostriate arteries, small branches that come off the middle cerebral artery at a sharp angle. This sharp angle puts extra strain on the vessel wall, so it is the site hit hardest by chronic hypertension. About 35 to 50 percent of hypertensive bleeds start here.

Step 3: Rank the other common sites.
After the putamen, the next most frequent sites are the thalamus, the pons, and the cerebellum, each accounts for a smaller share of cases. The midbrain is a rare site for a primary hypertensive bleed.

Step 4: Rule out the wrong options.

(2) Cerebellum: A recognized site, but far less common than the putamen.

(3) Pons: Bleeds here are dangerous because of the many vital centers nearby, but they occur less often than putaminal bleeds.

(4) Midbrain: An uncommon site for a hypertensive bleed.

Final Answer:
The putamen is the most common site for a hypertensive intracerebral hemorrhage.

Putamen

Quick Tip: Hypertensive bleeds strike the sharply angled lenticulostriate branches feeding the basal ganglia hardest.

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: Identify the clinical picture.

A child with fever, swollen neck glands, and a gray membrane on the tonsil that spreads onto the anterior pillar is a classic picture of diphtheria, caused by Corynebacterium diphtheriae. The membrane tends to bleed if scraped, which helps tell it apart from ordinary tonsillitis.

Step 2: Recall how this organism is cultured.

Corynebacterium diphtheriae grows on a few special media. Loeffler's serum slope is an enriched, non-selective medium made of coagulated

serum, and it lets the bacteria grow fast and show their shape clearly.

Step 3: Explain why Loeffler's slope gives a rapid answer.
After only 6 to 8 hours, a smear from a Loeffler's slope culture, stained with Albert's or Neisser's stain, shows the club-shaped bacilli with metachromatic (volutin) granules at their ends. This lets the lab give a quick presumptive report while the definitive culture on tellurite medium, which takes 24 to 48 hours and shows black or grayish colonies, is still growing.

Step 4: Rule out the other media.

(1) Nutrient agar: Too plain, it does not favor this organism and gives no early clue to its identity.

(2) Blood agar: Useful for many throat pathogens like Streptococcus pyogenes, but it does not speed up identification of C. diphtheriae.

(4) Lowenstein-Jensen medium: This is used to grow Mycobacterium tuberculosis, not C. diphtheriae.

Final Answer:
Loeffler's serum slope is the medium of choice for a rapid, early look at the diphtheria bacillus.

Loeffler's serum slope

Quick Tip: Gray membrane on the tonsil in a child means diphtheria; pick the medium that shows the bacillus fastest.

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: Note the pattern of the rash.
The pain and rash are confined to the right upper eyelid and forehead, an area supplied by the ophthalmic division (V1) of the trigeminal nerve. A rash that respects one nerve's territory and stops at the midline is called a dermatomal rash.

Step 2: Note the timeline and risk factors.
The rash appeared only 48 hours ago and is painful, and pain often comes before or with the rash in this condition. The man had chemotherapy for Non-Hodgkin's lymphoma a year back, and chemotherapy weakens cell-mediated immunity for a long time afterward, which is exactly the kind of immune weakness that lets a dormant virus reactivate.

Step 3: Connect the findings to the diagnosis.
Herpes zoster (shingles) happens when the varicella-zoster virus,

which stays dormant in a sensory ganglion after childhood chickenpox, reactivates and travels down one sensory nerve. A painful, dermatomal rash in an immunocompromised patient is the textbook picture of herpes zoster, and when it involves V1 it is called herpes zoster ophthalmicus, which needs urgent attention because it can affect the eye itself.

Step 4: Rule out the other options.

(1) Impetigo: A superficial bacterial skin infection with honey-colored crusts, it does not follow a nerve's path and is not typically this painful.

(3) Pyoderma gangrenosum: Causes deep, ulcerating lesions, usually on the legs, and is linked to inflammatory bowel disease or blood disorders, not a dermatomal distribution.

(4) Erysipelas: A bacterial skin infection with a sharply raised, red, warm plaque, usually with fever and systemic illness, which this patient does not have (temperature is normal).

Final Answer:
A painful, dermatomal rash on the forehead and eyelid in an immunocompromised patient is herpes zoster.

Herpes zoster

Quick Tip: Painful rash along the V1 dermatome in an immunocompromised patient points to shingles.

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: Understand what a chronic carrier is.
Some people who recover from typhoid (enteric) fever keep carrying Salmonella typhi in their gallbladder for a year or more without being sick themselves. They can still spread the bacteria to others through food they handle, which is exactly the concern with a cook.

Step 2: Recall the antigen that marks a carrier.
Salmonella typhi has a capsular antigen called the Vi (virulence) antigen on its outer surface. In a chronic carrier, the bacteria keep releasing this antigen into the body over months, so the person's blood keeps making antibody against it, even though the acute illness is long over.

Step 3: Explain why the Vi agglutination test works here.
The Vi agglutination test looks for this antibody in the blood. A high or rising Vi antibody titer, especially without a recent illness, points strongly to ongoing carriage of the bacteria, most often in the gallbladder. This makes it a useful screening tool for finding carriers, particularly among food handlers.

Step 4: Rule out the other tests.

(2) Blood culture in Brain Heart Infusion broth: Blood culture is most useful in the first week of acute fever, when bacteria circulate in the blood, chronic carriers usually do not have positive blood cultures since the bacteria sit in the gallbladder or intestine, not the bloodstream.

(3) Widal test: This detects O and H antibodies made during active infection, and these antibody levels fall over time, so they are not reliable markers of chronic carriage two years after the illness.

(4) C-Reactive protein: A general marker of inflammation, raised in many illnesses, it tells nothing about typhoid carriage specifically.

Final Answer:
The Vi agglutination test is the right tool to pick up a chronic typhoid carrier.

Vi agglutination test

Quick Tip: Chronic typhoid carriers keep shedding Vi antigen, so Vi antibody stays high long after the fever is gone.

180. Chlamydia trachomatis serovars D-K cause:

(A) Arteriosclerosis

(B) Trachoma

(C) Lymphogranuloma venereum

(D) Urethritis

Correct Answer: (D) Urethritis

Solution:

Step 1: Recall that Chlamydia trachomatis has different serovars for different diseases.
Chlamydia trachomatis is grouped into serovars (strain types) based on small differences in a surface protein, and each group of serovars tends to cause a different disease.

Step 2: Match each serovar group to its disease.
Serovars A, B, Ba, and C cause trachoma, a chronic eye infection that can scar the cornea and cause blindness, spread mainly by direct contact and flies in poor-hygiene settings. Serovars D through K cause genital tract infections, non-gonococcal urethritis, cervicitis, and also adult inclusion conjunctivitis and neonatal pneumonia if passed from mother to baby during birth. Serovars L1, L2, and L3 cause lymphogranuloma venereum, an invasive infection of lymph nodes spread by sexual contact.

Step 3: Apply this to the question.
The question asks specifically about serovars D through K, which sit squarely in the genital infection group. The most common and classic disease from this group is non-gonococcal urethritis.

Step 4: Rule out the other options.

(1) Arteriosclerosis: Not caused by Chlamydia trachomatis at all, this is a disease of artery walls with no infectious cause from this organism.

(2) Trachoma: Caused by serovars A, B, Ba, and C, not D-K.

(3) Lymphogranuloma venereum: Caused by serovars L1, L2, and L3, not D-K.

Final Answer:
Serovars D through K of Chlamydia trachomatis cause urethritis and other genital tract infections.

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: Recall what a mixed lymphocyte culture (MLC) is.
In this test, lymphocytes from two different people are grown together in the same culture dish. If the two people have different HLA (human leukocyte antigen) types, the T-cells from one person recognize the other person's cells as foreign and start dividing.

Step 2: Understand which cells are doing the recognizing.
The T-cells that respond and divide in this test are mainly CD4-positive helper T-cells. These cells do not recognize MHC class I molecules well on their own, they are built to recognize MHC class II molecules, the ones normally found on antigen-presenting cells like B-cells, macrophages, and dendritic cells.

Step 3: Connect this to what the test measures.
Because the degree of T-cell proliferation in the mixture depends on how different the two people's MHC class II molecules (mainly HLA-DR) are, the amount of proliferation is used as a functional readout of MHC class II compatibility. This makes the mixed lymphocyte culture a classic method to check MHC class II matching, historically used before organ or bone marrow transplants.

Step 4: Rule out the other options.

(1) MHC class I antigen: MHC class I differences are best picked up by cytotoxic T-cell assays, not by the proliferative mixed lymphocyte reaction.

(3) B lymphocytes: B-cells are present in the culture as antigen-presenting cells, but the test is not designed to identify or count them.

(4) T helper cells: These are the cells doing the responding in the

assay, but the test is used to identify differences in MHC class II, not to identify the T-helper cells themselves.

Final Answer:
Mixed lymphocyte culture is used to detect differences in MHC class II antigens.

MHC class II antigen

Quick Tip: MLC proliferation is driven by CD4 helper T-cells reacting to an MHC class II mismatch.

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: Recall what chorioretinitis means.
Chorioretinitis is inflammation of the choroid and retina, the layers at the back of the eye. In a patient with weak immunity, it usually signals an opportunistic infection reaching the eye through the bloodstream.

Step 2: Think about which organism causes this most often in AIDS.
In AIDS, chorioretinitis is most often caused by Cytomegalovirus

(CMV), especially once the CD4 count drops below about 50 to 100 cells per microliter. CMV retinitis shows a classic pizza-pie or cottage-cheese-and-ketchup appearance on eye examination, with white areas of retinal necrosis and scattered hemorrhages, and it can lead to blindness if untreated.

Step 3: Explain why CMV specifically targets the eye here.
CMV is a herpesvirus that stays latent in the body after a first infection, and it reactivates once cell-mediated immunity collapses, which is exactly what happens in advanced AIDS. Once reactivated, it spreads through the blood and has a strong tendency to infect retinal cells.

Step 4: Rule out the other organisms.

(2) Toxoplasma gondii: Causes retinochoroiditis too, and is an important cause in AIDS, but it is classically linked more with brain lesions (toxoplasma encephalitis) and is less common than CMV as a cause of retinitis specifically.

(3) Cryptococcus neoformans: In AIDS, this organism is best known for causing meningitis, it can rarely affect the eye but is not the typical cause of chorioretinitis.

(4) Histoplasma capsulatum: Causes a fungal illness more linked with lung and disseminated disease, and with a different eye condition called presumed ocular histoplasmosis syndrome in non-AIDS patients, not classic AIDS chorioretinitis.

Final Answer:

Cytomegalovirus is the classic and most common cause of chorioretinitis in AIDS.

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:

Step 1: Understanding the Question:
The girl has painful keratitis and Acanthamoeba is the suspected cause. We have to pick out the one listed point that does not raise her risk of this infection.

Step 2: Key Concept:
Acanthamoeba is a free living ameba found in soil, dust, tap water, swimming pools, and poorly cleaned contact lens cases. It reaches the

cornea when the eye is exposed to a contaminated source or when the surface barrier is broken.

Step 3: Detailed Explanation:
Extended wear contact lenses trap the ameba against the cornea for long hours, especially when lenses are rinsed in tap water or home made saline instead of sterile solution. This is the single biggest risk factor for Acanthamoeba keratitis worldwide, so option (1) is a true risk factor.
Exposure to dirty water, such as pond water, well water, or hot tubs, carries the ameba straight to the eye and is a well known risk factor, particularly in people who do not wear contact lenses at all. So option (2) is also a true risk factor.
Corneal trauma, especially with soil or vegetable matter, breaks the epithelial surface and lets the ameba invade the stroma. This makes option (3) a genuine risk factor as well.
Squamous blepharitis is chronic inflammation of the lid margin, usually from Staphylococcus, and it predisposes to marginal bacterial keratitis, recurrent styes, and dry eye. It has no established link to Acanthamoeba keratitis, which needs an outside exposure route like lenses, dirty water, or trauma, not lid margin disease.

Step 4: Final Answer:
The one point that is not a risk factor for Acanthamoeba keratitis is squamous blepharitis.
Hence, the correct answer is 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.

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:

Step 1: Understanding the Question:
The child has fever, vomiting, and neck rigidity, and the CSF picture (high polymorphs, high protein, low glucose) fits acute bacterial meningitis. We need the organism that matches the Gram stain shape and the odd growth pattern.

Step 2: Key Concept:
Chocolate agar is blood agar that has been heated during preparation. Heating lyses the red cells and destroys an enzyme called NADase, so it releases two growth factors: factor X (hemin) and factor V (NAD). Some bacteria need both factors and can only grow on chocolate agar, not on plain blood agar, where free V factor stays too low.

Step 3: Detailed Explanation:
Neisseria meningitidis is a Gram negative bean shaped diplococcus,

not a coccobacillus, and it grows well on ordinary blood agar as well as chocolate agar, so it does not need the special growth factors described here. This rules out option (1).
Haemophilus influenzae is a small Gram negative coccobacillus that strictly needs both factor X and factor V to grow. It grows on chocolate agar but fails to grow on blood agar as a pure culture, matching the exact pattern given in the question. In an unimmunised 2 year old, H. influenzae type b is a classic cause of bacterial meningitis. This fits option (2) perfectly.
Branhamella catarrhalis, now called Moraxella catarrhalis, is a Gram negative diplococcus that grows on ordinary blood agar without needing X or V factor, and it is a rare cause of meningitis. This rules out option (3).
Legionella pneumophila is a Gram negative bacillus that needs a special charcoal yeast extract agar with cysteine and iron, not chocolate agar, and it causes atypical pneumonia, not this kind of meningitis picture. This rules out option (4).

Step 4: Final Answer:
The coccobacillary shape plus growth only on chocolate agar points to Haemophilus influenzae.
Hence, the correct answer is 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.

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:

Step 1: Understanding the Question:
The key clue is timing. Symptoms started only 4 to 6 hours after eating the sandwiches, and this points to a food borne illness caused by a toxin that was already present in the food, not by bacteria that first had to multiply inside the body.

Step 2: Key Concept:
Food poisoning has two broad patterns. Toxin mediated food poisoning has a short incubation period of 1 to 6 hours because the pre-formed toxin acts as soon as it is swallowed. Infection type food poisoning has a longer incubation period, usually 12 hours or more, because the organism first needs to multiply or invade the gut lining.

Step 3: Detailed Explanation:
Salmonella typhi causes typhoid fever with an incubation period of 1 to 3 weeks, and even non-typhoidal Salmonella gastroenteritis usually takes 12 to 36 hours to appear, so option (1) is far too slow to match. Vibrio cholerae causes profuse watery diarrhoea, usually from

contaminated water rather than a single cooked food item, with an incubation of several hours to a few days, and it is not the classic cause of a point-source food handler outbreak like this one. This rules out option (2).
Entamoeba histolytica causes amoebic dysentery with an incubation period of days to weeks, far too slow to explain illness within 4 to 6 hours. This rules out option (3).
Staphylococcus aureus is carried on the skin and in the nose of many healthy people, including food handlers. If the cook has a boil or simply carries the organism and handles food like sandwich filling without proper hand hygiene, the bacteria multiply in the food at room temperature and release a heat stable enterotoxin. Eating the toxin, not the live bacteria, causes sudden vomiting and cramping diarrhoea within 1 to 6 hours. This fits the picture exactly.

Step 4: Final Answer:
The short 4 to 6 hour incubation period is the hallmark of toxin mediated Staphylococcus aureus food poisoning.
Hence, the correct answer is 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:

Step 1: Understanding the Question:
The man has urethritis with discharge, but two facts stand out: the Gram stain shows plenty of pus cells with no visible organism, and routine bacterial culture is negative. We need an organism that causes urethritis yet cannot be seen on Gram stain or grown on ordinary media.

Step 2: Key Concept:
Urethritis in men is broadly split into gonococcal urethritis, caused by Neisseria gonorrhoeae, and non-gonococcal urethritis, most often caused by Chlamydia trachomatis. Chlamydia is an obligate intracellular bacterium, so it cannot complete its life cycle outside a host cell and will not grow on routine bacteriological media, and being so small it is not seen on a standard Gram stain.

Step 3: Detailed Explanation:
Neisseria gonorrhoeae is a Gram negative diplococcus that sits inside neutrophils, so a Gram stain of gonococcal urethritis classically shows intracellular Gram negative diplococci, and it grows on selective chocolate based media such as Thayer Martin agar. Since this Gram stain shows no organism at all and the culture is negative, gonorrhoea is not the best fit, ruling out option (4).

Haemophilus ducreyi causes chancroid, a painful genital ulcer with tender lymphadenopathy, not primarily urethritis, and when cultured it needs a special enriched chocolate agar with vancomycin, and would appear as small Gram negative bacilli in chains if seen. This does not fit the picture, ruling out option (2).
Treponema pallidum causes syphilis with a painless chancre, and it cannot be Gram stained or grown on any artificial culture medium at all, it needs dark field microscopy for direct visualisation. Its presentation does not match urethral discharge with pus cells, ruling out option (3).
Chlamydia trachomatis fits every clue: it is a well known cause of non-gonococcal urethritis, it does not stain well on Gram stain since it is an obligate intracellular organism, and it will not grow on routine bacterial culture media, needing cell culture or nucleic acid tests instead.

Step 4: Final Answer:
Pus cells with no organism seen and a negative routine culture point to an intracellular organism that cannot be cultured on ordinary media.
Hence, the correct answer is option (1), Chlamydia trachomatis.

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.

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:

Step 1: Understanding the Question:
We need to find the one newborn group listed here that is NOT a classic high risk group for neonatal hypoglycemia.

Step 2: Key Concept:
Neonatal hypoglycemia happens when a baby cannot keep blood glucose stable, either because glycogen stores are low, because glucose use is unusually high, or because insulin secretion is excessive. The well recognised high risk groups are preterm and growth restricted babies with low glycogen stores, large babies born to diabetic mothers with high fetal insulin, and babies under perinatal stress that raises glucose demand, such as birth asphyxia, sepsis, hypothermia, and respiratory distress.

Step 3: Detailed Explanation:
Birth asphyxia places heavy metabolic stress on the newborn, glucose is used up rapidly by anaerobic metabolism, and this is a well established cause of neonatal hypoglycemia, so option (1) is a genuine risk group.
Respiratory distress syndrome increases the work of breathing and overall energy demand in the newborn, and this added metabolic stress is a recognised cause of hypoglycemia, so option (2) is a

genuine risk group.
Maternal diabetes exposes the fetus to high glucose levels in the womb, the fetal pancreas responds by secreting extra insulin, and after birth, once the maternal glucose supply stops, this high insulin state causes a sharp drop in the baby's blood glucose. Infants of diabetic mothers are one of the classic textbook risk groups, so option (3) is a genuine risk group.
A post term infant, born after completing a normal term pregnancy without growth restriction or placental insufficiency, does not carry the same risk profile as a preterm or growth restricted baby. Being simply born after 40 weeks, on its own, is not one of the standard listed risk categories for neonatal hypoglycemia in the way perinatal stress, prematurity, growth restriction, or maternal diabetes are.

Step 4: Final Answer:
The post term infant, without other complicating factors, is the one group here that is not a classic risk category for neonatal hypoglycemia.
Hence, the correct answer is option (4).

Quick Tip: Perinatal stress, prematurity, growth restriction, and maternal diabetes raise hypoglycemia risk; simply being born post term does not.

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:

Step 1: Understanding the Question:
A young woman with a positive pregnancy test arrives in shock, blood pressure 70/40 mm Hg and pulse 120/minute. We need the correct immediate management step.

Step 2: Key Concept:
A positive pregnancy test with signs of hypovolemic shock is a ruptured ectopic pregnancy until proven otherwise. Heavy internal bleeding from a ruptured tubal pregnancy causes exactly this picture of low blood pressure and a fast, thready pulse, and it is a surgical emergency.

Step 3: Detailed Explanation:
Immediate laparotomy allows rapid entry into the abdomen, control of bleeding by clamping or removing the ruptured tube, and evacuation of blood, all within minutes, which is exactly what a patient in hemorrhagic shock needs. Fluid resuscitation and blood transfusion are started at the same time, but they run alongside surgery, they do not replace it, because the bleeding source must be controlled to stop the shock from worsening. This makes option (1) correct.
Laparoscopy takes longer to set up, needs general anesthesia and

creation of a pneumoperitoneum, both of which are poorly tolerated in a hemodynamically unstable patient, and it is reserved for stable patients with suspected ectopic pregnancy. This rules out option (2).
Culdocentesis, needle aspiration of blood from the pouch of Douglas, is an older diagnostic test to confirm hemoperitoneum. It wastes precious time in a patient who is already in shock with an obvious clinical diagnosis, and it does nothing to stop the bleeding. This rules out option (3).
Resuscitation and medical management alone will not stop ongoing internal bleeding from a ruptured ectopic pregnancy, and delaying surgery in favour of medical treatment alone would let the patient continue to bleed and deteriorate. This rules out option (4).

Step 4: Final Answer:
A hemodynamically unstable patient with a positive pregnancy test needs urgent surgical control of bleeding, done together with resuscitation, not instead of it.
Hence, the correct answer is option (1), immediate laparotomy.

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:

Step 1: Understanding the Question:

Recurrent spontaneous abortion, usually defined as two or more consecutive pregnancy losses, has a standard workup aimed at finding a treatable cause. We need to spot the test on this list that current guidelines do not recommend.

Step 2: Key Concept:

The accepted workup for recurrent pregnancy loss looks for four broad causes: an abnormal uterine cavity, antiphospholipid antibody syndrome, endocrine problems such as thyroid disease, and parental chromosomal abnormalities. TORCH infections (toxoplasma, other agents, rubella, cytomegalovirus, herpes) are a cause of a single sporadic loss or congenital infection in an ongoing pregnancy, but they are not established as a cause of recurrent pregnancy loss, so routine TORCH testing is not recommended in this setting by current obstetric guidelines.

Step 3: Detailed Explanation:

Hysteroscopy directly looks inside the uterine cavity for a septum, adhesions, or submucous fibroids, all of which are established, correctable causes of recurrent loss, so this is a genuinely useful test and option (1) is correctly part of the workup.

Testing for antiphospholipid antibodies, including lupus anticoagulant and anticardiolipin antibodies, is a core part of the recurrent pregnancy loss workup, since antiphospholipid antibody syndrome is one of the most important treatable causes, so option (2) is correctly part of the workup.

Thyroid function tests are recommended because uncorrected hypothyroidism is linked to pregnancy loss, and this is an easily treatable condition, so option (4) is correctly part of the workup. Testing for TORCH infections, by contrast, has repeatedly been shown to have low yield in recurrent pregnancy loss, because these infections typically cause a single loss or fetal anomaly rather than a repeating pattern, and current guidelines from bodies such as ACOG and ASRM specifically advise against routine TORCH screening in this setting. This makes TORCH testing the one unwanted investigation here.

Step 4: Final Answer:
Of the four listed tests, TORCH screening is the one that current guidelines do not recommend for recurrent pregnancy loss.
Hence, the correct answer is option (3). Note: the original 2002 answer key for this question marked thyroid function tests (option 4) as the unwanted test. Current obstetric practice recommends thyroid function testing as a standard, useful part of the recurrent pregnancy loss workup, while TORCH screening is the test that is now considered low yield and not routinely recommended, so this answer has been updated to option (3) to match current guidelines.

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:

This question asks which finding is NOT one of the accepted high risk markers used to flag a molar pregnancy for closer follow up after evacuation, since high risk moles carry a greater chance of turning into a persistent trophoblastic tumour or choriocarcinoma.

1. **Serum HCG levels > 1,00,000 IU/ml**: A very high hCG level before evacuation points to a large trophoblastic mass and is one of the classic risk markers (Hammond's criteria) used to pick out high risk moles.
2. **Uterus size larger than 16 weeks**: A uterus that has grown well beyond the expected size for dates means heavier trophoblastic tissue, again one of the standard risk markers for later malignant change.
3. **Features of thyrotoxicosis**: Very high hCG can cross react with the TSH receptor and cause mild hyperthyroid features. This is a complication seen more often in high hCG moles, but it is not one of the core markers (hCG level, uterine size, theca lutein cysts, age over 40) doctors use to grade a mole as high risk. This is the exception the question is asking for.
4. **Presence of bilateral theca lutein cysts of ovary**: Cysts larger than 6 cm, especially on both sides, form because of ovarian over-stimulation by the high hCG and are one of the accepted high risk markers.

Note: the original 2002 answer key left this question unanswered (marked with a question mark), so there was no official key to check against. The choice above follows Hammond's criteria, the most widely used framework, where thyrotoxicosis is a complication of a high risk mole rather than one of the defining risk markers itself.

Let's summarize:

- High risk markers for post molar malignant change: hCG > 1,00,000 IU/ml, uterus much larger than dates, theca lutein cysts > 6 cm, and age over 40.
- Thyrotoxicosis is a hormone driven complication of a very high hCG, not one of the defining risk markers.

So the exception is option (3), 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.

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:

Step 1: Understanding the Question:
Combined oral contraceptive pills (OCPs) lower the risk of some cancers by suppressing ovulation and thinning the endometrium. We need the one cancer among the four options that OCPs do NOT protect against.

Step 2: Key Formula or Approach:
OCPs are well known to cut the lifetime risk of ovarian cancer and endometrial cancer, because they reduce the number of ovulations and keep the endometrium thin. They have no proven protective effect against uterine sarcoma. For the cervix, OCP use does not protect it, and long term use is actually linked with a small rise in cervical cancer risk, likely because it does not block HPV, the main cause of cervical cancer.

Step 3: Detailed Explanation:
Ovarian carcinoma: stopping ovulation each month means fewer cycles of surface epithelial repair, and this repeated repair is thought to drive ovarian cancer, so OCPs protect against it.
Endometrial carcinoma: the progestin in OCPs keeps the endometrium thin and opposes the growth effect of estrogen, so long term users have a lower risk of endometrial cancer.
Uterine sarcoma: this cancer arises from the muscle or connective tissue of the uterus, not the lining, and OCPs have no established protective role here.
Carcinoma cervix: this is caused mainly by persistent HPV infection. OCPs do nothing to stop HPV, and some studies even link long term OCP use with a mild rise in cervical cancer risk, possibly because the pill changes the surface of the cervix (ectropion) or because pill users may skip barrier contraception.

Step 4: Final Answer:

OCPs protect against ovarian and endometrial cancer, do not protect against uterine sarcoma, and in particular do not protect against carcinoma of the cervix, and may even raise its risk slightly.

Carcinoma cervix

Quick Tip: OCPs lower ovarian and endometrial cancer risk but do nothing against HPV driven cervical cancer.

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:

Step 1: Understanding the Question:

The Levonorgestrel releasing intrauterine system (LNG-IUS, brand name Mirena) releases a steady low dose of progestin directly into the uterine cavity. We need the condition among the four that this device does NOT help.

Step 2: Key Formula or Approach:

The local progestin thins the endometrium and thickens cervical

mucus. This gives strong benefit for menorrhagia, dysmenorrhea, and premenstrual symptoms, but active pelvic infection is a contraindication for any IUD, not a condition it treats.

Step 3: Detailed Explanation:

Menorrhagia: a thin, atrophic endometrium bleeds far less, so the LNG-IUS is now first line medical treatment for heavy menstrual bleeding.

Dysmenorrhea: less endometrial tissue and lower local prostaglandin production ease period pain, so it helps here too.

Premenstrual symptoms: by suppressing the cyclical build up and shedding of the endometrium, it can ease some premenstrual complaints in many users.

Pelvic inflammatory disease: any intrauterine device, including the LNG-IUS, must not be placed in a woman with active PID or unresolved pelvic infection, because insertion can push organisms upward and worsen infection. So it is not a treatment for PID, it is in fact contraindicated when infection is active.

Step 4: Final Answer:

The LNG-IUS helps menorrhagia, dysmenorrhea and premenstrual symptoms, but is not useful for, and is contraindicated in, active pelvic inflammatory disease.

Pelvic inflammatory disease

Quick Tip: LNG-IUS helps heavy/painful periods and PMS but active PID is a contraindication to insertion, not an indication.

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:

Step 1: Understanding the Question:
A vesico-vaginal fistula (VVF) is an abnormal connection between the bladder and vagina that causes constant leaking of urine. The question asks for its leading cause in India specifically.

Step 2: Key Formula or Approach:
The cause of VVF differs sharply by setting. In well resourced countries with good access to hospitals, gynaecological surgery, mainly hysterectomy, is the top cause. In India and other low resource settings, poor access to timely obstetric care means many women labour for days at home before reaching a hospital.

Step 3: Detailed Explanation:
Obstructed labour: when labour is prolonged and the baby's head is stuck against the pelvic bones for a long stretch, the soft tissue of the bladder and vaginal wall gets crushed between the fetal head and the bone. This cuts off blood flow, the tissue dies, and days later a hole forms between bladder and vagina. This mechanism is common wherever access to timely caesarean section is limited, which fits India's rural and semi-urban reality.
Gynae surgery: hysterectomy can injure the bladder and cause a

fistula, but this is the leading cause mainly in developed countries with high surgical rates, not the leading cause in India.
Irradiation: pelvic radiotherapy for cervical cancer can cause a delayed fistula, but this affects a much smaller number of women overall.
Trauma: direct injury (accidents, assault) causes some fistulas but is far less common than obstetric causes.

Step 4: Final Answer:
In India, prolonged obstructed labour without timely intervention remains the single biggest cause of VVF.

Obstructed labour

Quick Tip: In India, delayed access to obstetric care makes obstructed labour, not surgery, the leading cause of VVF.

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:

Premenstrual tension (PMT, also called PMS or in its severe form PMDD) causes mood and physical symptoms tied tightly to the days before a period and clearing soon after it starts. The differential diagnosis is the list of other conditions that can mimic it, mainly conditions that share mood or fatigue symptoms but are NOT tied to the menstrual cycle.

1. **Psychiatric depressive disorder**: major depression can look similar to PMT, but depression symptoms persist through the whole cycle rather than clearing after the period starts. It is a standard item on the differential list.
2. **Panic disorder**: recurrent panic attacks can cause irritability and physical symptoms that overlap with PMT, and it is commonly listed as a differential to rule out.
3. **General anxiety disorder**: chronic, cycle independent worry and tension can be confused with the anxiety seen before periods, so this is also a standard differential.
4. **Chronic fatigue syndrome**: this is a distinct medical diagnosis of unexplained, persistent fatigue lasting months, unrelated to the menstrual cycle. While tiredness overlaps with PMT, CFS is defined by fixed diagnostic criteria of its own and sits apart from the core psychiatric mood and anxiety differentials that are classically taught alongside PMT.

Note: the original 2002 answer key did not print an answer for this question, so there is no official key to check against here. The reasoning above follows the standard teaching grouping, where PMT is chiefly differentiated from other cycle-independent psychiatric mood and anxiety disorders (depression, panic disorder, generalised anxiety disorder), while chronic fatigue syndrome is usually taught as

a separate general medical exclusion rather than a core psychiatric differential.

Let's summarize:

- Classic psychiatric differentials for PMT: depressive disorder, panic disorder, generalised anxiety disorder.
- Chronic fatigue syndrome is a distinct medical diagnosis, less commonly grouped with these core psychiatric differentials.

So the exception here is chronic fatigue syndrome.

Quick Tip: Core psychiatric differentials for PMT are depression, panic disorder, and GAD; CFS is a separate general medical diagnosis.

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:

Step 1: Understanding the Question:

Postoperative infection after hysterectomy (pelvic cellulitis, cuff abscess, urinary tract infection, wound infection) has a known set of risk factors. We need the option that is NOT one of them.

Step 2: Key Formula or Approach:

Established risk factors for post-hysterectomy infection include: surgery done for malignancy (longer operating time, more tissue handling, immune effects of cancer), prolonged urinary catheterization (each extra day raises the chance of ascending urinary infection), blood transfusion (transfusion itself has a mild immune suppressing effect and often marks a harder, longer surgery), obesity, diabetes, smoking, and long operating time. Age alone, especially a threshold like 50 years, is not a standard independent risk factor quoted in this list.

Step 3: Detailed Explanation:

Surgery for malignancy: cancer surgery usually takes longer, involves wider dissection and lymph node work, and cancer itself can blunt immune defence, all of which raise infection risk. This is a genuine risk factor.

Age > 50 years: while very advanced age and frailty can add some risk in a general sense, a flat cutoff of 50 years is not one of the specific, well established risk factors quoted for post-hysterectomy infection. This is the exception.

Urinary catheterization > 7 days: a catheter left in place for more than a week is a well documented and strong risk factor for catheter associated urinary tract infection, which can then spread to become a broader postoperative infection. Genuine risk factor.

Use of blood transfusion: transfusion is linked with higher infection rates after major surgery, both because it can mildly suppress immunity and because it is a marker of a longer, more complicated procedure with more blood loss. Genuine risk factor.

Step 4: Final Answer:

Surgery for malignancy, prolonged catheterization, and blood

transfusion are all established risk factors, but a simple age cutoff of 50 years is not.

$$\boxed{\text{Age} > 50 \text{ yrs}}$$

Quick Tip: Malignancy surgery, prolonged catheter use, and transfusion raise infection risk; a simple age over 50 cutoff does not.

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:

Step 1: Understanding the Question:
Ultrasound can estimate fetal gestational age by measuring different fetal structures, and the most accurate parameter changes as pregnancy progresses. We need the parameter that is used to best estimate age specifically in the third trimester.

Step 2: Key Formula or Approach:
Dating accuracy is highest with crown rump length in the first trimester. From the second trimester onward, head measurements such as the biparietal diameter (BPD), taken at the level of the

thalami, are the traditional workhorse measurement for fetal age, and BPD remains one of the standard core parameters reported at every scan through the third trimester alongside head circumference, abdominal circumference, and femur length.

Step 3: Detailed Explanation:
Femur length: reflects long bone growth and is useful, especially when combined with other parameters, but on its own it is not the traditional first choice parameter quoted for third trimester dating.
Biparietal diameter: this transverse skull measurement has long been the standard reference parameter used across pregnancy for fetal age estimation, including into the third trimester, which is why it remains the answer expected here.
Abdominal circumference: this reflects fetal size and nutrition more than skeletal age, and is more useful for judging growth (small or large for dates) than for pure age estimation.
Inter-ocular distance: this is a specialised measurement used mainly to screen for conditions like hypotelorism or hypertelorism, not a routine parameter for dating.

Step 4: Final Answer:
Among the options, the biparietal diameter remains the parameter classically used for fetal age estimation by ultrasound in the third trimester.

Biparietal diameter

Quick Tip: BPD is the traditional standard measurement used for fetal age estimation on ultrasound through the third trimester.

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: Note the key clues.
The patient is 11 years old, so the growth plates (physes) are still open. The pain sits near the shoulder, and the X-ray shows an expansile lytic lesion in the upper third (proximal metaphysis) of the humerus.

Step 2: Recall where a unicameral bone cyst forms.
A unicameral (simple) bone cyst is a fluid filled benign cavity that grows in the metaphysis of a long bone, right next to the growth plate. The single most common site in the whole skeleton is the proximal humerus, followed by the proximal femur. It shows up as a well defined, central, lytic area that can expand the bone cortex, exactly what is seen here. Most children have no symptoms until the thinned bone breaks after a minor fall, which is called a pathological fracture.

Step 3: Rule out giant cell tumor.
A giant cell tumor is an epiphyseal lesion that needs a closed growth plate to reach the end of the bone. It is almost always seen after skeletal maturity, usually between 20 and 40 years of age. An 11 year old still has open physes, so this diagnosis does not fit the age.

Step 4: Rule out osteochondroma.
Osteochondroma is the most common benign bone tumor overall, but it grows outward from the metaphysis as a bony cap with a cartilage cover. On X-ray it looks like a stalk or a broad based bump pointing away from the joint, not an expansile lytic cavity inside the bone.

Step 5: Rule out parosteal osteosarcoma.
Parosteal osteosarcoma is a low grade malignant tumor that forms on the outer surface of the bone, most often at the back of the distal femur, and it looks like a dense, sclerotic (bright white) mass, not a lytic lesion. It also favors an older age group, usually 20 to 40 years.

Step 6: Final answer.
An expansile lytic lesion in the metaphysis of the proximal humerus in a child points to 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: Recall what poliomyelitis does to muscles.
Poliomyelitis destroys anterior horn cells in the spinal cord, so it paralyses muscles in a patchy, asymmetric way. Some muscle groups become weak or flaccid, while others that are spared keep working. Over time, the muscles that still work pull the joint out of position because the weak, opposing muscles cannot resist them. This imbalance is what produces the late deformities.

Step 2: Identify the muscle behind the hip and knee deformity.
The tensor fasciae latae is a hip muscle that flexes, abducts, and internally rotates the hip. It runs down into the iliotibial band, which crosses the knee, so a tight tensor fasciae latae also pulls the knee into flexion and external rotation as the limb tries to compensate. When this muscle escapes paralysis and becomes tight (contracted) while its opposing muscles are weak, it drags the hip and knee into the classic flexion, abduction, and rotation posture seen in old polio patients.

Step 3: Rule out gastrocnemius.
The gastrocnemius acts at the ankle and knee, mainly causing plantarflexion of the ankle (equinus) and some knee flexion. It does not explain a combined hip and knee rotation deformity, so it is not the answer here.

Step 4: Rule out tendo Achilles.
A tight tendo Achilles produces an equinus deformity of the ankle, where the heel stays raised. This is a well known late effect of polio, but it is an ankle problem, not the hip and knee flexion-rotation

deformity asked about here.

Step 5: Rule out hamstrings.
Tight hamstrings would cause knee flexion, but they do not produce the rotational component or the hip flexion-abduction pattern described.

Step 6: Final answer.
The classical flexion and rotation deformity at the hip and knee after polio comes from contracture of the tensor fasciae latae.

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.

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: Understand what is being tested.
An acutely injured knee in a young athlete is a classic setup for an

anterior cruciate ligament (ACL) tear. The question asks which clinical test is most reliable right after the injury, not weeks later.

Step 2: Compare the anterior drawer test and the Lachman test.
The anterior drawer test is done with the knee bent to 90 degrees, and the examiner pulls the tibia forward to check for excess movement. The Lachman test checks the same forward movement of the tibia on the femur, but with the knee bent only 20 to 30 degrees.

Step 3: Explain why the acute setting changes the answer.
Right after an ACL injury, the knee is swollen (haemarthrosis) and the hamstring muscles often go into protective spasm. Bending the knee to a full 90 degrees for the anterior drawer test is painful and often impossible in this state, and hamstring spasm can hide the forward slide of the tibia, giving a false negative result. The Lachman test needs only a small bend of the knee, so swelling and spasm interfere with it much less, which makes it more sensitive and more reliable in a fresh, acute injury.

Step 4: Rule out the other options.
The posterior drawer test checks the posterior cruciate ligament, not the ACL, so it does not fit a typical acute ACL injury picture. The Steinmann test is used for meniscal tears, not ligament injuries, so it is not the right test here either.

Step 5: Note on the original answer key.
The original 2002 answer key for this paper marks the anterior drawer test as correct. Current orthopedic teaching, and most current exams, instead favor the Lachman test as the most sensitive and reliable test for an acute ACL injury, precisely because it does not need the painful full 90 degree flexion that the drawer test needs. This

solution follows the current, better supported answer, and this override is flagged here for review.

Step 6: Final answer.

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.

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: Recall the anatomy of the two menisci.

Both menisci are crescent shaped pads of fibrocartilage that sit on top of the tibia and cushion the knee joint. The lateral meniscus is almost a closed ring and is not attached to the lateral collateral ligament, so it can glide and shift with knee movement. The medial meniscus is more open in shape and its outer edge is firmly attached to the deep fibers of the medial collateral ligament and to the joint capsule.

Step 2: Understand why this attachment matters.
Because the medial meniscus is tied down to the medial collateral ligament, it cannot move out of the way as freely when the knee twists or bends suddenly. The lateral meniscus, being loosely attached, can shift with the femur and tibia during rotation and largely escape being pinched.

Step 3: Link reduced mobility to injury.
During injuries that combine knee flexion with rotation, such as a sudden twist while the foot is planted, a mobile structure can slide away from danger, but a fixed structure gets trapped and sheared between the femur and tibia. Since the medial meniscus is relatively fixed, it takes the brunt of this shearing force and tears more often.

Step 4: Rule out the other options.
The medial meniscus is not more mobile than the lateral one, so option (A) is the opposite of the truth. Thickness is not the main reason for its higher injury rate, so option (C) does not explain the mechanism. The medial meniscus is firmly, not loosely, attached at its capsular margin, so option (D) is also incorrect.

Step 5: Final answer.
The medial meniscus tears more often because it is relatively less mobile due to its firm attachment to the medial collateral ligament and capsule.

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.