INI CET November 2026 Question Paper with Solutions

Question Paper with Solutions

Conducted by All India Institute of Medical Sciences (AIIMS), New Delhi

Time Allowed: 180 Minutes **Maximum Marks: 200**

General Instructions

(i) This is the **memory-based** question paper of **INI CET November 2026** (for the **January 2027** session), conducted by **AIIMS New Delhi** on **1 November 2026** in Computer Based Test (CBT) mode.

(ii) The full paper contains **200 objective multiple choice questions**; this PDF lists **16 questions** recalled by candidates.

(iii) The duration of the exam is **180 minutes** and the maximum score is **200 marks**.

(iv) Each question has **four options**. Most questions have one best answer; the paper also has multiple-correct, match-the-following and image-based questions.

(v) **Marking scheme: +1 mark** for a correct answer, $-\frac{1}{3}$ **mark** for an incorrect answer, and **0 marks** for an unattempted question.

(vi) For multiple-correct questions, select all the correct options.

1. A 4-year-old with nephrotic syndrome completed the steroid course one week ago. On follow-up the urine dipstick shows trace protein; the child is asymptomatic with no edema. What is the clinical status?

(A) Relapse

(B) Remission

(C) Steroid-resistant nephrotic syndrome

(D) Steroid-dependent nephrotic syndrome

Correct Answer: (B) Remission

Solution:

Step 1: Understanding the question:

The child has nephrotic syndrome and has just finished a steroid course. Now the urine dipstick shows only trace protein and there is no edema. We have to name the disease status.

In childhood nephrotic syndrome, status is always defined by the urine protein dipstick on early morning samples plus the clinical picture. The labels are remission, relapse, steroid dependent and steroid resistant.

Step 2: Recall the standard definitions:

Remission: urine albumin nil or trace (by dipstick) for 3 consecutive early morning samples.

Relapse: urine albumin 3+ or 4+ (or more than 40 mg/m2/hour) for 3 consecutive early morning samples, with or without edema.

Steroid dependent: two consecutive relapses while on alternate day steroids, or within 14 days of stopping steroids.

Steroid resistant: failure to reach remission despite 4 weeks of daily prednisolone at the standard dose (IAP guideline).

Step 3: Apply the definitions to this child:

The dipstick shows trace protein. That is the nil or trace range, which is the remission range.

The child has no edema and no symptoms, so the clinical picture also fits remission.

The child has already finished the steroid course and is now being followed up. So the disease responded to treatment and is not steroid resistant.

Step 4: Check option (A) Relapse:

A relapse needs heavy proteinuria, 3+ or 4+ on dipstick for 3 days in a row. Trace protein is far below this. So (A) is wrong.

Step 5: Check option (C) Steroid-resistant nephrotic syndrome:

Steroid resistance means proteinuria persists even after 4 weeks of daily steroids. This child has trace protein after finishing the course, so the urine cleared. So (C) is wrong.

Step 6: Check option (D) Steroid-dependent nephrotic syndrome:

Steroid dependence means the child relapses twice in a row while on alternate day steroids or within 14 days of stopping them. This child has not relapsed at all. The urine is nearly clear one week after stopping. So (D) is wrong.

Final Answer:

The child has nil or trace proteinuria, no edema, and a recent steroid course. This is remission, which is option (B). Relapse needs 3+ or 4+ protein, steroid dependence needs repeated relapses, and steroid resistance needs persistent proteinuria after 4 weeks of steroids.

Quick Tip to Solve: Remission means nil or trace protein on dipstick, while relapse needs 3+ or 4+ protein for 3 days.

2. Manic patient on lithium (therapeutic level) is started on a COX inhibitor for osteoarthritis and develops lithium toxicity symptoms. What is the mechanism?

(A) Action on glutathione of RBC

(B) Inhibition of renal prostaglandin synthesis decreasing GFR and lithium clearance

(C) Inhibition of hepatic microsomal enzymes

(D) Increased renal tubular secretion

Correct Answer: (B) Inhibition of renal prostaglandin synthesis decreasing GFR and lithium clearance

Solution:

Step 1: Understanding the question:

A manic patient is stable on lithium with a therapeutic blood level. A COX inhibitor (an NSAID) is added for osteoarthritis. Soon lithium toxicity symptoms appear. We need the mechanism.

Lithium has a narrow therapeutic range. Any drug that raises the blood level even a little can push the patient into toxicity.

Step 2: Recall how the body handles lithium:

Lithium is not metabolised. It is not bound to plasma proteins. Almost all of it leaves the body through the kidney.

It is filtered at the glomerulus. About 70-80% of the filtered lithium is reabsorbed in the proximal tubule, along with sodium and water. So anything that lowers filtration or increases sodium retention in the proximal tubule raises the lithium level.

Step 3: Link COX inhibitors to the kidney:

Prostaglandins (PGE2 and PGI2) made in the kidney dilate the afferent arteriole. They keep renal blood flow and GFR up, especially when the kidney is under stress.

A COX inhibitor blocks prostaglandin synthesis. The afferent arteriole narrows, renal blood flow and GFR fall, and the proximal tubule reabsorbs more sodium and lithium.

The net result is lower lithium clearance, a higher blood level, and toxicity. Tremor, vomiting, diarrhoea, ataxia and confusion are the usual symptoms.

Step 4: Check option (A) Action on glutathione of RBC:

Lithium does not act through red cell glutathione. Glutathione related hemolysis is seen with drugs like primaquine in G6PD deficiency. This has no link with lithium handling. So (A) is wrong.

Step 5: Check option (B) Inhibition of renal prostaglandin synthesis decreasing GFR and lithium clearance:

This matches the mechanism described above. NSAIDs reduce renal prostaglandins, GFR falls, and lithium clearance falls. So (B) is TRUE.

Step 6: Check option (C) Inhibition of hepatic microsomal enzymes:

Lithium is not metabolised by the liver, so liver enzyme inhibition cannot change its level. Enzyme inhibition matters for drugs like warfarin or theophylline. So (C) is wrong.

Step 7: Check option (D) Increased renal tubular secretion:

Increased secretion would remove more lithium and lower its level, which would reduce toxicity. The patient has toxicity, so the effect must be the opposite. So (D) is wrong.

Final Answer:

NSAIDs lower renal prostaglandins, which lowers GFR and lithium clearance, so lithium builds up. This is option (B). Thiazides, ACE inhibitors and low sodium intake raise lithium levels in a similar way.

Quick Tip to Solve: Lithium is cleared only by the kidney, and NSAIDs lower renal prostaglandins and GFR.

3. Patient with asthma and mild depression presenting with hemicranial headache attacks (about once a month) is started on migraine prophylaxis. Which agent is preferred?

(A) Propranolol

(B) Carbamazepine

(C) Topiramate

(D) Duloxetine

Correct Answer: (C) Topiramate

Solution:

Step 1: Understanding the question:

A patient with asthma and mild depression has hemicranial (one sided) headache attacks about once a month. This sounds like migraine. A prophylactic drug has been planned. We have to pick the best agent for this particular patient.

Two patient factors guide the choice: asthma and depression.

Step 2: Recall the drugs used for migraine prophylaxis:

Prophylaxis is usually advised when attacks are frequent or disabling, or when acute drugs fail. The question tells us a prophylactic drug is being started, so we choose by the patient profile.

First line prophylactic drugs are propranolol (and other beta blockers), amitriptyline, topiramate, valproate, and flunarizine. Venlafaxine and candesartan are also used.

Step 3: Use the asthma:

Propranolol is a non-selective beta blocker. It blocks beta-2 receptors in bronchial smooth muscle and can cause severe bronchospasm. It is contraindicated in asthma.

Propranolol can also worsen depression and fatigue in some patients. So it is a poor choice for this patient.

Step 4: Check option (A) Propranolol:

It is an effective migraine prophylactic, but asthma is a contraindication. So (A) is wrong.

Step 5: Check option (B) Carbamazepine:

Carbamazepine is used in trigeminal neuralgia, epilepsy and bipolar disorder. It is not an established drug for migraine prophylaxis. Valproate is the antiepileptic that is used for it. So (B) is wrong.

Step 6: Check option (C) Topiramate:

Topiramate is an antiepileptic with good evidence for migraine prevention. It does not affect the airways. It has also been used in bipolar disorder, alcohol dependence and obesity. It suits this patient best among the options. So (C) is the answer.

Step 7: Check option (D) Duloxetine:

Duloxetine is an SNRI used in depression, diabetic neuropathy and fibromyalgia. It has no established role in migraine prophylaxis. Venlafaxine, not duloxetine, has some evidence. So (D) is wrong.

Final Answer:

The beta blocker propranolol is unsafe in asthma, carbamazepine and duloxetine have no standard role in migraine prevention, and topiramate is an effective prophylactic drug. The answer is option (C).

Quick Tip to Solve: Beta blockers like propranolol are contraindicated in asthma, so pick the antiepileptic that prevents migraine.

4. Which of the following is a novel antidepressant?

(A) Fluoxetine (SSRI)

(B) Amitriptyline (TCA)

(C) Venlafaxine (SNRI)

(D) Vilazodone

Correct Answer: (D) Vilazodone

Solution:

Step 1: Understanding the question:

We are asked to pick the novel antidepressant from a list. Here, "novel" means a drug that does not belong to the older, classical groups, and usually has a mixed or new mechanism of action.

Step 2: Recall the main antidepressant groups:

Tricyclic antidepressants (TCAs): amitriptyline, imipramine, nortriptyline. They block reuptake of serotonin and noradrenaline.
SSRIs: fluoxetine, sertraline, escitalopram, paroxetine. They block only serotonin reuptake.
SNRIs: venlafaxine, duloxetine, desvenlafaxine. They block serotonin and noradrenaline reuptake.
Atypical and novel agents: bupropion, mirtazapine, trazodone, agomelatine, vortioxetine, and vilazodone.

Step 3: Understand vilazodone:

Vilazodone blocks the serotonin transporter, as an SSRI does. It also acts as a partial agonist at the 5-HT1A receptor. This dual action is meant to give a faster onset and fewer sexual and other side effects. It is taken with food to improve absorption.

Step 4: Check option (A) Fluoxetine (SSRI):

Fluoxetine is the classical SSRI. It has been used since the late 1980s. It is not considered novel. So (A) is wrong.

Step 5: Check option (B) Amitriptyline (TCA):

Amitriptyline is a tricyclic antidepressant and one of the oldest. So (B) is wrong.

Step 6: Check option (C) Venlafaxine (SNRI):

Venlafaxine is a standard SNRI. It belongs to an established group, so it is not called novel. So (C) is wrong.

Step 7: Check option (D) Vilazodone:

Vilazodone combines SSRI action with 5-HT1A partial agonism. It does not fit into the TCA, SSRI or SNRI group. It is a novel antidepressant. So (D) is TRUE.

Final Answer:

Fluoxetine, amitriptyline and venlafaxine belong to the well known SSRI, TCA and SNRI groups. Vilazodone, with its SSRI plus 5-HT1A partial agonist action, is the novel antidepressant. The answer is option (D).

Quick Tip to Solve: Pick the drug that acts as an SSRI plus a 5-HT1A partial agonist rather than a classical class member.

5. A 45-year-old with chronic diarrhoea and abdominal discomfort; stool examination reveals larvae of Strongyloides stercoralis. Which is the preferred drug for treatment?

(A) Nitazoxanide

(B) Metronidazole

(C) Paromomycin

(D) Ivermectin

Correct Answer: (D) Ivermectin

Solution:

Step 1: Understand the clinical picture:
The patient has chronic diarrhoea and abdominal discomfort, and the stool shows larvae of Strongyloides stercoralis. This is strongyloidiasis, an intestinal infection caused by a threadworm (a small roundworm).
The infective filariform larvae enter through the skin of the feet. They travel through the lungs to the small intestine, where the adult female lives in the mucosa.
The eggs hatch inside the gut, so rhabditiform larvae appear in the stool. Some of them turn into infective larvae within the same person (autoinfection), so the infection can last for years.

Step 2: Recall why treatment is important:
In people on steroids or with weak immunity, the larvae multiply fast. This causes hyperinfection and disseminated disease with a high death rate.
So every proven case needs full treatment with a drug that kills the larvae and the adult worms.

Step 3: Recall the drug of choice:
The preferred drug for strongyloidiasis is ivermectin. The usual dose is 200 micrograms per kg once a day, given for 1 to 2 days.
Ivermectin binds to glutamate-gated chloride channels in the nerve and muscle cells of the worm. Chloride rushes in, the cell membrane becomes hyperpolarised, and the worm is paralysed and expelled.
Albendazole is only an alternative, because its cure rate is lower. So option (D) is the best choice.

Step 4: Check option (A) Nitazoxanide:
Nitazoxanide blocks the pyruvate-ferredoxin oxidoreductase enzyme of anaerobic organisms. It is used mainly for Giardia and Cryptosporidium diarrhoea.
It is not the preferred drug for Strongyloides. So (A) is wrong.

Step 5: Check option (B) Metronidazole:
Metronidazole is a nitroimidazole. It works against anaerobic bacteria and protozoa such as Entamoeba histolytica, Giardia and Trichomonas.
It has no useful action on nematodes (roundworms). So (B) is wrong.

Step 6: Check option (C) Paromomycin:
Paromomycin is an aminoglycoside that is hardly absorbed from the gut. It is used as a luminal amoebicide and for Cryptosporidium in some cases.
It does not kill Strongyloides larvae. So (C) is wrong.

Final Answer:
The preferred drug for intestinal strongyloidiasis is ivermectin. This is option (D).

Quick Tip to Solve: Strongyloides is a roundworm, and the drug of choice is the macrocyclic lactone anthelmintic.

6. A 28-year-old male develops muscarinic toxicity following consumption of a mushroom. Which clinical manifestation is expected?

(A) Tachycardia

(B) Mydriasis

(C) Bradycardia

(D) Urinary retention

Correct Answer: (C) Bradycardia

Solution:

Step 1: Understand the poisoning:

Some mushrooms, such as Inocybe and Clitocybe species, contain muscarine. Muscarine is a direct agonist at muscarinic acetylcholine receptors.

It is not broken down by acetylcholinesterase, so its action lasts for hours. The patient shows features of excess parasympathetic stimulation (a cholinergic picture).

Step 2: Recall the effects of muscarinic stimulation:

Every organ with muscarinic receptors responds. The eye shows miosis (small pupil) and spasm of accommodation.

Glands give excess salivation, sweating, lacrimation and bronchial secretions. The lung shows bronchoconstriction.

The gut shows cramps, vomiting and diarrhoea. The bladder wall contracts and the sphincter relaxes, so there is urgency and incontinence.

The heart shows a fall in heart rate, because M2 receptors on the SA node slow the rate. This is bradycardia.

Step 3: Pick the expected finding:

Among the options, only bradycardia fits this picture. So option (C) is correct.

The usual antidote is atropine, which blocks the muscarinic receptors.

Step 4: Check option (A) Tachycardia:

A fast heart rate is a sign of vagal blockade, which happens with atropine or other anticholinergic drugs. Muscarine does the opposite. So (A) is wrong.

Step 5: Check option (B) Mydriasis:

Mydriasis (wide pupil) is caused by blocking the muscarinic receptor of the sphincter pupillae. Muscarinic toxicity gives miosis, not mydriasis. So (B) is wrong.

Step 6: Check option (D) Urinary retention:

Retention of urine is a feature of anticholinergic toxicity, because the detrusor muscle relaxes. In muscarinic toxicity the detrusor contracts, so the patient passes urine often or loses control. So (D) is wrong.

Final Answer:

Muscarinic toxicity from a mushroom produces bradycardia. This is option (C).

Quick Tip to Solve: Muscarinic excess means parasympathetic overactivity, so think of a slow heart rate and small pupils.

7. A 65-year-old male chronic smoker with fatigue, nausea and confusion; imaging shows small cell carcinoma with SIADH. Which drug can be used for management?

(A) Desmopressin

(B) Tolvaptan

(C) Thiazides

(D) Furosemide

Correct Answer: (B) Tolvaptan

Solution:

Step 1: Understand the problem:

Small cell carcinoma of the lung can make antidiuretic hormone (ADH) on its own. This causes the syndrome of inappropriate ADH secretion (SIADH).

ADH acts on V2 receptors in the collecting duct and inserts aquaporin-2 channels. The kidney then keeps too much water.

The result is euvolaemic hyponatraemia with a low serum osmolality and concentrated urine. Fatigue, nausea and confusion are the usual symptoms.

Step 2: Recall how SIADH is treated:

The first step is to treat the cause and restrict fluids. Severe or symptomatic hyponatraemia needs hypertonic saline.

For long-term control, the options are demeclocycline, urea and the vaptans.

Vaptans are V2 receptor antagonists. Tolvaptan is the oral drug and conivaptan is given intravenously. They block ADH at the collecting duct, so free water is passed out (aquaresis) and serum sodium rises.

Step 3: Pick the answer:

Among the options, tolvaptan is the targeted drug for this condition. So option (B) is correct.

The rise in sodium must be slow, no more than 8 to 10 mEq/L in 24 hours, to avoid osmotic demyelination.

Step 4: Check option (A) Desmopressin:

Desmopressin is a V2 agonist. It would copy the excess ADH and make the hyponatraemia worse. It is used for central diabetes insipidus, nocturnal enuresis and bleeding disorders. So (A) is wrong.

Step 5: Check option (C) Thiazides:

Thiazides are a known cause of hyponatraemia. They are used in nephrogenic diabetes insipidus, not in SIADH, and they would lower sodium further. So (C) is wrong.

Step 6: Check option (D) Furosemide:

A loop diuretic can be added with oral salt in some cases to increase the loss of free water. But it also causes sodium loss, and it is not a specific treatment for SIADH.

Of the choices given, tolvaptan is the targeted drug. So (D) is not the answer.

Final Answer:

The drug that can be used to manage SIADH here is tolvaptan, an oral V2 antagonist. This is option (B).

Quick Tip to Solve: SIADH has too much ADH action, so choose the drug that blocks the V2 receptor.

8. Which parameters are routinely monitored during magnesium sulfate maintenance therapy to detect magnesium toxicity?

More than one option may be correct.

(A) Respiratory rate (should be more than 14 per minute)

(B) Deep tendon reflexes (knee jerk present)

(C) Urine output (more than 100 mL in 4 hours)

(D) Serum sodium level every hour

Correct Answer: (A) Respiratory rate (should be more than 14 per minute); (B) Deep tendon reflexes (knee jerk present); (C) Urine output (more than 100 mL in 4 hours)

Solution:

Step 1: Understand why monitoring is needed:
Magnesium sulfate is used to prevent and treat eclamptic seizures. It has a narrow safety margin, so the mother must be watched for toxicity.
Magnesium is cleared by the kidneys. If urine output falls, the blood level rises quickly.
The usual levels are as follows. The therapeutic range is 4 to 7 mEq/L. The knee jerk is lost at about 8 to 10 mEq/L. Respiratory depression appears at about 12 to 15 mEq/L. Cardiac arrest can occur above 25 mEq/L.

Step 2: Recall the three bedside checks before every dose:
Before each maintenance dose, the nurse or doctor checks three things: the respiratory rate, the deep tendon reflexes and the urine output.
The next dose is given only if all three are in the safe range. If any one is not, the dose is held.

Step 3: Check statement (1) Respiratory rate:
Magnesium blocks neuromuscular transmission, so the respiratory muscles weaken. A fall in respiratory rate is a sign of toxicity.
The rate must stay at a safe level (at least 12 per minute in the standard teaching, and many wards use 14 or more as a working limit). The option states more than 14 per minute, which is within the safe range. So (1) is correct.

Step 4: Check statement (2) Deep tendon reflexes:
Loss of the patellar (knee) reflex is the earliest sign of magnesium excess. It appears before respiratory depression.
A present knee jerk shows that the level is still below the toxic range. So (2) is correct.

Step 5: Check statement (3) Urine output:
Urine output of at least 30 mL per hour, or about 100 mL in 4 hours, shows that the kidneys are clearing magnesium.
If the output falls below this, magnesium builds up and the dose must be cut or stopped. So (3) is correct.

Step 6: Check statement (4) Serum sodium level every hour:
Serum sodium has no link to magnesium toxicity, and it is not checked every hour. If a level is needed, it is the serum magnesium level, and that is checked only in selected cases such as renal impairment. So (4) is wrong.

Step 7: Remember the antidote:
If toxicity occurs, stop the infusion and give 10 mL of 10 percent calcium gluconate slowly by the intravenous route (over 10 minutes).

Final Answer:
Respiratory rate, deep tendon reflexes and urine output are the routine checks. The correct options

are 1, 2 and 3.

Quick Tip to Solve: Think of the three checks before each dose: breathing, knee jerk and urine.

9. Which of the following statements is FALSE regarding the pediatric larynx in comparison to the adult larynx?

(A) The paediatric larynx is funnel shaped while the adult larynx is cylindrical

(B) The narrowest part of the paediatric airway is the supraglottis

(C) The paediatric larynx lies high in the neck, so the epiglottis can touch the soft palate

(D) The anterior commissure lies at the inferior border of the thyroid cartilage, giving the vocal cords a slanted look

Correct Answer: (B) The narrowest part of the paediatric airway is the supraglottis

Solution:

Step 1: Understand what is being asked:
The question asks for the FALSE statement about the paediatric larynx compared with the adult larynx. Three of the four statements are true. So the job is to find the one that gets an anatomical fact wrong.

Step 2: Recall the key differences between infant and adult larynx:
The infant larynx is funnel shaped. It is narrow below and wide above. The adult larynx is cylindrical.
The infant larynx lies high in the neck, at about C3-C4. The adult larynx sits lower, at about C6.
The epiglottis in an infant is long, floppy and omega shaped. Its tip can touch or overlap the soft palate.
The narrowest part of the infant airway is the subglottis, at the level of the cricoid cartilage. In the adult it is the glottis (the space between the vocal cords).
The cricoid is the only complete ring of cartilage in the airway, so the subglottis cannot expand when it swells.

Step 3: Check option (1): funnel shaped versus cylindrical:
This matches the standard description. The infant larynx narrows from above downwards and is funnel shaped. The adult larynx has a more uniform, cylindrical outline. So option (1) is TRUE.

Step 4: Check option (2): narrowest part is the supraglottis:
This is wrong. The narrowest part of the infant airway is the subglottis at the cricoid ring, not the supraglottis.
The supraglottis (epiglottis, aryepiglottic folds, false cords) is actually wide in an infant.
This is why a small amount of subglottic oedema in a child causes severe stridor. Even 1 mm of swelling can cut the airway area by a large fraction.
So option (2) is FALSE, and this is the statement we want.

Step 5: Check option (3): high position of the larynx:
The larynx lies high in an infant, so the epiglottis can touch the soft palate. This lets the baby breathe through the nose while swallowing milk.
The exact vertebral level quoted by textbooks varies a little, but the core idea (high larynx, epiglottis reaching the soft palate) is accepted as true. So option (3) is TRUE in the sense the question intends.

Step 6: Check option (4): position of the anterior commissure:
In an infant the anterior commissure lies lower, near the inferior border of the thyroid cartilage. The posterior end of the vocal cords lies higher.
So the vocal cords run in a slanted line, from low in front to high behind. In an adult the cords are close to horizontal. So option (4) is TRUE.

Final Answer:
The false statement is that the narrowest part of the paediatric airway is the supraglottis. The narrowest part is the subglottis at the cricoid level. This is option (2). \[\boxed{\text{Option (2)}} \]

Quick Tip to Solve: The narrowest part of the infant airway is at the cricoid ring below the cords.

10. Young man after blunt chest trauma, severely breathless, hypotensive, absent breath sounds over right hemithorax with hyperresonant percussion (tension pneumothorax). Most appropriate immediate intervention when needle decompression is the fastest available method?

(A) Intravenous fluid bolus and observe

(B) CT scan of the chest

(C) Intercostal drain insertion after radiological confirmation

(D) Immediate needle decompression of the right pleural space

Correct Answer: (D) Immediate needle decompression of the right pleural space

Solution:

Step 1: Identify the condition:
A young man after blunt chest trauma is severely breathless and hypotensive. Breath sounds are absent over the right hemithorax and percussion is hyperresonant.
This is a tension pneumothorax. Air enters the pleural space through a one-way valve and cannot leave. Pressure builds up on that side.

Step 2: Understand why it is an emergency:
The high pressure collapses the lung and pushes the mediastinum to the opposite side. The great veins kink, so venous return to the heart falls.
Cardiac output drops, and the patient becomes hypotensive and may arrest. The problem is mechanical, so only release of the trapped air will fix it.

Step 3: Know the rule for management:
Tension pneumothorax is a clinical diagnosis (ATLS teaching). Treat it at once, before any X-ray or CT.
The first step is needle decompression. Insert a wide-bore cannula in the 2nd intercostal space in the midclavicular line (or the 4th-5th space in the anterior axillary line). Follow it with an intercostal chest drain.

Step 4: Check option (1): fluid bolus and observe:
Fluids may help blood pressure for a short time, but they do not remove the cause. Observing a patient with tension is dangerous. So option (1) is wrong.

Step 5: Check option (2): CT scan of the chest:
Taking an unstable patient to the scanner wastes time and can lead to death in the CT room. Imaging is not needed to make this diagnosis. So option (2) is wrong.

Step 6: Check option (3): drain after radiological confirmation:
A chest drain is the definitive treatment, but waiting for an X-ray first delays relief. The question says needle decompression is the fastest method available. So option (3) is wrong.

Step 7: Check option (4): immediate needle decompression:
This releases the trapped air within seconds, restores venous return and improves blood pressure and breathing. A chest drain can be placed after that. So option (4) is right.

Final Answer:
The best immediate step is needle decompression of the right pleural space. This is option (4). \[\boxed{\text{Option (4)}} \]

Quick Tip to Solve: Tension pneumothorax is a clinical diagnosis: decompress first, image later.

11. 29-year-old man with Crohn's disease with recurrent episodes of colicky abdominal pain, distension and vomiting. After initial decompression and resuscitation, imaging shows a localized fixed stricture with proximally dilated bowel loops. Obstructive symptoms persist despite medical management. Colon and rectum uninvolved. Best management?

(A) Right hemicolectomy

(B) Total proctocolectomy with end ileostomy

(C) Limited resection of the involved segment with primary anastomosis

(D) Restorative proctocolectomy with ileal pouch

Correct Answer: (C) Limited resection of the involved segment with primary anastomosis

Solution:

Step 1: Understand the problem:
A 29-year-old man has Crohn's disease with repeated colicky pain, distension and vomiting. Imaging shows one fixed stricture with dilated bowel above it.
Symptoms continue after medical treatment. The colon and rectum are normal. We need the best operation.

Step 2: Recall surgical principles in Crohn's disease:
Surgery in Crohn's disease does not cure it. The disease can return, so we save as much bowel as we can and avoid short bowel syndrome.
Indications for surgery include obstruction not settling with medical care, fistula, abscess, perforation, and failure of drugs.
For a localized stricture, the usual operation is limited resection of the diseased segment with primary anastomosis. Stricturoplasty is another bowel-sparing choice, mainly for short strictures or when much bowel was lost before.
There is no need to remove wide margins of normal bowel. Margins of about 2 cm are enough.

Step 3: Check option (1): right hemicolectomy:
A right hemicolectomy removes the caecum and ascending colon. It is used for ileocaecal disease or cancer of the right colon. Here the problem is a localized stricture and the colon is uninvolved, so it removes more than needed. Option (1) is wrong.

Step 4: Check option (2): total proctocolectomy with end ileostomy:
This takes out the whole colon and rectum. It is meant for severe, extensive colonic or perianal Crohn's disease. The colon and rectum are healthy here, so this is too radical. Option (2) is wrong.

Step 5: Check option (3): limited resection with primary anastomosis:
This removes only the stricture and joins healthy ends. It relieves the obstruction and keeps bowel length. This is the standard operation, so option (3) is right.

Step 6: Check option (4): restorative proctocolectomy with ileal pouch:
An ileal pouch (IPAA) is the operation of choice for ulcerative colitis. In Crohn's disease the disease may return in the pouch and cause fistulas and pouch failure. Also the rectum is not diseased here. Option (4) is wrong.

Final Answer:
A localized, medically refractory Crohn's stricture is treated by limited resection with primary anastomosis. This is option (3). \[\boxed{\text{Option (3)}} \]

Quick Tip to Solve: Crohn's disease recurs, so remove only the diseased stricture and save bowel length.

12. 70-year-old man with atrial fibrillation and severe abdominal pain with disproportionate abdominal signs (pain out of proportion), hemodynamically stable, acute mesenteric ischemia due to embolic arterial occlusion. Next step?

(A) Doppler

(B) Urgent CT angiography of abdomen and pelvis

(C) Plain abdominal radiograph

(D) Exploratory laparotomy

Correct Answer: (B) Urgent CT angiography of abdomen and pelvis

Solution:

Step 1: Recognise the condition:

A 70-year-old man with atrial fibrillation has severe abdominal pain out of proportion to the findings on examination. He is hemodynamically stable.

This is acute mesenteric ischemia from an embolus. Atrial fibrillation throws clots, and they usually lodge in the superior mesenteric artery (SMA), just beyond the origin of the middle colic artery.

Step 2: Recall how to confirm and plan treatment:

Early diagnosis matters, because bowel can die within hours.

In a stable patient without peritonitis, the best next step is urgent CT angiography of the abdomen. It shows the exact site of occlusion, the extent of collateral flow, and signs of bowel injury.

Immediate laparotomy is kept for patients with peritonitis, shock or other signs of dead bowel.

After the diagnosis, give heparin, then restore flow by embolectomy or endovascular therapy, and resect any non-viable bowel.

Step 3: Check option (1): Doppler:

Duplex Doppler can show proximal SMA blockage but depends on the operator. Bowel gas often blocks the view and the study is not reliable for the whole picture. It is not the best next step, so option (1) is wrong.

Step 4: Check option (2): urgent CT angiography:

CT angiography is fast, widely available and highly accurate. It is the investigation of choice. So option (2) is right.

Step 5: Check option (3): plain abdominal radiograph:

A plain film is often normal early on. Later signs such as bowel wall gas or free air appear late. It cannot confirm or exclude mesenteric ischemia. Option (3) is wrong.

Step 6: Check option (4): exploratory laparotomy:

Laparotomy is needed if there is peritonitis or instability. This man is stable, so imaging comes first. Option (4) is wrong in this setting.

Final Answer:

In a stable patient with suspected embolic mesenteric ischemia, the next step is urgent CT angiography. This is option (2). \[\boxed{\text{Option (2)}} \]

Quick Tip to Solve: Stable patient with AF and pain out of proportion: image the mesenteric vessels at once.

13. 45-year-old man 48 hours after onset of severe abdominal pain with generalized guarding and rigidity, pulse 132, BP 78/46, reduced urine output; imaging shows pneumoperitoneum. Management?

(A) Transfer directly for laparotomy without resuscitation

(B) Immediate resuscitation and IV broad-spectrum antibiotics while arranging urgent laparotomy for source control

(C) Laparoscopic drainage alone without treating the perforation

(D) Antibiotics alone and observe

Correct Answer: (B) Immediate resuscitation and IV broad-spectrum antibiotics while arranging urgent laparotomy for source control

Solution:

Step 1: Understanding the Concept:
This patient has generalized peritonitis from a perforated hollow viscus. The signs are guarding, rigidity and pneumoperitoneum (free air under the diaphragm) on imaging.
He has also been ill for 48 hours. A pulse of 132, BP of 78/46 and low urine output mean he is in septic shock with poor organ perfusion.

Step 2: Key Approach:
In peritonitis with shock, deaths happen mainly because of shock, not because of the delay in surgery by a few hours.
So the order of care is fixed: resuscitate first, give antibiotics early, then operate to control the source of contamination.
Source control means closing, resecting or diverting the perforation and washing out the peritoneal cavity. Antibiotics and fluids can never replace it.

Step 3: Resuscitation Steps:
Give a fluid bolus of crystalloid through large-bore IV lines. Add vasopressors if BP stays low after fluids.
Pass a urinary catheter to track urine output. Pass a nasogastric tube to decompress the stomach.
Start IV broad-spectrum antibiotics at once, covering Gram-negative bacilli, enterococci and anaerobes. Give oxygen and correct electrolytes.
This phase should be short and run in parallel with preparing the operating room. Do not wait for the patient to become fully normal, since the source of sepsis stays inside.

Step 4: Check option (A):
Taking a hypotensive, tachycardic patient to the operating room without resuscitation is dangerous. Anaesthetic induction drops BP further and can cause cardiac arrest.
So option (A) is wrong.

Step 5: Check option (B):
This gives resuscitation and antibiotics first and arranges urgent laparotomy for source control. This is the standard sequence for perforation peritonitis with shock.
So option (B) is correct.

Step 6: Check option (C):
Laparoscopic drainage alone leaves the hole in the bowel open. Contamination continues and sepsis gets worse.
A laparoscopic approach may be used by experts to close a perforation, but drainage without treating

the perforation is wrong. So option (C) is wrong.

Step 7: Check option (D):
Antibiotics with observation is non-operative management. It is used only for a few contained, localized perforations in a stable patient.
This man has generalized peritonitis and shock, so he needs surgery. So option (D) is wrong.

Final Answer:
The patient needs immediate resuscitation and IV broad-spectrum antibiotics while urgent laparotomy is arranged for source control. This corresponds to option (B).

Quick Tip to Solve: In peritonitis with shock, resuscitate and give antibiotics first, then operate for source control.

14. 36-year-old man with severe pain during and after defecation and occasional streaks of blood. Posterior midline anal fissure with exposed internal sphincter fibres and sentinel skin tag. Symptoms persist despite stool softeners and topical treatment; increased resting tone, normal continence, no previous sphincter injury. Most appropriate definitive surgical treatment?

(A) Uncontrolled manual anal dilatation (Lord's procedure)

(B) Advancement flap

(C) Excision of sentinel tag alone

(D) Lateral internal sphincterotomy

Correct Answer: (D) Lateral internal sphincterotomy

Solution:

Step 1: Understanding the Concept:
This is a chronic anal fissure. The signs are a posterior midline tear, exposed internal sphincter fibres and a sentinel skin tag at the lower end.
The cause is a vicious circle. A hard stool tears the anoderm, pain triggers spasm of the internal anal sphincter, the raised tone cuts blood flow to the posterior midline, and the tear fails to heal.

Step 2: Key Approach:
Acute fissures are treated medically with stool softeners, fibre, sitz baths and topical agents such as nitroglycerin or diltiazem.
A fissure is called chronic when it lasts more than 6 weeks or shows a sentinel tag and visible internal sphincter fibres. If medical care fails, surgery is done.
The surgery of choice is lateral internal sphincterotomy. It cuts the lower part of the internal sphincter, so the tone falls, the pain goes and the fissure heals.

Step 3: Apply to this patient:
He has symptoms despite stool softeners and topical treatment. He has increased resting tone, normal continence and no earlier sphincter injury.
All of these favour lateral internal sphincterotomy. Normal continence means there is a low risk of incontinence.

Step 4: Check option (A):
Lord's procedure is forceful stretching of the anal canal. It is uncontrolled, and it can tear the sphincters and cause incontinence.
It is no longer recommended. So option (A) is wrong.

Step 5: Check option (B):

An advancement flap is used for fissures with low or normal tone, for anal stenosis, or for cases where sphincterotomy would be risky. It also helps if there is a risk of incontinence.
This man has high tone and good continence, so it is not the first choice. So option (B) is wrong.

Step 6: Check option (C):
Excising the sentinel tag alone does not remove the cause, which is the sphincter spasm. The fissure stays and the pain continues.
So option (C) is wrong.

Step 7: Check option (D):
Lateral internal sphincterotomy divides the internal sphincter only, up to the level of the dentate line, and never touches the external sphincter. It heals more than 90% of chronic fissures.
So option (D) is correct.

Final Answer:
The definitive surgery for a chronic anal fissure with raised tone after failed medical care is lateral internal sphincterotomy. This corresponds to option (D).

Quick Tip to Solve: Chronic fissure with sentinel tag and high resting tone is treated surgically by lateral internal sphincterotomy.

15. Which hypothalamic nucleus is responsible for pulsatile GnRH secretion?

(A) Preoptic nucleus

(B) Supraoptic nucleus

(C) Arcuate nucleus

(D) Paraventricular nucleus

Correct Answer: (C) Arcuate nucleus

Solution:

Step 1: Understanding the Concept:
GnRH is released in pulses from the hypothalamus. The pulse rate matters: a fast pulse favours LH, and a slow pulse favours FSH.
The question asks which hypothalamic nucleus drives this pulsatile release, which is the GnRH pulse generator.

Step 2: Key Fact:
The GnRH pulse generator is made of KNDy neurons in the arcuate nucleus. KNDy stands for kisspeptin, neurokinin B and dynorphin, the three peptides these neurons make.
Neurokinin B excites the network, and dynorphin shuts it off. This gives rhythmic bursts of kisspeptin, which then stimulate GnRH neurons to release GnRH in pulses.
In women, the arcuate nucleus is also the site where oestrogen feedback acts through kisspeptin.

Step 3: Check option (A):
The preoptic area holds the cell bodies of many GnRH neurons, and it drives the pre-ovulatory LH surge through the anteroventral periventricular region.
It does not generate the pulses. So option (A) is wrong.

Step 4: Check option (B):
The supraoptic nucleus makes ADH (vasopressin) and some oxytocin. It has no role in GnRH pulses.
So option (B) is wrong.

Step 5: Check option (C):

The arcuate nucleus holds the KNDy neurons that act as the GnRH pulse generator. So option (C) is correct.

Step 6: Check option (D):
The paraventricular nucleus makes oxytocin, ADH, CRH and TRH. It is not the GnRH pulse generator. So option (D) is wrong.

Final Answer:
The arcuate nucleus, through its KNDy neurons, produces pulsatile GnRH secretion. This corresponds to option (C).

Quick Tip to Solve: KNDy (kisspeptin, neurokinin B, dynorphin) neurons of the arcuate nucleus drive GnRH pulses.

16. All of the following are risk factors for primary open angle glaucoma (POAG) except:

(A) Positive family history

(B) Myopia

(C) Hypermetropia

(D) Involvement of the MYOC gene

Correct Answer: (C) Hypermetropia

Solution:

Step 1: Understanding the Concept:
Primary open angle glaucoma (POAG) is a chronic optic neuropathy with an open anterior chamber angle. The cause is poor aqueous outflow through the trabecular meshwork, which raises intraocular pressure.
The question asks for the one factor that is NOT a risk factor for POAG.

Step 2: Known risk factors for POAG:
Raised intraocular pressure, older age and a positive family history. The risk is higher in first degree relatives.
Myopia, especially high myopia. Diabetes mellitus, hypertension, black race and thin central corneal thickness also raise the risk.
Genes such as MYOC (myocilin, on 1q, the GLC1A locus), OPTN and WDR36 are linked to POAG.

Step 3: Check option (A):
A positive family history is a well-known risk factor. So option (A) is a risk factor, and it is not the answer.

Step 4: Check option (B):
Myopia is an accepted risk factor for POAG. Myopic eyes have a weaker lamina cribrosa and a higher chance of optic nerve damage. So option (B) is a risk factor.

Step 5: Check option (C):
Hypermetropic eyes are small, with a shallow anterior chamber and a narrow angle. These eyes are prone to primary angle closure glaucoma, not POAG.
So hypermetropia is not a risk factor for POAG, and option (C) is the answer.

Step 6: Check option (D):
Mutations of the MYOC gene (myocilin) are the best known genetic cause of POAG, mainly juvenile and early adult forms. So option (D) is a risk factor.

Final Answer:

Hypermetropia predisposes to angle closure glaucoma, not to POAG. This corresponds to option (C).

Quick Tip to Solve: Hypermetropia (small eye, narrow angle) predisposes to angle closure glaucoma, not POAG.