



NEET PG · 30 AUGUST 2026

# NEET PG 2026 Recall Paper & Solutions

Paper khatam. Ab baithkar dekhiye kya poocha tha, sahi answer kya tha, aur kyun — poora paper, solved and explained, subject by subject.

**188**

QUESTIONS SOLVED

**91**

STRAIGHT FROM OUR LECTURES

**9**

EXAMINER PATTERNS

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# 91 / 180 questions

## More than 50% of this paper was asked directly from our lectures.

Har green-tagged question is document mein iska proof hai — wahi fact, wahi topic, humare lecture mein pehle se padhaya hua.

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Memory-based recall — compiled from what students remembered of the paper, not the official paper. Wording is approximate; medicine is exact.

## What this is

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NEET PG 2026 (30 August) ka recall paper — compiled by Team Clinical Guruji from the questions students remembered after the exam, solved and explained by us, subject by subject. Wording approximate ho sakti hai — **medicine exact hai**.

Paper mein 180 questions the; cross-checking ke baad humne **188 unique questions** reconstruct kiye. In mein se 7-8 phantom recalls ho sakte hain — do students ka ek hi question alag-alag yaad rakhna. Humne unhe bhi solve karke rakha hai: ek extra solved question aapki revision ka nuksan nahi karta.

## Reading the cards

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Har question card pe:

- **Answer** — humara verified answer, apni medical reasoning se.
- **Explanation** — kyun ye answer sahi hai, aur main distractor galat kyun hai.
- 💡 **Pearl** — the one line worth remembering for your next attempt.
- 🖼️ — exam mein yahan image thi (ECG/clinical photo/instrument); humne describe kiya hai kya dikhaya gaya tha.
- ★ **Asked directly from our Rank Booster lectures** — ye tag un questions par hai jinka tested fact humne Rank Booster course mein as-it-is padhaya tha. Har tag ke peeche lecture-wise proof maujood hai.

Sabse zyada kaam ki cheez aakhri section mein hai: **is paper ke examiner patterns** — kyunki agla paper naya hoga, par examiner ki aadatein nahi badaltin.

## 01 Anatomy

6 recalled questions

### Q1 ANATOMY

Which ligament is primarily responsible for maintaining the anteversion of the uterus (the ~90-degree angle the cervical axis makes with the vagina)?

- A Cardinal ligament
- B Round ligament
- C Uterosacral ligament
- D Uterosacral plus round ligament
- E Pubocervical ligament

**Answer** Round ligament

Anteversion is the forward angle (about 90 degrees) between the axis of the cervix and the axis of the vagina. Standard anatomy references (Gray's Anatomy, BD Chaurasia) credit the round ligaments with maintaining anteversion by pulling the fundus forward, while the uterosacral ligaments brace the cervix backward against that pull. The cardinal and pubocervical ligaments are chiefly mechanical supports against prolapse, not determinants of the anteverted angle.

💡 Round ligament fundus ko aage kheenchta hai = anteversion; uterosacral cervix ko peeche pakadta hai.

### Q2 ANATOMY

An anatomical dissection image of the gluteal region with the gluteus maximus labelled was shown. Which movement is facilitated by this muscle?

 Anatomical dissection of the gluteal region with the gluteus maximus labelled 'G. max'.

- A Lateral rotation
- B Lateral rotation & Extension
- C Medial rotation & Flexion

**Answer** Lateral rotation & Extension (of the hip)

Gluteus maximus is the chief extensor of the hip and a powerful lateral (external) rotator, acting mainly when rising from sitting, climbing stairs, and running. Its uppermost fibres assist abduction via the iliotibial tract and lowest fibres can adduct, but extension plus lateral rotation are the primary tested actions and match the marked exam answer. Medial rotation and flexion belong to the anterior fibres of gluteus medius/minimus and tensor fasciae latae, not gluteus maximus.

💡 G. max = uthne-chadhne wali muscle - hip extension + external rotation.

**Q3** ANATOMY

★ Asked directly from our Rank Booster lectures

After surgery in the posterior triangle of the neck, a woman is unable to comb her hair (variants recalled: unable to shrug the shoulder, unable to raise the arm above the shoulder). Which nerve is injured?

- A Spinal accessory nerve
- B Dorsal scapular nerve
- C Long thoracic nerve

**Answer** Spinal accessory nerve

The spinal accessory nerve (CN XI) runs superficially across the posterior triangle and is the classic nerve injured during surgery or lymph node biopsy there. Trapezius paralysis causes inability to shrug the shoulder and loss of scapular rotation needed to abduct the arm above 90 degrees, e.g., combing hair. Long thoracic nerve injury (serratus anterior, winged scapula) is classically associated with axillary surgery such as mastectomy, not the posterior triangle.

💡 Posterior triangle surgery + kandha nahi utha sakte = CN XI, trapezius gaya.

**Q4** ANATOMY

A child presents with multiple renal and ureteric anomalies. The kidney develops from which of the following parts? (An embryological germ-layer diagram was shown.)

 Embryological cross-section showing ectoderm, endoderm and mesoderm, with mesoderm divided into paraxial, intermediate and lateral plate portions.

- A Paraxial mesoderm
- B Intermediate mesoderm
- C Yolk sac
- D Allantois

**Answer** Intermediate mesoderm

The intermediate mesoderm forms the nephrogenic cord, which gives rise to the pronephros, mesonephros and metanephros - hence the kidneys, ureters (via the ureteric bud from the mesonephric duct) and gonads. Paraxial mesoderm forms somites (axial skeleton and skeletal muscle), and the lateral plate forms body wall and gut serosa, so anomalies of the kidney and ureter point to the intermediate mesoderm.

💡 Kidney-ureter-gonad = intermediate mesoderm; somites paraxial se.

**Q5** ANATOMY

★ Asked directly from our Rank Booster lectures

Esophageal varices represent a portosystemic anastomosis between which two veins?

**Answer** Left gastric vein (portal) and azygos vein (systemic, via esophageal tributaries)

At the lower esophagus, esophageal tributaries of the left gastric vein (portal system) anastomose with esophageal veins draining into the azygos/hemiazygos system (systemic). In portal hypertension this channel dilates to form esophageal varices, the most clinically important portosystemic anastomosis because of its bleeding risk.

💡 Varices = left gastric (portal) milta hai azygos (systemic) se - portal HTN ka sabse khatarnak junction.

**Q6**

ANATOMY

★ Asked directly from our Rank Booster lectures

Coronary angiography shows a block in the left anterior descending (LAD) artery. Which area's blood supply is compromised?

**Answer** Anterior two-thirds of the interventricular septum (plus the anterior wall of the left ventricle and apex)

The LAD runs in the anterior interventricular groove and supplies the anterior two-thirds of the interventricular septum via septal perforators, along with the anterior LV wall and the apex. The posterior one-third of the septum is supplied by the posterior descending artery, which arises from the right coronary artery in right-dominant circulation.

💡 LAD = widow-maker: anterior 2/3 septum + anterior wall + apex.

## 02 Physiology

7 recalled questions

### Q7 PHYSIOLOGY

★ Asked directly from our Rank Booster lectures

A student is performing an experiment using the equipment shown in the diagram (a spirometer). Which of the following can be measured with it?

 Line drawing of a seated person performing a spirometry test on a tabletop spirometer.

- A Total Lung capacity
- B Residual Volume
- C Functional residual capacity
- D Vital capacity

**Answer** Vital capacity

A simple spirometer measures only air that moves through the mouth — TV, IRV, ERV, and therefore vital capacity (and FVC/FEV1). Residual volume cannot be exhaled, so RV and every capacity containing it (FRC, TLC) require helium dilution, nitrogen washout, or body plethysmography. The apparent source disagreement is only stem polarity ('can' vs 'cannot' be measured) — medically all sources say the same thing, and the on-screen question asked 'can be measured' with vital capacity marked.

💡 Jo saans bahar aa hi nahi sakti (RV) woh spirometer kaise napega — RV, FRC, TLC teeno bahar.

### Q8 PHYSIOLOGY

★ Asked directly from our Rank Booster lectures

A man admitted after a road traffic (bike) accident develops right-sided motor weakness with decreased vibration/joint sensation, and loss of pain and temperature on the left side below the lesion. What is the diagnosis and the side of the cord lesion?

- A hemisection of the right side of the cord
- B hemisection of the left side of the cord

**Answer** Brown-Séquard syndrome — hemisection of the right side of the spinal cord (ipsilateral to the motor and dorsal-column loss)

In cord hemisection, the corticospinal tract and dorsal columns are interrupted ipsilaterally (motor weakness, vibration/proprioception loss on the side of the lesion), while the spinothalamic tract — which crosses within one or two segments of entry — is lost contralaterally (pain and temperature on the opposite side). Right-sided weakness with left-sided pain/temperature loss therefore localizes to a right hemisection. All sources agree on Brown-Séquard; the different recalled left/right versions follow the same rule.

💡 Taakat aur position sense usi taraf jaati hai, dard-garmi ulti taraf — lesion hamesha weakness wali side pe.



**Q9** PHYSIOLOGY

Match the sensory receptors marked 1, 2 and 3 in the skin diagram.

 Anatomical diagram of skin layers with sensory receptors labeled 1-4: Pacinian corpuscle, Ruffini endings, free nerve endings, and Merkel cells.

- A Pacinian, Free nerve ending and Ruffini endings
- B Ruffini, Merkel's and free nerve endings
- C Pacinian, Ruffini endings and Free nerve ending
- D Ruffini, Pacinian and Krause's bulb

**Answer** Pacinian corpuscle, Ruffini endings and free nerve ending (option C)

The deep, onion-like lamellated receptor in the dermis/subcutis is the Pacinian corpuscle (vibration, deep pressure); the spindle-shaped dermal receptor is the Ruffini ending (sustained pressure, skin stretch); and the unencapsulated terminals reaching the epidermis are free nerve endings (pain, temperature). Merkel cells (epidermal, fine touch) were the distractor.

 Onion dikhe toh Pacinian — vibration wala; stretch ka Ruffini, dard ka nanga (free) nerve ending.

**Q10** PHYSIOLOGY

★ Asked directly from our Rank Booster lectures

A cardiac (ventricular) action potential with phases 0-4 marked was shown. Which channel events occur in phase 3?

 Graph of a cardiac action potential with phases 0, 1, 2, 3, 4 labeled; phase 3 annotated as repolarization.

- A Na<sup>+</sup> channel opens
- B Ca<sup>++</sup> channel opens
- C Ca<sup>++</sup> channel close and K<sup>+</sup> opens
- D Slow depolarization

**Answer** Calcium channels close and potassium channels open (decreased Ca<sup>2+</sup> influx, increased K<sup>+</sup> efflux — rapid repolarization)

Phase 3 of the ventricular action potential is rapid repolarization: L-type calcium channels inactivate/close while delayed rectifier potassium channels open, so potassium efflux dominates and returns the membrane to resting potential. All three sources describe exactly this in slightly different words — Na<sup>+</sup> channel opening is phase 0, Ca<sup>2+</sup> opening is phase 2 (plateau), and slow depolarization belongs to pacemaker phase 4.

 Phase 3 = calcium ka gate band, potassium ka darwaza khula — repolarization ki seedhi dhalan.

**Q11** **PHYSIOLOGY**

An action potential tracing was shown. Match the state of the voltage-gated sodium channel during resting membrane potential, depolarization, and repolarization.

 Graph of a neuronal action potential (mV vs ms) with resting membrane potential, depolarization, repolarization, and hyperpolarization phases labeled.

- A** Open, Closed, Inactive
- B** Closed, Open, Inactive
- C** Inactive, Closed, Open
- D** Closed, Inactive, Open

**Answer** **Closed (at RMP) → Open (during depolarization) → Inactivated (during repolarization)**

At resting membrane potential the activation (m) gate is closed and the inactivation (h) gate open — the channel is closed but available; depolarization opens the m gate (channel open, Na<sup>+</sup> influx); shortly after, the h gate shuts, putting the channel in the inactivated state during repolarization — this inactivation underlies the absolute refractory period.

💡 Na<sup>+</sup> channel ki kahani: band → khula → inactivated — aur inactivated hi absolute refractory period ka raaz hai.

**Q12** **PHYSIOLOGY**

An adult male recovering from a head injury develops intense thirst, drinks about 8 liters of water per day, and passes large volumes of dilute urine; blood sugar is normal. Which hypothalamic nucleus (and its descending tract) is affected?

- A** Paraventricular
- B** Supraoptic
- C** Suprachiasmatic
- D** Arcuate

**Answer** **Supraoptic (and paraventricular) nuclei — damage to the supraoptico-hypophyseal (hypothalamo-hypophyseal) tract causing central diabetes insipidus**

Polyuria with dilute urine, polydipsia, and normal glucose after head trauma is central diabetes insipidus from ADH deficiency. ADH (vasopressin) is synthesized mainly in the supraoptic nucleus (with roughly one-sixth from the paraventricular nucleus, which is the chief oxytocin source) and transported down the supraopticohypophyseal tract to the posterior pituitary. The suprachiasmatic nucleus is the circadian pacemaker and does not produce the circulating ADH pool.

💡 ADH ka factory supraoptic hai; suprachiasmatic sirf body-clock chalata hai — DI me usse mat pheno.

**Q13**

PHYSIOLOGY

★ Asked directly from our Rank Booster lectures

A 48-year-old man has persistent fatigue, joint pain, and generalized skin hyperpigmentation. Serum ferritin is markedly elevated with transferrin saturation of 65%, and genetic analysis confirms a homozygous HFE mutation. What is the underlying mechanism of iron overload?

**A** increased ferroportin

**B** increased plasma iron

**Answer** **Decreased hepcidin → decreased degradation of ferroportin (increased ferroportin) → increased intestinal iron absorption**

The HFE mutation (hereditary hemochromatosis) impairs hepatic hepcidin production; since hepcidin normally binds ferroportin and triggers its internalization and degradation, low hepcidin leaves ferroportin abundant on enterocytes and macrophages, driving unregulated iron export into plasma and progressive iron overload. All three sources describe this same hepcidin-ferroportin axis; among the recalled options, 'increased ferroportin' is the correct choice.



Hepcidin hai iron ka darwaan; HFE me darwaan gayab, ferroportin ka darwaza khula — iron andar hi andar bharta jaata hai.

## 03 Biochemistry

8 recalled questions

### Q14 BIOCHEMISTRY

★ Asked directly from our Rank Booster lectures

A patient post-bariatric surgery presents with neurological manifestations including tingling sensation, gait abnormality, and sensory and motor weakness. Which assay should be performed?

- A B12 assay
- B Thiamine assay

**Answer Vitamin B12 assay**

A purely neurological picture after bariatric surgery - tingling, sensory ataxia with gait abnormality, and sensorimotor weakness - is subacute combined degeneration from vitamin B12 malabsorption (loss of intrinsic factor and gastric acid after bypass). Thiamine deficiency after bariatric surgery presents as Wernicke encephalopathy (confusion, ophthalmoplegia, ataxia) or as wet beriberi with high-output cardiac failure, neither of which is described in this stem.

💡 Bariatric ke baad: sirf nerves kharab = B12; dil (raised JVP, edema) + nerves = thiamine.

### Q15 BIOCHEMISTRY

★ Asked directly from our Rank Booster lectures

A patient experiences abdominal pain without peritoneal signs, precipitated by fasting or a low carbohydrate diet, which subsides after hemin administration. What is the most likely diagnosis?

**Answer Porphobilinogen deaminase (HMB synthase) deficiency - acute intermittent porphyria**

Neurovisceral abdominal pain without peritoneal signs, precipitated by fasting or low carbohydrate intake and relieved by hemin (which represses ALA synthase 1), is the classic attack of acute intermittent porphyria, caused by porphobilinogen deaminase deficiency. The photosensitive porphyrias (porphyria cutanea tarda, erythropoietic protoporphyria) cause skin lesions, not neurovisceral attacks.

💡 Pet dard, koi peritoneal sign nahi, hemin se aaram = AIP - enzyme PBG deaminase.

### Q16 BIOCHEMISTRY

★ Asked directly from our Rank Booster lectures

A patient presents with an X-linked inherited urea cycle defect, showing accumulation of carbamoyl phosphate and orotic acid, and reduced citrulline. What is the most likely enzyme deficiency?

**Answer Ornithine transcarbamylase (OTC) deficiency**

OTC deficiency is the only X-linked urea cycle disorder. Blocked conversion of carbamoyl phosphate plus ornithine to citrulline makes citrulline low, while excess carbamoyl phosphate spills into the pyrimidine pathway producing orotic aciduria with hyperammonemia. Hereditary orotic aciduria (UMP synthase deficiency) has orotic acid but no hyperammonemia, and citrullinemia has high, not low, citrulline.

💡 X-linked + orotic aciduria + low citrulline + high ammonia = OTC deficiency, fixed combination.

**Q17** BIOCHEMISTRY

Which specific phospholipid is associated with tumor proliferation and invasion, promoting glycolysis and suppressing oxidative phosphorylation (Warburg effect)?

- A Lysophosphatidic acid
- B Phosphatidylserine
- C Cardiolipin
- D Diphosphatidylglycerol inhibition of electron transport chain

**Answer** Lysophosphatidic acid

Lysophosphatidic acid (LPA) is the prototype oncolipid: acting through LPA1-6 G-protein-coupled receptors it drives tumor cell proliferation, migration and invasion, and reprograms metabolism toward aerobic glycolysis (Warburg effect). Cardiolipin and diphosphatidylglycerol are the same molecule (a mitochondrial inner-membrane lipid needed for electron transport chain function) - its appearance twice in the recalled options indicates garbled recall, and it is not the lipid credited with proliferation/invasion signaling.

💡 Cancer ka signalling lipid = LPA - proliferation, invasion aur Warburg, sab isi ke naam.

**Q18** BIOCHEMISTRY

A patient with bronchogenic carcinoma (lung mass) has elevated, heat-stable alkaline phosphatase, with no bone or liver metastasis. Which type of alkaline phosphatase is likely elevated?

- A Non-specific alkaline phosphatase (NSAP)
- B Placental alkaline phosphatase (PLAP)
- C Germ cell alkaline phosphatase (GCAP)
- D Intestinal alkaline phosphatase (IAP)
- E Tissue non-specific alkaline phosphatase (TNAP)

**Answer** Placental alkaline phosphatase (PLAP) - Regan isoenzyme

Ectopic production of a heat-stable alkaline phosphatase by bronchogenic carcinoma is the Regan isoenzyme, which is biochemically identical to placental alkaline phosphatase. Tissue non-specific (liver/bone/kidney) ALP is heat-labile and would imply hepatic or skeletal disease, which the stem excludes.

💡 'Regan can stand the heat' - heat-stable ALP from a lung cancer = placental (Regan) isoenzyme.

**Q19** BIOCHEMISTRY

A patient from Rajasthan presents with mottled enamel and skeletal changes. A DEXA scan was shown with a markedly raised score (Z score of 5 recalled). What is the diagnosis?

 Clinical photograph of teeth showing mottled enamel with brown staining and pitting (dental fluorosis); a DEXA readout with a markedly raised score accompanied it.

**Answer** Endemic skeletal fluorosis (excess fluoride)

Rajasthan is an endemic high-fluoride groundwater belt; mottled, pitted enamel plus skeletal changes with markedly INCREASED bone density on DEXA (strongly positive score) is pathognomonic of skeletal fluorosis. Osteoporosis gives a negative score; no other condition combines dental mottling with dense, brittle bone and interosseous membrane ossification.

💡 Rajasthan + daagdaar daant + patthar jaisi dense haddi = fluorosis.

**Q20** BIOCHEMISTRY

A diabetic patient on adequate insulin still has persistently elevated blood glucose levels (>250 mg/dL). Cortisol levels are elevated. What is the most likely reason for the persistent hyperglycemia?

- A** Inhibition of hepatic glycogenolysis
- B** Translocation of GLUT4 transporters
- C** Increased insulin sensitivity
- D** Counter-regulatory action of hormones causing insulin resistance

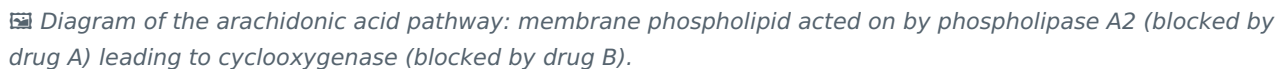
**Answer** Counter-regulatory action of hormones causing insulin resistance

Cortisol is a counter-regulatory hormone: it stimulates hepatic gluconeogenesis and induces peripheral insulin resistance, so glucose stays high despite adequate insulin dosing. GLUT4 translocation and increased insulin sensitivity would LOWER glucose, and cortisol promotes rather than inhibits hepatic glucose output, so the other options act in the wrong direction.

💡 Cortisol high = insulin ka kaam sabotage - resistance plus gluconeogenesis.

**Q21** BIOCHEMISTRY

In the arachidonic acid pathway, drug A inhibits phospholipase A2, while drug B inhibits cyclooxygenase (COX). Which correctly identifies drugs A and B?

 Diagram of the arachidonic acid pathway: membrane phospholipid acted on by phospholipase A2 (blocked by drug A) leading to cyclooxygenase (blocked by drug B).

- A** A = Steroid; B = NSAID
- B** A = NSAID; B = Steroid
- C** A and B are both steroids
- D** A and B are both NSAIDs

**Answer** A = Steroid (glucocorticoid); B = NSAID

Glucocorticoids induce annexin-A1 (lipocortin), which inhibits phospholipase A2 - blocking release of arachidonic acid and hence BOTH the prostaglandin and leukotriene arms. NSAIDs act one step lower, inhibiting cyclooxygenase only, so prostaglandin synthesis stops while leukotriene synthesis continues.

💡 Steroid pathway ke upar se rokta hai (PLA2), NSAID neeche se (COX) - isliye steroid dono branch band karta hai.

**50%+ of this paper was asked directly from our Rank Booster lectures.**

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## 04 Pharmacology

11 recalled questions

### Q22 PHARMACOLOGY

A patient treated with penicillin for infective endocarditis develops hemolysis with a positive Coombs test. An image showed antibody formation (T helper cell activating B cells), an RBC, and splenic destruction. What is the mechanism of the hemolysis?

 Schematic showing T-helper-cell-driven B-cell antibody production against a drug-coated RBC, with destruction of the antibody-coated RBC in the spleen.

- A Molecular mimicry
- B Complement mediated lysis
- C RBC membrane alteration
- D Drug binds and makes it a complete antigen
- E Defective apoptosis of autoantibody producing cells

**Answer** Drug binds and makes it a complete antigen (hapten mechanism)

High-dose penicillin causes immune hemolytic anemia by the classic hapten mechanism: the drug binds covalently to RBC membrane proteins, the hapten-carrier complex becomes a complete antigen, and IgG anti-penicillin antibodies coat the cells, which are then destroyed extravascularly in the spleen — exactly what the exam image depicted (antibody formation plus splenic destruction), with a positive IgG direct Coombs test. Complement-mediated intravascular lysis is the immune-complex 'innocent bystander' mechanism of drugs like quinidine, not penicillin.

💡 Penicillin hemolysis = hapten — drug RBC pe chipak ke complete antigen banta hai, spleen mein safaya hota hai.

### Q23 PHARMACOLOGY

★ Asked directly from our Rank Booster lectures

A traveler develops persistent diarrhea and is diagnosed with giardiasis that is resistant to metronidazole. What is the next drug of choice?

- A Nitazoxanide
- B Paromomycin
- C Spiramycin
- D Miltefosine

**Answer** Nitazoxanide

Nitazoxanide is the recommended next agent for nitroimidazole (metronidazole/tinidazole)-refractory giardiasis. Paromomycin is mainly reserved for giardiasis in pregnancy, miltefosine is for leishmaniasis, and spiramycin is for toxoplasmosis in pregnancy - none treats resistant Giardia.

💡 Metro fail? Nita on call - nitazoxanide for stubborn Giardia.

**Q24** PHARMACOLOGY

During a surgical procedure with total intravenous anesthesia, an opioid with extremely rapid onset and offset, metabolized mainly through plasma and tissue esterase enzymes, is required. Which drug is most likely useful in this case?

- A** Fentanyl
- B** Alfentanil
- C** Remifentanyl
- D** Sufentanil

**Answer** **Remifentanyl**

Remifentanyl's ester linkage is hydrolyzed by non-specific plasma and tissue esterases (organ-independent - and note, NOT pseudocholinesterase as one recall session stated), giving ultra-rapid onset/offset and a context-insensitive half-time of about 3-4 minutes, ideal for TIVA infusions with propofol and for day-care surgery. Fentanyl, sufentanil and alfentanil depend on hepatic CYP metabolism and accumulate with prolonged infusion.

💡 Remi = remote se on/off - esterase turant kaat de, liver ka intezaar nahi.

**Q25** PHARMACOLOGY

A 60-year-old woman with advanced cancer on palliative therapy including morphine for pain is experiencing severe constipation. What drug can reduce the gastrointestinal side effect without affecting morphine's analgesic action?

- A** Alvimopan
- B** Naltrexone
- C** Naloxone
- D** Nalmefene

**Answer** **Alvimopan**

Alvimopan is a peripherally acting mu-opioid receptor antagonist (PAMORA) that is excluded from the CNS, so it reverses the opioid effect on the gut without touching central analgesia; the recalled 'LV mopan' is the same drug misheard. Naloxone, naltrexone and nalmefene cross the blood-brain barrier and would reverse analgesia and precipitate withdrawal. (Among PAMORAs, methylnaltrexone and naloxegol are the agents formally approved for opioid-induced constipation in palliative care — alvimopan is licensed for post-operative ileus — but it was the only peripheral antagonist in the recalled option set.)

💡 Peripheral antagonist chuno: pet saaf, dard-rahata safe.



**Q26** PHARMACOLOGY

An elderly man with heart failure taking spironolactone develops gynecomastia (bilateral breast enlargement). What is the mechanism of this side effect?

- A Peripheral androgen receptor blocker
- B Decrease testosterone synthesis
- C Increase level of prolactin
- D Estrogen receptor agonist
- E Decrease androgen binding protein

**Answer** Peripheral androgen receptor blockade

Spironolactone causes gynecomastia chiefly by competitive blockade of peripheral androgen receptors - the same anti-androgen action exploited when it is used for hirsutism in PCOS. It also modestly inhibits testosterone synthesis, but receptor blockade is the primary mechanism and the better answer when both options appear; it does not raise prolactin or directly stimulate estrogen receptors.

💡 Spironolactone = anti-aldosterone + anti-androgen - isi liye ladies mein hirsutism ki dawa, gents mein gynecomastia ki wajah.

**Q27** PHARMACOLOGY

A 68-year-old patient with symptomatic chronic heart failure with reduced ejection fraction, recently hospitalized for worsening heart failure despite guideline-directed therapy, has vericiguat added to his treatment. Which of the following best describes the mechanism of action of vericiguat?

- A Direct stimulation of cyclic GMP production
- B Nitric oxide mediated activation of cyclic GMP

**Answer** Direct stimulation of soluble guanylate cyclase, increasing cyclic GMP production

Vericiguat directly stimulates soluble guanylate cyclase, boosting cGMP production independently of (and synergistically with) nitric oxide; it earned its HFrEF indication for patients with recent worsening heart failure in the VICTORIA trial, and riociguat is the same-class drug for pulmonary hypertension. NO-mediated cGMP generation is how nitrates work - the distractor.

💡 Vericiguat NO ka mohtaaj nahi - seedha sGC ko jagaata hai.

**Q28** PHARMACOLOGY

A 60-year-old woman with urinary urgency, increased frequency, and urge incontinence (overactive bladder) developed severe dry mouth on an antimuscarinic agent. The physician switches her to mirabegron. What is the mechanism of action of mirabegron?

- A M3 agonist
- B M3 antagonist
- C Beta-3 agonist
- D Alpha-1 blocker

**Answer** Beta-3 agonist

Mirabegron is a beta-3 adrenergic agonist that relaxes the detrusor muscle during the storage phase, increasing bladder capacity without the antimuscarinic burden of dry mouth and constipation. M3 antagonists (oxybutynin, solifenacin) are the class she could not tolerate, and alpha-1 blockers treat outflow obstruction, not urgency.

💡 Overactive bladder + dry mouth problem = beta-3 se bladder ko 'be at ease' - mirabegron.

**Q29** PHARMACOLOGY

A 50-year-old man with chronic bronchitis has thick, tenacious bronchial secretions that are difficult to expectorate. He is prescribed N-acetylcysteine (NAC) as a mucolytic agent. Which of the following best explains its mechanism of action?

- A It breaks down the disulfide bonds, which reduces mucus viscosity
- B It improves surfactant production
- C It inhibits vagus-mediated bronchial secretion
- D It increases ciliary movement by beta-2 action

**Answer** It breaks down the disulfide bonds in mucus glycoproteins, reducing mucus viscosity

NAC's free sulfhydryl group cleaves the disulfide bridges cross-linking mucus glycoproteins, dropping sputum viscosity so it can be expectorated. The same thiol chemistry replenishes glutathione, which is why NAC doubles as the paracetamol-poisoning antidote. Surfactant stimulation is bromhexine/ambroxol territory, and vagal or ciliary mechanisms are distractors.

💡 NAC = disulfide cutter - balgam bhi patla, liver bhi safe.

**Q30** PHARMACOLOGY

★ Asked directly from our Rank Booster lectures

A 30-year-old woman started on an oral contraceptive while receiving rifampicin for tuberculosis develops an unintended pregnancy a few months later. What is the most likely mechanism responsible for the oral contraceptive failure?

- A Enzyme induction

**Answer** Microsomal (CYP) enzyme induction by rifampicin

Rifampicin is a potent inducer of hepatic microsomal enzymes (CYP3A4 and others), accelerating the metabolism of ethinyl estradiol and progestins so that contraceptive hormone levels fall and the pill fails. This is a pharmacokinetic interaction at the level of metabolism - enzyme induction.

💡 Rifampicin sabka inducer - OCP, warfarin, sabki dawa fail karwa deta hai.

**Q31** PHARMACOLOGY

Avasimibe lowers cholesterol by acting on which pathway?

**Answer** Inhibition of ACAT (acyl-CoA:cholesterol acyltransferase), blocking cholesterol esterification

Avasimibe is an ACAT (acyl-CoA:cholesterol acyltransferase) inhibitor: it blocks intracellular esterification of cholesterol, and was developed (and later abandoned in trials) as an anti-atherosclerotic lipid-modifying agent. It is also a known CYP3A4 inducer — trivia occasionally attached to it.

💡 Avasimibe = ACAT inhibitor - cholesterol ki ester-factory pe taala.

**Q32** PHARMACOLOGY

★ Asked directly from our Rank Booster lectures

What is the antidote for paracetamol poisoning?

**Answer** N-acetylcysteine

N-acetylcysteine replenishes hepatic glutathione, detoxifying the reactive NAPQI metabolite that accumulates once sulfation and glucuronidation are saturated in paracetamol overdose. It is most effective within about 8 hours of ingestion but is given even late in established hepatotoxicity.

💡 PCM ka zeher NAPQI, uski kaat NAC - glutathione ka refill.

## 05 Pathology

14 recalled questions

### Q33 PATHOLOGY

★ Asked directly from our Rank Booster lectures

A liver biopsy (patient with transfusion-dependent thalassemia and iron overload) is shown on routine H&E staining. The hepatocytes contain abundant coarse brown granular pigment, suggesting excessive iron deposition. Which stain confirms iron?

 H&E liver biopsy showing coarse brown granular hemosiderin pigment within hepatocytes.

- A Perls' Prussian blue stain
- B Masson trichrome stain
- C Oil Red O stain
- D Periodic acid-Schiff (PAS) stain

Answer **Perls' Prussian blue stain**

Perls' Prussian blue is the specific histochemical stain for ferric iron (hemosiderin), confirming secondary hemochromatosis in a chronically transfused thalassemic. The distractors stain other materials: Masson trichrome stains collagen/fibrosis, Oil Red O stains neutral fat, and PAS stains glycogen and fungal walls.

 Tissue mein iron dikhana ho to ek hi naam - Perls' Prussian blue.

### Q34 PATHOLOGY

A testicular biopsy shows a large, well-circumscribed, solid, bulky, homogeneous mass with fibrous septa, clear cells, and lymphocytic infiltrate. Immunohistochemistry shows OCT3/4, Nanog, and c-kit (CD117) positivity. AFP and beta-hCG are negative/normal. What is the diagnosis?

- A Seminoma
- B Embryonal carcinoma
- C Yolk sac tumor
- D Choriocarcinoma

Answer **Seminoma**

Sheets of clear cells divided by fibrous septa with lymphocytic infiltrate, positive for OCT3/4, Nanog, and c-kit (CD117), is classic seminoma. Embryonal carcinoma is also OCT3/4-positive but is CD30-positive and c-kit-negative with hemorrhagic, ill-defined growth; negative AFP excludes yolk sac tumor and negative hCG excludes choriocarcinoma. (One recall mentioned alkaline phosphatase negativity - likely a recall error, since seminoma is typically PLAP-positive - but the c-kit/OCT3/4 profile still clinches the diagnosis.)

 Testis tumor with CD117 + OCT3/4 + fried-egg cells + lymphocytes = seminoma, seedha jawab.

**Q35**

PATHOLOGY

★ Asked directly from our Rank Booster lectures

A central/hilar lung tumor in a smoker shows synaptophysin positivity. Biopsy describes small cells with a high nuclear-to-cytoplasmic ratio and increased mitosis. What is the likely diagnosis?

- A Small cell carcinoma
- B Large cell cancer
- C Lymphoma
- D Adenocarcinoma

**Answer** Small cell carcinoma

A central tumor of small cells with scant cytoplasm (high N:C ratio), nuclear molding/crush artifact, brisk mitoses, and neuroendocrine marker (synaptophysin) positivity in a smoker is small cell lung carcinoma. Lymphoma is LCA-positive and synaptophysin-negative; adenocarcinoma is peripheral and TTF-1/napsin-positive with gland formation; large cell carcinoma shows large cells with prominent nucleoli.

💡 Hilar mass + chhote crushed cells + synaptophysin = small cell carcinoma.

**Q36**

PATHOLOGY

★ Asked directly from our Rank Booster lectures

A 45-year-old woman presents with a rapidly enlarging breast mass, approximately twice the size of the opposite breast, with an irregular nodular surface on gross examination. Histology shows a leaf-like pattern with increased stromal cellularity and stromal atypia; the mitotic index is low, with no significant stromal overgrowth or tumor necrosis. What is the most likely diagnosis?

- A Fibroadenoma
- B Phyllodes tumor
- C Invasive ductal carcinoma
- D Fibrocystic change

**Answer** Phyllodes tumor

Leaf-like stromal fronds projecting into cleft-like spaces (phyllodes is Greek for leaf-like) with increased stromal cellularity in a rapidly enlarging breast mass is a phyllodes tumor; low mitotic count without stromal overgrowth or necrosis grades it as benign. Fibroadenoma is the key distractor but lacks stromal hypercellularity/atypia and rapid disproportionate growth.

💡 Patte jaisa pattern + cellular stroma = phyllodes - fibroadenoma ka bada, tez bhai.

**Q37**

PATHOLOGY

★ Asked directly from our Rank Booster lectures

A 6-year-old child presents with leukocoria. H&E histology of the intraocular tumor shows Flexner-Wintersteiner rosettes. Which of the following is most likely to promote malignant transformation?

 H&E histology showing Flexner-Wintersteiner rosettes (true rosettes with a central lumen), characteristic of retinoblastoma.

- A RB1 gain of function
- B TP53 gain of function
- C RB1 loss of function
- D TP53 loss of function

**Answer** RB1 loss of function

Leukocoria in a child with Flexner-Wintersteiner (true) rosettes on histology is retinoblastoma, caused by biallelic loss of the RB1 tumor suppressor gene on 13q14 per Knudson's two-hit hypothesis. RB1 is a tumor suppressor, so it transforms by LOSS of function - a 'gain of function' of a tumor suppressor is the deliberately wrong distractor.

💡 Tumor suppressor kabhi gain se cancer nahi karta - retinoblastoma = RB1 loss of function, dono alleles.

**Q38**

PATHOLOGY

★ Asked directly from our Rank Booster lectures

A female patient presents with recurrent mucocutaneous bleeding, bruising, and menorrhagia (family history in some recalls). Bleeding time is elevated and APTT is prolonged. The ristocetin aggregation test is abnormal, and Factor VIII activity is 50% of normal. What is the most likely diagnosis?

- A Von Willebrand disease
- B Bernard-Soulier syndrome
- C Glanzmann thrombasthenia
- D Hemophilia A

**Answer** von Willebrand disease

Only von Willebrand disease derails BOTH arms: platelet adhesion (prolonged bleeding time, abnormal ristocetin aggregation) and the intrinsic pathway (vWF stabilizes factor VIII, so FVIII falls to ~50% and APTT prolongs). Bernard-Soulier and Glanzmann are pure platelet disorders with normal APTT, and hemophilia A (X-linked, mostly males) has normal bleeding time and ristocetin testing.

💡 BT bhi kharab, APTT bhi kharab, ristocetin fail = vWD - do bimariyon ka combo ek gene mein.

**Q39**

PATHOLOGY

An elderly male, treated for ischemic heart disease for 10 years, dies of acute decompensated heart failure. At autopsy the lungs are heavy, firm, and reddish-brown. What is the characteristic microscopic finding of this chronic pulmonary congestion?

- A Hemosiderin-laden macrophages
- B Lipid-laden macrophages
- C Anthracotic pigment
- D Neutrophilic infiltrate

**Answer** Hemosiderin-laden macrophages (heart failure cells)

Chronic left-sided heart failure causes longstanding pulmonary venous congestion with alveolar microhemorrhages; alveolar macrophages phagocytose the extravasated red cells and accumulate hemosiderin, becoming 'heart failure cells' and producing the firm reddish-brown lungs (brown induration). Anthracotic pigment is inhaled carbon seen in smokers and city dwellers and has nothing to do with congestion; neutrophils would indicate bronchopneumonia.

💡 Brown induration of lung = heart failure cells = hemosiderin-laden macrophages.

**Q40**

PATHOLOGY

★ Asked directly from our Rank Booster lectures

What metaplastic change characterizes Barrett's esophagus?

**Answer** Intestinal metaplasia - columnar epithelium with goblet cells

In Barrett's esophagus, chronic gastroesophageal reflux converts the distal esophageal stratified squamous epithelium into intestinal-type columnar epithelium containing goblet cells. This intestinal metaplasia is the premalignant substrate for esophageal adenocarcinoma via the dysplasia-carcinoma sequence.

💡 Esophagus mein goblet cell mila = Barrett confirm - adenocarcinoma ka waiting room.

**Q41** PATHOLOGY

★ Asked directly from our Rank Booster lectures

A patient presents with lower lobe (panacinar) emphysema and abnormal liver function tests. Liver biopsy shows PAS-positive, diastase-resistant globules. What is the diagnosis?

**Answer** Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin deficiency is the one condition combining lower-lobe panacinar emphysema (unopposed elastase activity) with liver disease; the misfolded PiZ protein polymerizes in hepatocytes as PAS-positive, diastase-resistant globules. Smoking-related emphysema is centriacinar and upper-lobe and does not involve the liver.

💡 Neeche ke lobe ka emphysema + liver mein PAS-positive globules = A1AT deficiency - ek protein, do organ.

**Q42** PATHOLOGY

★ Asked directly from our Rank Booster lectures

A patient presents with very high blood pressure. Kidney biopsy shows an onion-skin appearance of the blood vessels. What is the likely diagnosis/finding?

- A Hyperplastic arteriolosclerosis
- B Malignant hypertension
- C Polyarteritis nodosa
- D Hyaline arteriolosclerosis

**Answer** Hyperplastic arteriolosclerosis

Concentric, laminated ('onion-skin') proliferation of smooth muscle cells in arteriolar walls, often with fibrinoid necrosis, is hyperplastic arteriolosclerosis - the vascular lesion of malignant hypertension. Hyaline arteriolosclerosis (homogeneous pink wall thickening) belongs to benign hypertension and diabetes, and polyarteritis nodosa shows transmural fibrinoid necrosis of medium vessels, not concentric lamination.

💡 Onion skin = hyperplastic arteriolosclerosis = malignant HTN ka biopsy signature.

**Q43** PATHOLOGY

★ Asked directly from our Rank Booster lectures

A question on molecular mimicry - most likely asking which condition is an example of it (recalled contenders: Guillain-Barre syndrome, PSGN, acute rheumatic fever/endocarditis).

**Answer** Acute rheumatic fever (molecular mimicry — the classic example)

The classic molecular mimicry disease is acute rheumatic fever — streptococcal M protein cross-reacting with cardiac myosin. Guillain-Barre syndrome (Campylobacter lipo-oligosaccharide vs nerve gangliosides) and PSGN are the other standard examples the options may have carried.

💡 Molecular mimicry ke poster boys: rheumatic fever, GBS, aur PSGN - teeno mein antibody galat ghar pe attack karti hai.

**Q44** PATHOLOGY

In a patient with IgG-positive toxoplasmosis, what is the mechanism by which Toxoplasma gondii causes lesions?

**Answer** Reactivation of latent cysts with immune-mediated inflammation

Toxoplasma CNS lesions arise from reactivation of latent tissue cysts (IgG-positive latent infection) with tachyzoite-driven cell lysis and necrosis, amplified by the host immune-mediated inflammatory response.

💡 Toxo ka damage do-tarfa hai - parasite ki direct necrosis + host ki inflammation.

**Q45**

PATHOLOGY

★ Asked directly from our Rank Booster lectures

An image-based question showing a flow cytometry scatter plot in the context of RBC membrane destruction. What is the diagnosis being evaluated?

 Flow cytometry scatter plot with cell populations across four quadrants, used to demonstrate CD55/CD59 expression for PNH diagnosis.

**Answer** Paroxysmal nocturnal hemoglobinuria - loss of CD55/CD59 on flow cytometry

PNH results from an acquired PIGA mutation in a hematopoietic stem cell, causing loss of GPI anchors and hence absence of the complement-regulatory proteins CD55 (DAF) and CD59 (MIRL) on blood cells, producing complement-mediated intravascular hemolysis. Flow cytometry demonstrating the CD55/CD59-deficient population (or FLAER assay) is the diagnostic test of choice.

 PNH confirm karna hai to flow cytometry pe CD55/59 gayab dikhaao - Ham test ab history hai.

**Q46**

PATHOLOGY

★ Asked directly from our Rank Booster lectures

What is the characteristic biopsy finding in minimal change disease?

**Answer** Normal light microscopy; electron microscopy shows diffuse effacement of podocyte foot processes

In minimal change disease the glomeruli appear normal on light microscopy and immunofluorescence is negative; the diagnosis rests on electron microscopy showing diffuse effacement (fusion) of podocyte foot processes. This is why it is the classic 'EM-only' diagnosis in a child with nephrotic syndrome.

 Minimal change = LM pe kuch nahi, EM pe sab kuch - foot process effacement.

## 06 Microbiology

13 recalled questions

### Q47 MICROBIOLOGY

★ Asked directly from our Rank Booster lectures

An image of a flexible endoscope was shown. What is the appropriate method for its sterilization/high-level disinfection?

 Schematic line drawing of a flexible endoscope.

- A 2 percent Glutaraldehyde / CIDEX
- B 160 degrees dry heat / Hot air oven
- C 121 degree / Autoclave
- D H<sub>2</sub>O<sub>2</sub>

**Answer** 2% glutaraldehyde (Cidex)

Flexible endoscopes are heat-labile semi-critical devices — autoclaving and hot-air ovens would destroy them — so they undergo high-level disinfection with 2% glutaraldehyde (Cidex) or ortho-phthalaldehyde. All four sources gave glutaraldehyde; hydrogen peroxide (plasma) systems are used for rigid scopes like laparoscopes/arthroscopes, which was the distractor.

💡 Scope garmi se marta hai, germ Cidex se — flexible endoscope hamesha 2% glutaraldehyde.

### Q48 MICROBIOLOGY

★ Asked directly from our Rank Booster lectures

What is the mechanism of action of diphtheria toxin?

- A EF2

**Answer** ADP-ribosylation of elongation factor-2 (EF-2), inhibiting protein synthesis

Diphtheria toxin fragment A ADP-ribosylates elongation factor-2, halting ribosomal translocation and shutting down host protein synthesis - the same target as Pseudomonas exotoxin A. By contrast, cholera and pertussis toxins ADP-ribosylate G-proteins, and Shiga toxin cleaves 28S rRNA.

💡 Diphtheria aur Pseudomonas - dono EF-2 ke dushman.

### Q49 MICROBIOLOGY

★ Asked directly from our Rank Booster lectures

Which enrichment medium promotes the rapid early growth of Corynebacterium diphtheriae?

- A Loeffler's medium
- B Tinsdale medium
- C Tellurite medium
- D LJ medium

**Answer** Loeffler's serum slope (Loeffler's medium)

Loeffler's serum slope is the enrichment medium on which C. diphtheriae grows within 6-8 hours and shows metachromatic (Babes-Ernst) granules on Albert stain - exactly matching 'rapid early growth'. Tellurite-containing media (McLeod, Tinsdale) are selective-indicator media that need around 48 hours and are used for isolation from mixed flora, and LJ medium is for mycobacteria.

💡 Loeffler = lightning fast (6-8 hrs); tellurite = selective par sust (48 hrs).



**Q50**

MICROBIOLOGY

★ Asked directly from our Rank Booster lectures

A patient with sepsis develops fever, a rapidly progressing purpuric rash, hypotension, hyponatremia, hyperkalemia, and shock; gram-negative diplococci and CSF findings are consistent with meningococcal infection. Imaging demonstrates bilateral adrenal hemorrhage. What is this syndrome called?

- A Waterhouse-Friderichsen syndrome
- B Addison's crisis
- C Conn's syndrome
- D Cushing's syndrome

**Answer** Waterhouse-Friderichsen syndrome

Fulminant meningococemia complicated by bilateral adrenal hemorrhage causes acute adrenal insufficiency - hypotension, hyponatremia, hyperkalemia, purpura and shock - and this constellation is Waterhouse-Friderichsen syndrome, most classically due to *Neisseria meningitidis*. Addison's crisis is the nearest distractor, but the acute infective purpuric setting with demonstrated adrenal hemorrhage defines WFS. (Sources that recalled the stem as asking for the causative agent answered *N. meningitidis* - the same clinical entity; the recalled option set shows the exam asked for the syndrome.)

💡 Meningococcus + adrenal blast = Waterhouse-Friderichsen; sepsis ke saath steroids bhi yaad rakho.

**Q51**

MICROBIOLOGY

★ Asked directly from our Rank Booster lectures

An HIV-positive patient presents with chronic watery diarrhoea. Stool examination shows acid-fast oocysts measuring 4-6 microns. What is the most likely organism?

 Question referenced stool microscopy showing small (4-6 micron) acid-fast oocysts.

- A *Cryptosporidium*
- B *Cyclospora*
- C *Isospora*
- D *Giardia*

**Answer** *Cryptosporidium*

In HIV with chronic watery diarrhea, uniformly acid-fast oocysts of 4-6 microns are *Cryptosporidium*. *Cyclospora* oocysts are larger (8-10 microns) and variably acid-fast, *Cystoisospora* (*Isospora*) is much larger (~25 microns) and elliptical, and *Giardia* cysts are not acid-fast at all - oocyst size is the discriminator the examiner wanted.

💡 Chhota (4-6 um) = Crypto; double (8-10) = *Cyclospora*; sabse bada = *Isospora*.

**Q52**

MICROBIOLOGY

★ Asked directly from our Rank Booster lectures

A 4-year-old child developed left parotid swelling, followed by right parotid swelling the next day, which subsided in one week. This condition could have been prevented by vaccination with which strain?

- A Oka strain
- B RA 27/3 strain
- C Jeryl Lynn strain

**Answer** Jeryl Lynn strain

Sequential bilateral parotitis in a child is mumps, and the mumps component of the MMR vaccine uses the live attenuated Jeryl Lynn strain (Indian brands may use L-Zagreb, which was not offered). Oka is the varicella vaccine strain and RA 27/3 is the rubella strain - both distractors.

💡 Mumps ka 'Jerry' = Jeryl Lynn; Oka = chickenpox, RA 27/3 = rubella.

**Q53**

MICROBIOLOGY

★ Asked directly from our Rank Booster lectures

Given CSF findings of increased protein, normal glucose, and lymphocytosis, what is the most common viral cause of this meningitis?

- A Echovirus
- B Adenovirus
- C HSV
- D Epstein-Barr virus
- E CMV

**Answer** Echovirus (enterovirus group)

Lymphocytic CSF with mildly raised protein and normal glucose is the picture of viral meningitis, and enteroviruses - represented by echovirus among the options - are its most common cause in immunocompetent patients. HSV-1 causes encephalitis more than meningitis, and adenovirus, EBV and CMV are uncommon culprits.

💡 CSF sugar normal + lymphocytes = viral; virus ka baap = enterovirus/echo.

**Q54**

MICROBIOLOGY

★ Asked directly from our Rank Booster lectures

A previously healthy 28-year-old woman presents with a severe headache for several days, followed by new onset seizure. There is no history of trauma. MRI of the brain demonstrates multiple cystic lesions measuring approximately 0.6-1.7 cm, involving the periventricular and meningeal regions. What is the most likely causative organism?

📷 MRI brain showing multiple cystic lesions (0.6-1.7 cm) in periventricular and meningeal regions.

- A Taenia solium
- B Cryptococcus neoformans
- C Toxoplasma gondii
- D Plasmodium falciparum

**Answer** Taenia solium

Multiple small cystic brain lesions with new-onset seizures in an immunocompetent young adult are neurocysticercosis from Taenia solium larvae - the most common cause of acquired epilepsy in India. Toxoplasma produces ring-enhancing lesions essentially in the immunocompromised, Cryptococcus gives gelatinous pseudocysts in advanced HIV, and P. falciparum causes diffuse cerebral malaria, not discrete cysts.

💡 Healthy Indian + seizure + multiple chhote cysts = NCC (T. solium) till proven otherwise.

**Q55**

MICROBIOLOGY

In cholera diarrhea, increased cyclic AMP leads to increased activity of which channel, causing secretion?

**Answer** Chloride channel (CFTR)

Cholera toxin ADP-ribosylates Gs-alpha, locking adenylyl cyclase in the active state; the resulting cAMP surge activates CFTR chloride channels on the apical enterocyte membrane, and chloride efflux drags sodium and water into the lumen - the rice-water stool. The same CFTR channel that is defective in cystic fibrosis is here pathologically overactive.

💡 Cholera = CFTR full speed - wahi channel jo CF mein band rehta hai.

**Q56** MICROBIOLOGY

Cholera toxin and pertussis toxin both cause an increase in cyclic AMP. How do they achieve this?

- A Gi inhibition
- B Phosphodiesterase
- C Adenylyl cyclase

**Answer** Both increase adenylyl cyclase activity - cholera toxin ADP-ribosylates and permanently activates Gs, while pertussis toxin ADP-ribosylates and inactivates Gi

The step common to both toxins is increased adenylyl cyclase activity: cholera toxin locks stimulatory Gs-alpha ON, whereas pertussis toxin switches inhibitory Gi-alpha OFF - both converge on more cAMP. 'Gi inhibition' alone describes only pertussis toxin (cholera toxin does not touch Gi), and phosphodiesterase - the enzyme that degrades cAMP - is inhibited by neither.

💡 Cholera accelerator dabata hai (Gs ON), pertussis brake kaat deta hai (Gi OFF) - engine (adenylyl cyclase) dono mein race karta hai.

**Q57** MICROBIOLOGY

Several students from the same school develop severe abdominal cramps and bloody diarrhea. Shiga toxin-producing E. coli (enterohemorrhagic E. coli) is suspected. Which reference method is considered the gold standard?

- A Vero cell cytotoxicity assay
- B Sorbitol MacConkey agar
- C ELISA

**Answer** Vero cell cytotoxicity assay

The stem asks for the reference/gold-standard method: the Vero cell cytotoxicity assay (with antibody neutralization) is the historical gold standard for demonstrating Shiga toxin - EHEC is literally 'verocytotoxigenic E. coli' (VTEC). Sorbitol MacConkey agar is the routine screening culture for the sorbitol non-fermenting O157:H7 serotype, but it misses non-O157 and sorbitol-fermenting strains, so it is not the reference method; ELISA is a rapid toxin screen.

💡 EHEC ka doosra naam hi VTEC hai - gold standard Vero cell pe; SMAC sirf screening.

**Q58** MICROBIOLOGY

A patient presents with hypopigmented, hypoesthetic skin lesions and thickened peripheral nerves suggesting leprosy. Which of the following statements about leprosy is correct?

- A Bacteriology index is fragmented bacilli divided by total bacilli.
- B Lepromin test represents type 3 hypersensitivity.
- C Mycobacterium leprae is stained using modified 20% H2SO4.
- D In lepromatous leprosy, numerous bacilli may be found within foamy cells.

**Answer** In lepromatous leprosy, numerous bacilli may be found within foamy cells

In lepromatous leprosy the bacilli multiply unchecked inside lipid-laden macrophages (Virchow/foam cells), often in clumps called globi - the correct statement. The bacteriological index grades the density of ALL bacilli on a logarithmic scale (the solid-staining fraction is the morphological index), lepromin positivity is type IV delayed hypersensitivity, and M. leprae is decolorized with 5% (not 20%) H2SO4 because it is less acid-fast than M. tuberculosis.

💡 LL = foam cells bhare globi; lepromin = type 4; acid sirf 5%.

**Q59**

MICROBIOLOGY

A question on Mollaret (recurrent benign aseptic/lymphocytic) meningitis - which virus is the most common cause?

**Answer** **Herpes simplex virus type 2 (HSV-2)**

The recalled phrase 'molar tooth sign in the context of meningitis with HSV' is almost certainly a mishearing of 'Mollaret' — the recalled question pairs Mollaret meningitis with the answer HSV. Mollaret meningitis is recurrent benign lymphocytic meningitis, and HSV-2 is its most common cause. The true molar tooth sign is an MRI midbrain finding of Joubert syndrome, unrelated to meningitis — a distractor trap if both appeared.

💡 Baar-baar aane wala benign aseptic meningitis = Mollaret = HSV-2; molar tooth sign alag cheez hai (Joubert).

**50%+ of this paper was asked directly from our Rank Booster lectures.**

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## 07 Forensic

7 recalled questions

### Q60 FORENSIC

★ Asked directly from our Rank Booster lectures

In a pregnancy complicated by substantial congenital anomaly of the fetus, termination of pregnancy requires approval from the state-level Medical Board after which period of gestation?

**Answer** After 24 weeks of gestation (Medical Board approval required)

Under the MTP (Amendment) Act 2021, one RMP can opine up to 20 weeks and two RMPs up to 24 weeks for special categories of women. Beyond 24 weeks, termination is permitted only for substantial fetal abnormality, and only with the approval of the state-level Medical Board (gynecologist, pediatrician, radiologist/sonologist plus members notified by the state government). All three sources gave the same answer in different words.

💡 MTP ka simple ladder: 1 doctor till 20 weeks, 2 doctors till 24, uske baad sirf Medical Board — aur sirf substantial fetal anomaly ke liye.

### Q61 FORENSIC

★ Asked directly from our Rank Booster lectures

A motorcyclist from a road traffic crash is brought to casualty unconscious, with severe head injuries, by police personnel. No relatives are present. What should the surgeon on duty do regarding consent for emergency surgery?

- A Wait for the consent
- B Consent from police is needed
- C No consent needed
- D Wait for relative

**Answer** No consent needed — proceed with life-saving emergency treatment/surgery

Under the doctrine of emergency (Section 30 of the Bharatiya Nyaya Sanhita 2023, corresponding to old Section 92 IPC), an act done in good faith for a person's benefit without consent is protected when the patient cannot consent and no guardian is available in time. Waiting for relatives or seeking police consent (police have no legal standing to consent for treatment) would constitute negligent delay.

💡 Life-threatening emergency me consent ka intezaar mat karo — law tumhare saath hai (BNS Sec 30), delay tumhare khilaaf.

### Q62 FORENSIC

An X-ray of the elbow showing the olecranon process at the upper end of the ulna was shown. What is the age of appearance and fusion of the olecranon ossification center?

 Elbow X-ray demonstrating the olecranon secondary ossification center at the upper end of the ulna.

**Answer** Appears at 8-10 years; fuses by 15-17 years

The secondary ossification center of the olecranon appears around 8-10 years (it is the 'O' before the external epicondyle in the CRITOE sequence, appearing ~10 years) and fuses with the shaft of the ulna by about 15-17 years (earlier in girls). The on-screen recall frame also marked 'Appear at 8-10 and fuse by 15-17 yrs' as the answer; the minor 15-17 vs 16-17 spread across sources is the same range, not a genuine dispute.

💡 Elbow forensic shortcut: olecranon aata hai ~10 saal pe, judta hai 15-17 pe — medicolegal age window for juvenile vs adult.

**Q63** FORENSIC

In a suspicious poisoning case, 5 ml of gastric aspirate was collected during gastric lavage. A filter paper impregnated with silver nitrate brought close to the sample turned black. What is the likely poisoning?

**Answer Aluminium phosphide (Celphos) poisoning**

Aluminium phosphide releases phosphine (PH<sub>3</sub>) gas on contact with gastric acid; phosphine reduces silver nitrate on the filter paper to black silver/silver phosphide — the classic bedside silver nitrate test. The recalled 'aluminium phosphate' is an obvious mis-recall of phosphide.

💡 Silver nitrate paper kala = phosphine = Celphos; the wheat-pill poison with no antidote — diagnosis is the whole game.

**Q64** FORENSIC

A patient employed in a steel alloy manufacturing plant presents with progressive rigidity, bradykinesia, and gait difficulty resembling Parkinson's disease. What toxicity does this indicate?

- A** Manganese
- B** Arsenic
- C** Vitamin A toxicity
- D** Carbon disulfide toxicity

**Answer Manganese toxicity (manganism)**

Chronic manganese exposure — classically in steel/ferroalloy, welding, and mining workers — damages the globus pallidus and produces a Parkinsonism-like picture (rigidity, bradykinesia, 'cock-walk' gait) that responds poorly to levodopa. Carbon disulfide can also cause parkinsonism but the occupational setting is the rayon/rubber industry, not steel alloy manufacture.

💡 Steel factory + Parkinson jaisa patient = manganese; rubber/rayon factory version is carbon disulfide.

**Q65** FORENSIC

A 32-year-old primigravida presents with absent fetal movements. On autopsy of the dead fetus, the ossification center at the lower end of the femur is present. What is the estimated fetal age?

 Autopsy/radiograph finding of an ossification center at the lower end of the fetal femur.

**Answer More than 36 weeks (about 9 months / near term)**

The ossification center of the distal (lower) end of the femur appears at about 36-38 weeks (9th month) of intrauterine life and is a classic medicolegal marker of a full-term fetus. The upper end of the tibia appears around the 10th month, and the upper end of the femur only after birth. The on-screen recall confirmed 'fetal age is more than 36 weeks'.

💡 Lower end of femur dikha = 36+ weeks — the courtroom marker of a term fetus.

**Q66****FORENSIC**

A 6-year-old boy with a history of anal sexual assault presents with pain on defecation. The anal area appears normal on initial inspection. What is the most appropriate position/method for examining the passive agent in anal intercourse?

- A** Lithotomy position
- B** Proctoscopic procedure
- C** Examination of the buttocks to look for relaxation of the anus
- D** Knee chest position

**Answer** **Knee-chest (knee-elbow) position**

The standard forensic examination of the passive agent of sodomy is performed in the knee-chest (knee-elbow) position, which gives the best exposure of the anus and perianal region and allows assessment of sphincter tone with gentle lateral traction of the buttocks. Lithotomy is used for female genital examination, and proctoscopy is an adjunct, not the primary method of positioning.



Sodomy victim examination = knee-elbow position; lithotomy is for the gynae couch, not this exam.

## 08 PSM

15 recalled questions

Q67

PSM

★ Asked directly from our Rank Booster lectures

A patient involved in a building collapse is conscious, oriented, shouting for help, and walking. He has abrasions on both legs with some bleeding, but no life- or limb-threatening injuries. What triage colour should be assigned?

- A Red
- B Yellow
- C Green

Answer **Green**

In mass-casualty triage (START and similar systems), any casualty who can walk is 'walking wounded' and is tagged green — minor injuries that can safely wait. Red is for immediately life-threatening but salvageable injuries and yellow for serious injuries whose treatment can be delayed; superficial abrasions with minor bleeding in an ambulatory, oriented patient meet neither.

💡 Chal ke aaya aur chilla raha hai = green — awaaz aur chaal dono zinda hone ki nishani.

Q68

PSM

In a population of 10,000, an outbreak of Japanese encephalitis occurred with 800 cases, of which 40 died. Calculate the case fatality rate (CFR).

Answer **5%**

CFR = (deaths due to the disease / total cases of the disease)  $\times 100 = 40/800 \times 100 = 5\%$ . The population figure is a distractor — it is the denominator for attack rate or death rate, never for CFR, whose denominator is cases only.

💡 CFR ka denominator hamesha CASES hote hain, population nahi —  $40/800 = 5\%$ .

Q69

PSM

★ Asked directly from our Rank Booster lectures

In a study, 70 out of 7,000 exposed individuals developed the disease, while 3 out of 3,000 unexposed individuals developed it. Calculate the attributable risk proportion.

Answer **90% (0.9)**

Incidence in exposed =  $70/7000 = 1\%$ ; incidence in unexposed =  $3/3000 = 0.1\%$ ; relative risk = 10. Attributable risk proportion (attributable fraction in the exposed) =  $(RR - 1)/RR = 9/10 = 90\%$ , or equivalently  $(1.0 - 0.1)/1.0$ . The absolute risk difference (0.9% or 9 per 1,000) answers a different question — the absolute attributable risk, not the proportion.

💡  $AR\% = (RR-1)/RR$  — RR 10 ka matlab, exposed group ki 90% bimari exposure ke sar.



**Q70**

PSM

★ Asked directly from our Rank Booster lectures

A study was done in different countries to correlate their fat intake consumption and incidence of heart disease. The result is demonstrated in the image (scatter plot). What study design is represented here?

 Scatter plot of fat consumption versus incidence of heart disease, with each point representing a country.

- A Cross sectional study
- B Case control study
- C Ecological study
- D Cohort study

**Answer** Ecological study

When the unit of analysis is a population or group (countries) rather than individuals, and aggregate exposure is correlated with aggregate outcome, the design is an ecological (correlational) study — the country-level scatter plot is the giveaway. Cross-sectional, case-control and cohort designs all collect exposure and outcome data on individuals; ecological studies are hypothesis-generating and prone to the ecological fallacy.

💡 Jab data point ek DESH hai, insaan nahi — ecological study; fat-intake vs CHD wala scatter iska poster-boy hai.

**Q71**

PSM

As per the demographic cycle, if the Indian population is entering stage four, what will be the primary focus for public health interventions?

- A Maternal care improvement
- B Immunization services expansion
- C Reduce cancer screening
- D Geriatric healthcare improvement and reduce non-communicable diseases

**Answer** Geriatric healthcare improvement and reduce non-communicable diseases

Stage four of the demographic cycle (low stationary phase) has low, balanced birth and death rates, so the population ages and the epidemiological burden shifts from maternal-child and infectious conditions to chronic disease. Public health priorities accordingly move to geriatric care and prevention/control of non-communicable diseases, rather than expanding MCH or immunization services designed for a young, growing population.

💡 Stage 4 = budhaati aabaadi — MCH se focus shift karke geriatrics + NCD control.

**Q72**

PSM

When planning to build a new tertiary hospital, where the time required and specific tasks are largely unknown, which project management technique should be used?

- A PERT
- B Critical Path Method
- C Management by Objectives
- D Gantt charts

**Answer** PERT (Program Evaluation and Review Technique)

PERT is a probabilistic network technique built for projects whose activity durations are uncertain — it uses optimistic, pessimistic and most-likely time estimates. The Critical Path Method is deterministic and assumes known activity times, Gantt charts merely display a schedule of known tasks, and Management by Objectives is a general management philosophy, not a project-scheduling technique.

💡 Time ka pata nahi = PERT; time pakka pata hai = CPM.

**Q73**

PSM

Researchers found a correlation between age and PEFR. It is noted that PEFR increases with age, but the increase is not observed in all. What is the likely type of correlation?

 Scatter diagram of age versus PEFR/FVC showing an overall upward trend with visible scatter of points.

- A Strong positive
- B Weak positive
- C Weak negative
- D Strong negative

**Answer** Weak positive correlation

The two variables rise together, so the correlation is positive; but the wide scatter — different PEFR/FVC values at the same age, with the increase 'not observed in all' — means the points do not hug a straight line, i.e., the correlation coefficient is modestly positive rather than close to +1. A strong positive correlation would require tight clustering of points along the upward line.

 Direction upar + scatter zyada = weak positive — line ki taraf jhukav hai, chipakna nahi.

**Q74**

PSM

★ Asked directly from our Rank Booster lectures

Match the following biomedical waste items with their correct colour-coded disposal bags/containers (placenta, blood-soiled gauze/cotton, IV tubes/catheters/soiled gloves, cytotoxic drug vials including broken ones, syringe with needle, metallic implants).

**Answer** Placenta - yellow; blood-soiled gauze/cotton - yellow; IV tubes, catheters and blood-soiled gloves - red; cytotoxic drugs including broken cytotoxic vials - yellow; syringe with needle - white (sharps container); metallic implants - blue

Under the Biomedical Waste Management Rules 2016 (as amended), yellow bags take human anatomical waste (placenta), soiled blood-contaminated dressings, and discarded/cytotoxic medicines including broken cytotoxic vials; red bags take contaminated recyclable plastics (IV tubes, catheters, gloves); white translucent puncture-proof containers take all sharps including syringes with needles; and blue containers take broken glassware and metallic body implants.

 Body ka maal + dawai yellow, plastic red, sharp white, glass-metal blue — BMW 2016 ka rangmanch.

**Q75**

PSM

★ Asked directly from our Rank Booster lectures

A man residing in Uttarakhand presents with fever. On examination, an eschar is present near the umbilicus (image shown). Which of the following is the most likely diagnosis and causative organism?

 Clinical photograph of a black necrotic eschar with surrounding erythema (near the umbilicus).

- A Scrub typhus and Orientia tsutsugamushi
- B Endemic typhus - rickettsial pox
- C Rocky Mountain spotted fever - R. rickettsiae

**Answer** Scrub typhus caused by Orientia tsutsugamushi

Fever with a painless black necrotic eschar at the bite site in a resident of the Himalayan foothills is classic scrub typhus, caused by Orientia tsutsugamushi and transmitted by larval trombiculid mites (chiggers); treatment is doxycycline. Rocky Mountain spotted fever is a disease of the Americas, and rickettsialpox (R. akari, mouse-mite borne) is not the endemic-typhus pairing offered — the geography and vector settle it.

 Pahadi ilaaka + bukhar + kala eschar = scrub typhus — mite ke kaatne ki cigarette-burn nishani.

**Q76**

PSM

A family with two children (a female aged 9 years 10 months and a male aged 10 months) lives in a single living room of 184 square feet. Based on which criteria can overcrowding be assessed?

- A Persons per room
- B Sex separation
- C Floor space

**Answer** All three criteria — persons per room, floor space, and sex separation — indicate overcrowding here

By Park's counting rules the infant under 1 year is not counted and the 9-year-10-month child counts as half a unit, giving  $2 + 0.5 = 2.5$  units in one room — exceeding the 2-persons-per-room standard. A room of 184 sq ft ( $>110$  sq ft) accommodates only 2 persons by the floor-space standard, again exceeded by 2.5 units. And the girl, being over 9 years, sharing a sleeping room with an opposite-sex person who is not her spouse violates the sex-separation criterion — so all three standards are breached.

💡 Overcrowding ke teen taraazu — persons/room, floor space, sex separation; aur ginti mein  $<1$  saal wala zero, 1-10 saal wala aadha.

**Q77**

PSM

An image showed concentric circles illustrating the Rule of Halves for hypertension, with labelled/coloured segments from the whole community down to adequately treated patients. Identify the labelled segments.

 Concentric circle diagram of the Rule of Halves for hypertension, with coloured nested circles from the whole community down to adequately treated hypertensives (9 labelled segments).

- A Whole community
- B Normotensive subjects
- C Hypertensive subjects
- D Undiagnosed hypertension
- E Diagnosed hypertension
- F Diagnosed but untreated
- G Diagnosed and treated
- H Inadequately treated
- I Adequately treated

**Answer** Rule of Halves cascade — outermost to innermost: whole community → normotensive vs hypertensive subjects → undiagnosed vs diagnosed hypertension → diagnosed-untreated vs diagnosed-treated → inadequately vs adequately treated; the innermost segment is the adequately treated group

The Rule of Halves states that only about half of hypertensives in the community are aware/diagnosed, half of those diagnosed are on treatment, and half of those treated are adequately controlled — so the nested circles shrink from the whole community to a small innermost core of adequately treated patients. Whichever segment the paper asked about is read off this cascade; the innermost circle (the segment most sources recall being asked) is 'adequately treated'.

💡 Rule of Halves: har step pe aadhe kho jaate hain — diagnose, treat, control; centre mein bachta hai sirf adequately-treated ka chhota sa gola.

**Q78**

PSM

A patient with pulmonary tuberculosis has resistance to rifampicin, isoniazid, levofloxacin, and bedaquiline. What is the correct classification?

- A** MDR or RR TB with H+R and fluoroquinolone resistance
- B** MDR or RR TB with resistance to INH, rifampicin, quinolone, and either bedaquiline or linezolid

**Answer** **XDR-TB — MDR/RR-TB with additional resistance to any fluoroquinolone plus at least one Group A drug (bedaquiline or linezolid)**

By the WHO 2021 revised definitions, MDR-TB is resistance to isoniazid and rifampicin; adding any fluoroquinolone makes it pre-XDR-TB; and adding at least one further Group A drug (bedaquiline or linezolid) makes it XDR-TB. This patient is resistant to H + R + levofloxacin + bedaquiline, which meets the full XDR-TB definition — the first option describes only pre-XDR-TB and is the trap.

💡 MDR + quinolone = pre-XDR; usme bedaquiline ya linezolid bhi gaya = XDR (WHO 2021).

**Q79**

PSM

A lecturer divided a large group of people into smaller groups after the lecture to make them participate actively. What teaching method/approach is this?

- A** Active participation
- B** One to one communication

**Answer** **Active participation (small-group discussion — the classic Park term for splitting a large lecture audience into small discussion groups is the 'buzz group' technique)**

Breaking a large, passive lecture audience into small groups so that shy members can speak is a group-teaching approach designed to promote active participation; it is emphatically not one-to-one communication. In Park's health-communication chapter this specific manoeuvre is named the 'buzz group' technique — if that word appeared among the real options it would be the best answer; among the recalled options, 'active participation' is correct.

💡 Bada lecture → chhote bolne wale groups = buzz group; maqsad ek hi — active participation.

**Q80**

PSM

Regarding the stages of the family life cycle, what defines the contraction phase?

**Answer** **The contraction phase begins when the first child leaves home and ends when the last child leaves home**

The family life cycle runs: formation (marriage) → extension (birth of first child to birth of last child) → completed extension → contraction (first child leaves home to last child leaves home) → completed contraction (empty nest) → dissolution (death of a spouse). The contraction phase is therefore bracketed by the departures of the first and last children.

💡 Extension = bachche aana shuru se aakhri tak; contraction = bachche jaana shuru se aakhri tak.

**Q81**

PSM

A disease presents with respiratory and CNS symptoms; 90-95% of infections are subclinical and only 1-2% show clinical symptoms (graph shown). Identify the disease.

 Graph/table of the clinical spectrum of infection showing subclinical (90-95%) versus clinical (1-2%) proportions.

- A** Polio
- B** JE
- C** Yellow fever

**Answer** **Polio**

The classic iceberg of poliovirus infection (Park) is about 90-95% inapparent/subclinical infection, 4-8% abortive minor illness, 1-2% non-paralytic (CNS) disease, and under 1% paralytic polio — with respiratory involvement in bulbar disease. Japanese encephalitis is the tempting distractor, but its subclinical ratio is far more extreme (only about 1 in 250-1,000 infections becomes symptomatic), so the quoted 90-95%/1-2% split is polio's signature breakdown.

 90-95% subclinical, 1-2% clinical — yeh polio ka iceberg hai; JE mein to iceberg aur bhi doob jaata hai (1 in 250-1000).

## 09 Medicine

19 recalled questions

### Q82 MEDICINE

★ Asked directly from our Rank Booster lectures

A patient has a pituitary adenoma and a pancreatic neuroendocrine lesion on imaging, along with hypercalcemia. Which of the following statements is most appropriate regarding the underlying syndrome?

 MRI showing a pituitary adenoma and abdominal CT showing a pancreatic lesion were reportedly displayed.

- A It is associated with a mutation on chromosome 11
- B The patient should be evaluated for medullary carcinoma of the thyroid
- C The patient should be evaluated for a RET proto-oncogene mutation
- D The pancreatic lesion is associated with von Hippel-Lindau syndrome

**Answer** It is associated with a mutation on chromosome 11 (MEN1 / menin gene)

The triad of parathyroid (hypercalcemia), pancreatic, and pituitary involvement is MEN1 (Wermer syndrome), caused by inactivating mutations of the MEN1 gene encoding menin on chromosome 11q13. Medullary thyroid carcinoma and RET proto-oncogene mutations belong to MEN2, and von Hippel-Lindau is a separate syndrome (chromosome 3) — so those options are the classic cross-syndrome traps.

 3 Ps (parathyroid, pancreas, pituitary) = MEN1 = menin, chromosome 11; RET aur MTC MEN2 ke hain.

### Q83 MEDICINE

★ Asked directly from our Rank Booster lectures

Anti-mitochondrial antibodies (AMA) are most characteristically seen in which condition?

- A Primary biliary cholangitis (PBC)
- B Primary sclerosing cholangitis (PSC)
- C Autoimmune hepatitis
- D Wilson's disease

**Answer** Primary biliary cholangitis (PBC)

AMA, directed against the E2 component of the pyruvate dehydrogenase complex, is positive in about 95% of PBC and is its most sensitive and specific serological marker; the typical patient is a middle-aged woman with pruritus, fatigue, and a raised ALP. PSC is instead associated with ulcerative colitis and atypical p-ANCA, and autoimmune hepatitis with anti-smooth muscle or anti-LKM antibodies.

 Middle-aged woman, itching, high ALP, AMA positive — PBC, aankh band karke.

**Q84** MEDICINE

A patient presents with recurrent episodes of angioedema. There is a history of similar episodes in family members, suggesting a hereditary condition. Which of the following is the most likely underlying defect?

 Clinical photographs of lip/ facial angioedema and hand swelling.

- A Deficiency of C1 esterase inhibitor
- B Deficiency of C3
- C Deficiency of C5
- D Deficiency of factor H

**Answer** Deficiency of C1 esterase inhibitor

Recurrent angioedema without urticaria plus a positive family history is hereditary angioedema, an autosomal dominant deficiency (or dysfunction) of C1 esterase inhibitor causing unchecked kallikrein activity and bradykinin-mediated swelling of the face, extremities, gut (abdominal pain), and airway; low C4 is the screening clue. Factor H deficiency causes atypical HUS, not angioedema — and note that HAE is bradykinin-mediated, so antihistamines and steroids do not work.

💡 Family history + swelling without itching/urticaria = C1 esterase inhibitor deficiency — bradykinin ka khel, histamine ka nahi.

**Q85** MEDICINE

★ Asked directly from our Rank Booster lectures

A 40-year-old patient presents with recurrent painful oral ulcers and a recent episode of genital ulceration. Images of both the oral and genital ulcers are provided. What is the most likely diagnosis?

 Clinical photographs showing an aphthous oral ulcer, scrotal/vulvar ulcers, and erythema nodosum on the leg.

- A Behçet disease
- B IgA vasculitis
- C Takayasu arteritis
- D Systemic lupus erythematosus

**Answer** Behçet disease

Recurrent oral aphthae plus genital ulcers (which characteristically scar), with skin lesions such as erythema nodosum and possible uveitis, define Behçet disease — a variable-vessel vasculitis with pathergy positivity, common along the old Silk Route. SLE causes oral ulcers that are typically painless and lacks the scarring genital ulcers, which is why it is the closest distractor.

💡 Mouth + genital ulcers + red shins (erythema nodosum) = Behçet — genital ulcer scar chhod ke jaata hai.

**Q86** MEDICINE

★ Asked directly from our Rank Booster lectures

A pregnant woman develops dyspnea 3 hours after receiving a blood transfusion. She has bilateral pulmonary infiltrates and non-cardiogenic pulmonary edema; renal function is normal. What is the most likely diagnosis?

- A TRALI
- B TACO (Transfusion-Associated Circulatory Overload)
- C Fluid overload

**Answer** TRALI (Transfusion-Related Acute Lung Injury)

Acute hypoxemia with bilateral infiltrates and non-cardiogenic pulmonary edema within 6 hours of transfusion defines TRALI, most often caused by donor anti-HLA/anti-neutrophil antibodies priming recipient neutrophils in the lung. TACO, the key distractor, is hydrostatic (cardiogenic) edema from volume overload — with raised BNP, hypertension, and response to diuretics — and the stem explicitly says the edema is non-cardiogenic.

💡 Transfusion ke 6 ghante mein white-out lungs, non-cardiogenic = TRALI; overload ho toh TACO.

**Q87** MEDICINE

★ Asked directly from our Rank Booster lectures

A patient presents with the pentad of TTP — fever, neurological deficits, thrombocytopenia, renal impairment, and schistocytes on peripheral smear. What is the management?

- A Plasma exchange
- B Steroids
- C Eculizumab
- D Fresh frozen plasma (FFP)

**Answer** Plasma exchange (plasmapheresis)

TTP is caused by autoantibody-mediated ADAMTS13 deficiency leaving ultra-large vWF multimers that shred RBCs into schistocytes; urgent therapeutic plasma exchange both removes the antibody and multimers and replaces ADAMTS13, cutting mortality from about 90% to under 20%. Plain FFP infusion only supplies enzyme without removing antibody and is merely a bridge when exchange is unavailable, eculizumab is for atypical HUS, and platelet transfusion is relatively contraindicated.

💡 TTP dikhte hi plasma exchange — FFP infusion adhoora ilaaj hai, aur platelets toh aag mein ghee.

**Q88** MEDICINE

A cancer patient with chronic kidney disease develops markedly elevated uric acid in the setting of tumor lysis syndrome after chemotherapy. Which is the drug of choice for the hyperuricemia?

- A Rasburicase
- B Allopurinol
- C Febuxostat

**Answer** Rasburicase

Rasburicase is recombinant urate oxidase that rapidly degrades already-formed uric acid into the far more soluble allantoin, making it the drug of choice for established hyperuricemia in tumor lysis syndrome, especially with renal impairment. Allopurinol and febuxostat only block new urate synthesis via xanthine oxidase — useful for prophylaxis but useless against the uric acid already circulating; remember to rule out G6PD deficiency before rasburicase.

💡 TLS mein uric acid ban chuka hai — usse todne wala rasburicase hi kaam aayega, allopurinol sirf aage banna rokta hai.

**Q89** MEDICINE

A smoker is diagnosed with limited-stage small cell lung cancer with a good performance status. What is the preferred induction management?

- A Surgery followed by radiotherapy
- B Chemoradiation
- C Chemotherapy plus targeted therapy
- D Thoracic cytoreduction followed by chemotherapy
- E Concurrent radio plus chemo

**Answer** Concurrent chemoradiation (platinum-etoposide with concurrent thoracic radiotherapy)

Limited-stage SCLC in a fit patient is treated with concurrent platinum-etoposide chemotherapy plus thoracic radiotherapy, followed by consideration of prophylactic cranial irradiation — concurrent delivery outperforms sequential (NCCN/ESMO). Surgery has a role only in the rare T1-2N0 tumor, and SCLC has no approved oncogene-targeted therapy, so 'chemotherapy plus targeted therapy' is a trap.

💡 Small cell = systemic disease from day one — cut nahi, concurrent chemo-RT karo.



**Q90**

MEDICINE

★ Asked directly from our Rank Booster lectures

A patient with acromegaly is receiving octreotide but shows no significant improvement, and IGF-1 levels remain elevated. Which drug should be added to the treatment regimen?

- A GnRH
- B Relaxin
- C Pegvisomant
- D Leuprolide

**Answer Pegvisomant**

Pegvisomant is a growth hormone receptor antagonist that blocks GH action at the liver, normalizing IGF-1 in over 90% of patients, and is the standard add-on or switch for acromegaly resistant to somatostatin receptor ligands like octreotide. GnRH analogues and leuprolide act on the gonadotropin axis and have no role in GH excess.

💡 Octreotide fail in acromegaly? Block the receptor itself — pegvisomant, IGF-1 se monitor karo (GH nahi).

**Q91**

MEDICINE

★ Asked directly from our Rank Booster lectures

A 30-year-old man presents with fever, fatigue, arthralgia, and an expanding erythematous skin lesion with central clearing (erythema migrans) after a trekking trip in a tick-endemic area. An image of the target lesion was shown. What is the first-line drug treatment?

 Clinical photograph of erythema migrans — an expanding annular erythematous 'bull's-eye/target' lesion with central clearing.

- A Doxycycline
- B Azithromycin
- C Linezolid
- D Penicillin

**Answer Doxycycline**

Erythema migrans after a trek in tick country is early Lyme disease (*Borrelia burgdorferi*), and oral doxycycline is the first-line treatment (IDSA guidance; also covers possible co-infection with *Anaplasma*). Ceftriaxone is reserved for neuroborreliosis or Lyme carditis with high-grade block, and amoxicillin/azithromycin are alternatives when doxycycline cannot be used — linezolid has no role.

💡 Trek + tick + target lesion = Lyme — doxycycline first, ceftriaxone sirf heart/brain involvement pe.

**Q92**

MEDICINE

A patient with severe aplastic anemia is not eligible for stem cell transplantation. Which combination represents the most appropriate medical treatment strategy?

- A Only eltrombopag
- B Eltrombopag with cyclosporine
- C Blood transfusion
- D ATG with cyclosporine with eltrombopag

**Answer ATG with cyclosporine with eltrombopag**

For severe aplastic anemia when allogeneic transplant is not possible, the current standard is triple therapy: horse antithymocyte globulin plus cyclosporine (immunosuppression against the T-cell attack on marrow) with upfront eltrombopag (a thrombopoietin receptor agonist that stimulates residual stem cells) — the RACE trial (NEJM 2022) showed markedly higher complete responses with the triple combination, now reflected in Harrison's and BSH guidance. Blood transfusion is purely supportive, and eltrombopag alone is inadequate first-line.

💡 Severe aplastic anemia, no donor: ATG + cyclosporine + eltrombopag — teeno saath, RACE trial ka standard.

**Q93**

MEDICINE

★ Asked directly from our Rank Booster lectures

A patient has low-grade fever and headache for 2 weeks with irritability, neck rigidity and papilledema. Lumbar puncture shows increased mononuclear cells, low sugar and increased proteins. CECT head shows increased basal meningeal enhancement and obstructive hydrocephalus. What is the diagnosis?

 Axial contrast-enhanced CT brain images showing basal meningeal enhancement and dilated ventricles (obstructive hydrocephalus).

- A TBM
- B Viral meningitis
- C Bacterial meningitis
- D Cryptococcal meningitis

**Answer** TBM (tuberculous meningitis)

A subacute (2-week) meningitis with lymphocytic CSF, high protein and low glucose, plus basal meningeal enhancement, hydrocephalus, and vasculitic infarcts on contrast CT is the classic imaging-clinical triad of tuberculous meningitis, especially in India. Viral meningitis has normal glucose and a benign course, pyogenic meningitis is hyperacute with neutrophilic CSF, and cryptococcal meningitis typically occurs in the immunosuppressed with a positive India ink/antigen.

 2 hafte ka bukhar-sardard + basal enhancement + hydrocephalus = TBM until proven otherwise.

**Q94**

MEDICINE

A 40-year-old hypertensive patient presents with hypokalemia and metabolic alkalosis. The aldosterone-renin ratio is high and the saline loading (confirmatory) test is positive. What is the next best step to localize the source?

- A CT abdomen
- B CT guided biopsy
- C MRI abdomen
- D MIBG scan
- E Adrenal vein sampling

**Answer** Adrenal vein sampling

With primary hyperaldosteronism confirmed, the decisive step before surgery is lateralization, and adrenal vein sampling is the gold standard because CT can both miss microadenomas and mislabel non-functioning incidentalomas — per the Endocrine Society guideline, patients over 35 need AVS even when CT shows an adenoma. CT-guided biopsy has no role in aldosteronoma, and MIBG is for pheochromocytoma.

 Conn confirmed? CT dekh lo, par surgery se pehle faisla adrenal vein sampling hi karta hai.

**Q95**

MEDICINE

★ Asked directly from our Rank Booster lectures

A patient presents with low serum calcium and high parathyroid hormone (PTH) levels. What is the most likely diagnosis?

- A Pseudo-hypoparathyroidism
- B Hypoparathyroidism
- C Pseudo-pseudo-hypoparathyroidism

**Answer** Pseudo-hypoparathyroidism

Hypocalcemia with an appropriately elevated PTH indicates end-organ resistance to PTH — pseudohypoparathyroidism (GNAS mutation; type 1a adds Albright hereditary osteodystrophy: short stature, round face, short 4th/5th metacarpals). In true hypoparathyroidism PTH is low, and in pseudo-pseudohypoparathyroidism the AHO phenotype is present but calcium, phosphate and PTH are all normal.

 Low Ca + high PTH = hormone hai par sunta koi nahi — pseudohypoparathyroidism (receptor resistance).

**Q96**

MEDICINE

★ Asked directly from our Rank Booster lectures

A patient presents with tearing chest pain, and examination shows unequal blood pressure in the left and right arms. Chest X-ray shows a widened mediastinum. Which is the best modality for diagnosis?

 Chest X-ray demonstrating a widened mediastinum was part of the vignette.

- A CTA
- B TTE Echo
- C ECG
- D Cardiac biomarkers

**Answer** CTA (CT angiography)

Tearing chest pain with interarm BP discrepancy and a widened mediastinum is acute aortic dissection, and CT angiography is the investigation of choice — rapid, widely available, and able to define the flap, extent and branch involvement for classification and surgical planning. Transthoracic echo is insensitive for the descending aorta (TEE is the alternative in unstable patients), and ECG/troponin chiefly serve to exclude the MI mimic.

 Tearing pain + unequal arm BP + wide mediastinum = dissection — seedha CT angio, echo pe time waste nahi.

**Q97**

MEDICINE

★ Asked directly from our Rank Booster lectures

A patient develops progressive, symmetric, ascending weakness (starting in the legs, then upper limbs) with intact sensation, 3 weeks after an episode of diarrhea. What CSF finding is characteristic of this condition?

- A High protein with normal cell count
- B Low glucose

**Answer** High protein with normal cell count (albuminocytological dissociation)

Ascending flaccid weakness after *Campylobacter jejuni* diarrhea is Guillain-Barré syndrome, whose CSF hallmark is albuminocytological dissociation — elevated protein with a normal cell count, best seen after the first week. Low CSF glucose indicates infective (bacterial/tubercular) meningitis and has no place in GBS, an immune-mediated polyradiculoneuropathy, not an infection of the CSF.

 GBS ka CSF: protein upar, cells normal — albuminocytological dissociation, glucose ko haath nahi lagta.

**Q98****MEDICINE**

A patient presents with flaccid paralysis of all four limbs and areflexia without sensory loss, following a recent infection (URI/UTI). A strength-duration curve is shown with increased chronaxie and decreased excitability. Identify the condition.

 *Strength-duration curve plotting stimulus strength versus duration: normal curve with rheobase marked, and an abnormal curve shifted upward/rightward indicating increased chronaxie (denervation).*

- A** GBS
- B** Lambert-Eaton
- C** Myasthenia
- D** AMAN

**Answer** **AMAN (acute motor axonal neuropathy)**

A pure motor post-infectious flaccid quadriparesis with areflexia is within the GBS spectrum, but a strength-duration curve shifted up and right (increased chronaxie, reduced excitability) signifies axonal degeneration/denervation of muscle — pointing specifically to the axonal variant, AMAN (anti-GM1/GD1a antibodies, often post-Campylobacter), rather than classic demyelinating GBS where the axon is initially spared. Myasthenia and LEMS are neuromuscular junction disorders with normal nerve excitability curves and no areflexia-with-denervation pattern.

 Denervation-type strength-duration curve in a GBS-like patient = axon mara hai — AMAN, not plain GBS.

**Q99****MEDICINE****★ Asked directly from our Rank Booster lectures**

A patient with hypokalemia (with QT prolongation and ST depression on ECG) fails to improve despite correction with potassium chloride. What underlying electrolyte abnormality should be suspected?

 *ECG reportedly showing hypokalemia changes (ST depression, QT/QU prolongation).*

- A** Hypomagnesemia
- B** Hypermagnesemia

**Answer** **Hypomagnesemia**

Hypokalemia refractory to KCl replacement is caused by coexisting hypomagnesemia: magnesium normally inhibits ROMK channels in the distal nephron, so when magnesium is low the kidney keeps wasting potassium no matter how much is infused — magnesium must be replaced first. Hypermagnesemia does the opposite (retains potassium) and is the giveaway wrong option.

 Potassium chadha rahe ho par level nahi chadh raha? Pehle magnesium bharo.

**Q100**

MEDICINE

A 22-year-old woman presents with a six-month history of recurrent severe headache partially relieved by analgesics. BP is 210/120 mmHg, HR 100/min. CT angiography shows a beaded appearance of the renal artery (fibromuscular dysplasia). What is the best management?

 CT angiography showing the classic beaded ('string of beads') appearance of the renal artery, diagnostic of fibromuscular dysplasia.

- A** Renal artery stenting
- B** ACEI/ARB
- C** Percutaneous Transluminal balloon angioplasty
- D** Nephrectomy

**Answer** **Percutaneous transluminal balloon angioplasty (without stenting)**

Renovascular hypertension in a young woman with the classic 'string of beads' renal artery is fibromuscular dysplasia, and its treatment of choice is percutaneous transluminal balloon angioplasty alone, which is usually curative; stents are reserved for angioplasty failure, dissection, or atherosclerotic (ostial) renal artery stenosis. ACE inhibitors help control BP but do not treat the stenosis, and nephrectomy has no role in a salvageable kidney.

 Young lady + string of beads = FMD — balloon se phoolao, stent ki zaroorat nahi.

**50%+ of this paper was asked directly from our Rank Booster lectures.**

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## 10 Surgery

16 recalled questions

### Q101 SURGERY

A patient, 5 days post-laparotomy, develops about 50 ml of fecal discharge from the main wound. The patient is stable and tolerating oral fluids. What is the preferred management?

- A avoid laparotomy
- B don't go for ideal resection
- C stop oral intake and start TPN
- D maintain nutrition

**Answer** Conservative management — stop oral intake and start total parenteral nutrition (low-output enterocutaneous fistula)

A 50 ml/day fecal discharge in a stable patient is a low-output enterocutaneous fistula; the majority close spontaneously with conservative care — bowel rest/nutritional support (TPN), fluid-electrolyte correction, skin protection, and sepsis control. Early re-exploration at day 5, through an inflamed, hostile abdomen with no peritonitis, is contraindicated; surgery is reserved for failure to close after weeks, high output, distal obstruction, or sepsis.

💡 Low-output fistula + stable patient = SNAP (Sepsis, Nutrition, Anatomy, Procedure later) — chaaku last me aata hai.

### Q102 SURGERY

★ Asked directly from our Rank Booster lectures

A female patient with primary sclerosing cholangitis and ulcerative colitis has a 2.5 cm gallbladder polyp. Which of the following is true regarding cholecystectomy?

**Answer** Cholecystectomy is indicated — in PSC the size threshold for operating on a gallbladder polyp is lowered (about 5-8 mm), and a 2.5 cm polyp mandates cholecystectomy in any case

Gallbladder polyps in primary sclerosing cholangitis carry a much higher malignancy risk, so guidelines lower the cholecystectomy threshold from the usual 10 mm to roughly 5-8 mm (some advise removal of any polyp in PSC if the patient is fit). Independently, a polyp of 2.5 cm (>18-20 mm) has a high probability of carcinoma and is a firm indication for cholecystectomy — both the size criterion and the lowered PSC threshold point to the same answer.

💡 PSC me gallbladder polyp ka benefit-of-doubt khatam — 5 mm bhi ho toh nikaalne ki sochte hain, 2.5 cm toh pakka OT.

**Q103** SURGERY

During laparoscopic cholecystectomy, immediately after trocar insertion, the patient develops sudden hypotension (BP 70/40) and tachycardia, and the view through the port is obscured by a rapidly collecting pool of blood. What is the next step in management?

- A** Remove trocar
- B** Don't remove
- C** Increased insufflation pressure
- D** Exploratory laparotomy with trocar in situ

**Answer** Immediate exploratory laparotomy with the trocar left in situ

Sudden hemodynamic collapse with pooling blood after trocar entry signals a major retroperitoneal vessel injury (aorta, IVC, iliacs). The trocar must not be removed — it partially tamponades the hole and marks the injury track — and the correct response is immediate conversion to open laparotomy for proximal/distal vascular control. Raising insufflation pressure would worsen venous injury and risks gas embolism.

💡 Trocar se bada vessel chhida toh trocar hi cork hai — usse chhedo mat, pet kholo.

**Q104** SURGERY

A child presents with a ventrally placed ectopic urethral opening with ventral curvature of the penis (hypospadias). What is the appropriate management?

- A** Immediate circumcision
- B** Prolonged catheterization
- C** Observation
- D** Repair the defect

**Answer** Repair the defect (urethroplasty) — circumcision must NOT be done

Hypospadias is managed by surgical repair (urethroplasty with chordee correction), ideally completed between about 6 and 18 months of age. Circumcision is contraindicated beforehand because the dorsal preputial skin is the main flap/graft material used for the reconstruction — the classic trap option.

💡 Hypospadias me foreskin hi spare part hai — pehle circumcision kiya toh repair ka kapda hi kaat diya.

**Q105** SURGERY

★ Asked directly from our Rank Booster lectures

An image shows necrotizing fasciitis of the perineum. What is the diagnosis?

 Clinical photograph of necrotizing fasciitis involving the perineum/genital region.

**Answer** Fournier's gangrene

Fournier's gangrene is rapidly progressive polymicrobial necrotizing fasciitis of the perineum, genitalia, and perianal region, typically in diabetic or immunocompromised men. It is a surgical emergency treated with aggressive serial debridement plus broad-spectrum antibiotics.

💡 Perineum ki necrotizing fasciitis ka naam hi Fournier's gangrene hai — naam yaad, chaaku taiyaar.

**Q106** SURGERY

On the Cardiff-Lucknow nodularity scale (grading of breast nodularity in benign breast disease), what score corresponds to extensive nodularity involving most of the breast, but not the entire breast?

**Answer** **Score 3**

The Lucknow-Cardiff scale grades breast nodularity from 0 (none) to 4 (nodularity involving the whole breast); extensive nodularity involving most but not all of the breast corresponds to grade 3. A deliberately obscure question — but the answer is consistent with the scale's structure.

💡 0 se 4 ka ladder: 'poora breast' = 4, 'zyada-tar breast' = 3 — one step short of total.

**Q107** SURGERY

A patient with painless progressive jaundice and weight loss has perihilar cholangiocarcinoma. Imaging shows the tumor extending up to and involving the confluence of the right and left hepatic ducts, without extension into the secondary intrahepatic ducts. What is the Bismuth-Corlette type?

**Answer** **Bismuth-Corlette Type II**

In the Bismuth-Corlette classification of perihilar cholangiocarcinoma (Klatskin tumor): Type I is confined to the common hepatic duct below the confluence, Type II reaches and involves the confluence but not the secondary ducts, Types IIIa/IIIb extend into the right/left secondary ducts respectively, and Type IV involves both sides or is multifocal. A tumor reaching the confluence without extension into either hepatic ductal system is Type II.

💡 Klatskin ka hisaab: confluence tak = II, ek taraf ghusa = III, dono taraf = IV.

**Q108** SURGERY

What is the purpose of making slits (meshing) in a split-thickness skin graft?

- A exudation
- B surface area matching
- C improve conformance

**Answer** **To allow drainage (exudation) of fluid, preventing seroma/hematoma collection beneath the graft**

Meshing serves three purposes — egress of blood and exudate so no collection lifts the graft off its bed, expansion of graft surface area, and better conformance to irregular contours — but the primary reason grafts fail is fluid collecting underneath, so drainage/exudation is the best single answer as recalled. This matches the source's answer.

💡 Graft ke neeche paani jama = graft mara; mesh ke chhed usi paani ke escape routes hain.

**Q109** SURGERY

★ Asked directly from our Rank Booster lectures

During thyroidectomy, the surgeon ligated the superior thyroid artery away from the upper pole of the gland. Post-operatively the patient (a singer) is unable to sing high-pitched notes. What is the cause?

**Answer** **Injury to the external branch of the superior laryngeal nerve (paralysis of the cricothyroid muscle)**

The external branch of the superior laryngeal nerve runs close to the superior thyroid artery pedicle; ligating the artery away from (high above) the upper pole catches the nerve, paralyzing the cricothyroid — the vocal cord tensor — so the voice loses its high pitch. This is why the superior thyroid artery is ligated as close to the gland as possible (the recurrent laryngeal nerve, by contrast, dictates ligating the inferior thyroid artery away from the gland).

💡 Superior pedicle ko gland ke paas baandho, warna singer ka 'sur' — external laryngeal nerve — chala jayega.



**Q110** SURGERY

★ Asked directly from our Rank Booster lectures

Calculate the GCS score for a patient who speaks inappropriate words, does not open eyes to painful stimulus, and withdraws to pain.

**Answer** **GCS = 8 (E1 + V3 + M4)**

No eye opening even to pain scores E1, inappropriate words score V3, and withdrawal from pain scores M4, totalling 8. A GCS of 8 or less defines severe head injury/coma and is the classic threshold for considering definitive airway protection ('GCS 8 — intubate').

💡 E1 V3 M4 = 8 — aur 8 ya usse kam matlab tube ready rakho.

**Q111** SURGERY

A patient with renal cell carcinoma presents with a suspicious (right-sided / non-reducible) varicocele. What is the most likely reason for the varicocele?

- A testis involvement
- B RCC with IVC involvement
- C ureter compression
- D lymphatic obstruction

**Answer** **RCC with venous (renal vein / IVC) involvement — tumor thrombus obstructing gonadal venous drainage**

RCC has a strong propensity to invade the renal vein and propagate into the IVC; the right gonadal vein drains directly into the IVC (the left drains into the left renal vein), so a new right-sided or non-collapsing varicocele signals venous obstruction by tumor thrombus. Both sources agree; the varicocele is a red-flag sign, not from lymphatics or ureteric compression.

💡 Right-sided ya khada rehne par bhi na girne wala varicocele = kidney/IVC me kuch pak raha hai — scan karo.

**Q112** SURGERY

★ Asked directly from our Rank Booster lectures

A 34-year-old man with untreated enteric fever (third week of illness) develops sudden severe abdominal pain, distension, and features of peritonitis. X-ray shows free gas under the right dome of the diaphragm. What is the diagnosis/cause?

📷 Erect abdominal/chest X-ray showing free gas under the right dome of the diaphragm (pneumoperitoneum).

**Answer** **Ileal perforation from necrosis/ulceration of Peyer's patches, causing perforation peritonitis — treated by emergency laparotomy**

In the third week of untreated typhoid, hyperplastic Peyer's patches in the terminal ileum ulcerate and necrose, perforating the antimesenteric border; free gas under the diaphragm confirms hollow-viscus perforation. The sources' answers (diagnosis: perforation peritonitis; cause: Peyer's patch necrosis; treatment: surgery) are complementary parts of the same picture, not a dispute — management is resuscitation plus emergency laparotomy with repair/resection.

💡 Typhoid ka teesra hafta = Peyer's patch phatne ka hafta; gas under diaphragm dikhi toh seedha OT.

**Q113** SURGERY

What is the management of Barrett's esophagus with high-grade dysplasia?

- A** endoscopic mucosal resection
- B** radiofrequency ablation

**Answer** **Endoscopic eradication therapy — radiofrequency ablation for flat high-grade dysplasia (endoscopic mucosal resection is used when there is a visible/nodular lesion)**

Current guidelines (ACG 2022) recommend endoscopic eradication over surveillance or esophagectomy for HGD: any visible or nodular lesion is first removed by EMR (which also stages depth), and the remaining flat Barrett's segment is ablated with RFA. For a stem that mentions no nodule — as recalled here — RFA is the preferred single answer; EMR alone is correct only when a discrete visible lesion is described.

💡 Barrett + HGD: nodule dikhe toh kaato (EMR), flat ho toh jalao (RFA) — surgery ab last resort hai.

**Q114** SURGERY

★ Asked directly from our Rank Booster lectures

An elderly man has lower urinary tract symptoms. Uroflowmetry shows a maximum urine flow rate of 8 ml/second and a post-void residual volume of 275 ml. What is the diagnosis?

**Answer** **Bladder outlet obstruction (most commonly due to benign prostatic hyperplasia)**

A Qmax below 10 ml/s on uroflowmetry strongly suggests bladder outlet obstruction (normal >15 ml/s), and a post-void residual of 275 ml confirms significantly incomplete emptying. In an elderly male, BPH is the overwhelmingly likely cause; urodynamics can distinguish obstruction from detrusor underactivity if needed.

💡 Qmax < 10 + PVR badhaa hua in an old man = prostate ka traffic jam.

**Q115** SURGERY

An image showed a surgeon using a diathermy setup (hand-held electrode, with a return/grounding pad on the patient). What type of diathermy is this?

 *Diagram of a monopolar diathermy setup: active electrode in the surgeon's hand, return electrode (ground pad) on the patient's thigh, and a foot pedal with coagulation (blue) and cutting (yellow) pedals.*

- A** Monopolar
- B** Bipolar

**Answer** **Monopolar diathermy**

In monopolar diathermy the current passes from a pencil-like active electrode through the patient to a large return electrode (grounding pad) on the thigh — the pad in the image is the giveaway. Bipolar diathermy uses forceps-like instruments where current passes only between the two tips, needing no patient plate (safer with pacemakers).

💡 Pad laga hai toh monopolar, forceps hai toh bipolar — plate hi pehchaan hai.

**Q116** SURGERY

What is the management for toxic megacolon?

**Answer** Intensive medical therapy first (NPO, decompression, IV fluids, IV steroids if IBD, broad-spectrum antibiotics); urgent subtotal colectomy with end ileostomy if there is perforation or no improvement within 48-72 hours

Toxic megacolon (usually complicating ulcerative colitis or C. difficile colitis) is initially managed medically with bowel rest, nasogastric decompression, aggressive resuscitation, IV corticosteroids for IBD, and antibiotics; surgery — subtotal colectomy with end ileostomy — is required urgently for perforation, massive hemorrhage, or failure to improve within 48-72 hours. 'Urgent colectomy' reflects the surgical endpoint; if the exam options contrasted medical vs surgical, colectomy is the definitive treatment.

💡 Toxic megacolon: 48-72 ghante ki mohlat medical management ko — sudhre nahi ya phate, toh subtotal colectomy.

## 11 OBG

17 recalled questions

### Q117 OBG

A hysterectomy specimen showed a fibroid that was submucosal and distorting the uterine cavity, with the greater part of it lying intramurally. What is its FIGO type?

 Gross hysterectomy specimen (cut section) showing a submucosal fibroid distorting the uterine cavity, shown beside the FIGO type 0-8 classification diagram.

**Answer** FIGO Type 2

In the FIGO leiomyoma classification, Type 0 is entirely intracavitary (pedunculated), Type 1 is submucosal with less than 50% intramural extension, and Type 2 is submucosal with 50% or more of the fibroid intramural while still distorting the cavity. A largely intramural fibroid that indents the endometrial cavity is therefore Type 2; Type 3 abuts the endometrium without cavity distortion.

💡 Cavity distort + zyada hissa wall ke andar = FIGO Type 2 — the 50% rule decides 1 vs 2.

### Q118 OBG

★ Asked directly from our Rank Booster lectures

A gross image of a hysterectomy specimen was shown with a clinical profile of heavy menstrual bleeding and dysmenorrhea. The uterus was diffusely and symmetrically enlarged. Identify the condition.

 Gross photographs of a hysterectomy specimen: symmetrically enlarged uterus with thickened, trabeculated myometrium on cut section, consistent with adenomyosis.

**Answer** Adenomyosis

Adenomyosis — endometrial glands and stroma within the myometrium — classically presents with heavy menstrual bleeding and progressive dysmenorrhea in a diffusely, symmetrically enlarged 'globular' uterus; the cut surface shows a thickened, trabeculated myometrium, sometimes with tiny hemorrhagic foci. A fibroid uterus, the key distractor, is typically asymmetrically enlarged with discrete whorled nodules.

💡 Symmetric globular uterus + painful heavy periods = adenomyosis; asymmetric lumpy uterus = fibroid.

### Q119 OBG

★ Asked directly from our Rank Booster lectures

A 42-year-old woman has heavy menstrual bleeding due to a 3 cm intramural fibroid that does not distort the uterine cavity. If hysterectomy is not performed, what is the best alternative (medical) management?

- A Copper IUD
- B Danazol
- C LNG IUD
- D No treatment (if asymptomatic)

**Answer** LNG-IUS (levonorgestrel intrauterine system)

For heavy menstrual bleeding with a small intramural fibroid and an undistorted cavity, the LNG-IUS is the recommended first-line uterus-sparing option (NICE HMB guidance) — it markedly reduces blood loss and is usable because the cavity is not distorted. Copper IUD worsens menstrual bleeding, and danazol is obsolete for this indication due to androgenic side effects.

💡 Cavity distorted nahi hai toh Mirena fit hai — LNG-IUS is the hysterectomy-avoider for fibroid HMB.

**Q120** OBG

★ Asked directly from our Rank Booster lectures

A patient diagnosed with molar pregnancy (ultrasound showing snowstorm/honeycomb appearance) has undergone suction evacuation. What is the next step in management?

 *Ultrasound showing the classic snowstorm/honeycomb appearance of multiple cystic spaces in a hydatidiform mole.*

- A Weekly beta HCG monitoring
- B Methotrexate for chemoprophylaxis
- C Hysterectomy
- D Ultrasound as and when indicated

**Answer** Weekly beta-hCG monitoring

After evacuation of a hydatidiform mole, serial serum beta-hCG is measured weekly until it normalizes, then monthly (about 6 months), with reliable contraception during follow-up — this is how persistent gestational trophoblastic neoplasia is detected early. Routine methotrexate chemoprophylaxis is not standard, and hysterectomy is reserved for selected older women who have completed their family.

💡 Mole nikal diya toh kaam khatam nahi — weekly beta-hCG hi asli follow-up hai.

**Q121** OBG

Which of the following is an absolute contraindication for External Cephalic Version (ECV)?

 *Schematic four-step diagram of external cephalic version, showing manual rotation of the fetus from breech to cephalic presentation.*

- A Dead fetus
- B Primi
- C Transverse lie
- D Fixed flexed breech
- E 3.5 kg baby

**Answer** Dead fetus (intrauterine fetal demise)

ECV exists solely to convert a malpresentation into cephalic so a healthy vaginal delivery of a live baby can occur; with a dead fetus the procedure has no purpose (and a dead fetus in breech can simply be delivered vaginally), making it the absolute contraindication among the options. Primigravidity, fetal weight around 3.5 kg, and a flexed breech are not contraindications, and both sources plus the on-screen marked answer agreed.

💡 ECV ka maqsad hi zinda bachhe ki normal delivery hai — dead fetus me ECV karna hi bemaani.

**Q122** OBG

★ Asked directly from our Rank Booster lectures

A 39-week pregnant patient presents with rupture of membranes for 12 hours and mild blood-stained show, with no abdominal pain or uterine tenderness. Ultrasound shows no low-lying placenta, NST is normal, the patient is stable, and the presentation is vertex. What is the next step in management?

- A conservative management
- B tocolysis
- C induction of labor
- D cesarean section

**Answer** Induction of labor

This is term pre-labor rupture of membranes (PROM) in a stable patient with vertex presentation and no placenta previa; current practice (ACOG, TermPROM evidence) is to offer induction of labor rather than prolonged expectant management, as it reduces chorioamnionitis without increasing cesarean rates. Tocolysis has no role at term, and there is no indication for cesarean here.

💡 Term + membranes ruptured + vertex + stable = induce, don't wait for infection to make the decision for you.

**Q123** OBG

A 65-year-old woman with cardiac illness presents with uterine prolapse. She desires a quick procedure with minimal morbidity. Which surgical option is most appropriate?

- A Sacrocolpopexy
- B Shirodkar (sling procedure)
- C Le Fort's operation
- D Ward Mayo (hysterectomy)

**Answer** Le Fort's operation (partial colpocleisis)

Le Fort colpocleisis is a short, low-morbidity obliterative procedure performable under regional/local anesthesia — ideal for an elderly, medically high-risk woman who is no longer sexually active. Sacrocolpopexy and sling procedures are longer reconstructive operations, and Ward-Mayo vaginal hysterectomy carries more surgical risk than this cardiac patient warrants.

💡 Old, frail, no coital function needed = Le Fort — close the vagina, close the case.

**Q124** OBG

★ Asked directly from our Rank Booster lectures

A patient at 38 weeks has well-controlled gestational diabetes mellitus and a Bishop score of 7. At what gestational age should induction of labor be considered?

**Answer** At 39 completed weeks (39 0/7 to 40 6/7 weeks)

For GDM that is well controlled (especially diet-controlled, GDM-A1), ACOG recommends delivery between 39 0/7 and 40 6/7 weeks; delivery before 39 weeks is reserved for poorly controlled diabetes or other complications. The favorable Bishop score of 7 predicts successful induction but does not itself advance the timing.

💡 Sugar controlled = baby ko 39 weeks tak pakne do; jaldi induction sirf uncontrolled GDM me.

**Q125** OBG

An Rh-negative isoimmunized pregnant woman (Coombs positive, previous isoimmunization, no fetal hydrops) at 28 weeks has an MCA Doppler showing a peak systolic velocity of 1.65 MoM. What is the interpretation and appropriate management?

 MCA Doppler tracing/report showing a peak systolic velocity of 1.65 multiples of the median.

- A** Mild anemia
- B** Moderate to severe anemia
- C** No anemia

**Answer** **Moderate-to-severe fetal anemia; at 28 weeks proceed to cordocentesis (fetal blood sampling) with intrauterine transfusion if confirmed**

An MCA peak systolic velocity greater than 1.5 MoM predicts moderate-to-severe fetal anemia (Mari criteria), so 1.65 MoM at 28 weeks mandates cordocentesis to confirm the hematocrit and allow intrauterine transfusion. MCA Doppler loses reliability after about 34-35 weeks, at which point delivery is considered instead — hence cordocentesis is NOT done in all patients irrespective of gestational age.

 MCA-PSV > 1.5 MoM = anemic fetus; before 34 weeks needle (cordocentesis + IUT), after 34 weeks deliver.

**Q126** OBG

A post-operative patient who was voiding normally presents with continuous dribbling of urine from the vagina. On the three-swab test (methylene blue instilled into the bladder), the uppermost swab is wet with urine but not stained blue. What is the diagnosis?

**Answer** **Ureterovaginal fistula**

In the three-swab test, blue staining of the upper swab indicates a vesicovaginal fistula, blue staining of the lower swab suggests urethral leakage, while an upper swab that is wet with clear (unstained) urine means urine is entering the vagina directly from the ureter — a ureterovaginal fistula, classically after pelvic surgery with ureteric injury. Preserved normal voiding supports this, since the bladder circuit is intact.

 Upur wala swab geela but blue nahi = ureter ka leak, bladder ka nahi.

**Q127** OBG

A vesicovaginal fistula is diagnosed on post-operative day 8 after a cesarean section for a stillbirth (obstetric VVF). What is the management?

- A** Repair immediately
- B** Reopen
- C** Delayed repair

**Answer** **Delayed repair (after about 8-12 weeks), with continuous bladder catheterization in the interim**

A VVF presenting in the post-operative period is repaired only after local edema, inflammation, and tissue sloughing have settled — classically 8-12 weeks — because repair through inflamed tissue breaks down. Meanwhile continuous catheter drainage keeps the bladder decompressed, and a small fresh fistula may even close spontaneously on prolonged catheterization.

 Fresh VVF pe turant tanka mat lagao — catheter daalo, tissue ko thanda hone do, phir repair.

**Q128** OBG

Ovarian hyperstimulation syndrome (OHSS) is associated with which receptor abnormality?

**Answer** Gain-of-function (activating) mutation of the FSH receptor

Spontaneous and familial recurrent OHSS is caused by activating (gain-of-function) mutations of the FSH receptor, which make the mutant receptor promiscuously responsive to hCG (and even TSH), driving massive follicular stimulation in pregnancy (Vasseur et al., NEJM 2003). The distractor 'gain of function of LH' is wrong — the ligand that triggers OHSS is hCG acting on the hypersensitive FSH receptor, not an LH-receptor mutation.

💡 OHSS genetics = FSH receptor ka gain-of-function — mutant FSHR hCG ki bhi sunta hai.

**Q129** OBG

★ Asked directly from our Rank Booster lectures

A patient with hydrosalpinx is undergoing IVF. What is the management of the hydrosalpinx before embryo transfer to improve the outcome?

- A Clipping (proximal clipping)
- B Salpingectomy
- C Ultrasound-guided aspiration of hydrosalpinx
- D Salpingostomy
- E Aspirate the fluid
- F Obstruction of both tubes

**Answer** Laparoscopic salpingectomy (or proximal tubal occlusion/clipping) before embryo transfer

Hydrosalpinx fluid is embryotoxic and refluxes into the cavity, roughly halving implantation rates; Cochrane evidence shows laparoscopic salpingectomy or proximal tubal occlusion before IVF significantly improves live birth rates. Ultrasound-guided aspiration is inferior because the fluid re-accumulates, and salpingostomy risks recurrence.

💡 Hydrosalpinx = zehreela paani upstairs mat jaane do — clip it or clip it out before transfer.

**Q130** OBG

★ Asked directly from our Rank Booster lectures

In an ectopic pregnancy, which clinical findings or patient characteristics indicate surgical management (i.e., when is medical management with methotrexate NOT appropriate)?

- A Patient is unstable
- B Patient is not willing for follow-up
- C Beta-HCG is more than 5000
- D Cardiac activity is present
- E Sac size is more than 4 cm
- F Woman's family is complete
- G HCG less than 1500
- H Sac size less than 4 cm

**Answer** Surgery is indicated when the patient is hemodynamically unstable, unwilling/unable to follow up, beta-hCG > 5000 IU/L, fetal cardiac activity is present, adnexal mass > 4 cm, or the family is complete

Methotrexate is reserved for stable patients with an unruptured ectopic, beta-hCG below about 5000 IU/L, mass under 3.5-4 cm, no fetal cardiac activity, and reliable follow-up; violation of any criterion (or a ruptured ectopic) mandates surgery.

💡 MTX ke 4 door-closers: unstable, hCG > 5000, sac > 4 cm, cardiac activity — koi bhi ho toh OT chalo.



**Q131** OBG

★ Asked directly from our Rank Booster lectures

A pregnant patient has a TSH of 6.4-6.8 mIU/L, normal free T4, and positive anti-TPO antibodies. What is the diagnosis?

- A Overt hypothyroidism
- B Subclinical hypothyroidism
- C Thyrotoxicosis
- D Gestational thyrotoxicosis

**Answer** Subclinical hypothyroidism

Elevated TSH (above the pregnancy upper limit of ~4 mIU/L per ATA 2017) with a normal free T4 defines subclinical hypothyroidism; overt hypothyroidism requires a low free T4. The positive anti-TPO antibodies indicate autoimmune (Hashimoto) etiology and, importantly, tip the balance toward levothyroxine treatment in pregnancy when TSH exceeds 4.

💡 High TSH + normal T4 = subclinical; pregnancy me TPO-positive ho toh treat bhi karna hai.

**Q132** OBG

★ Asked directly from our Rank Booster lectures

A 30-year-old woman, after an uncomplicated vaginal delivery, develops pelvic pain and thrombophlebitis in the postpartum period, followed by sudden dyspnea and pleuritic chest pain. What is the most appropriate investigation to confirm the diagnosis?

**Answer** CT pulmonary angiography (CTPA)

Postpartum pelvic (septic) thrombophlebitis followed by sudden dyspnea and pleuritic chest pain indicates pulmonary embolism, and CT pulmonary angiography is the confirmatory investigation of choice. Both sources gave the identical answer in different words; V/Q scanning is an alternative only when CTPA is contraindicated.

💡 Postpartum leg/pelvic clot + sudden breathlessness = PE; CTPA seals the diagnosis.

**Q133** OBG

What is the critical titer for the Indirect Coombs Test (ICT) in Rh isoimmunization?

**Answer** 1:16

In Rh-isoimmunized pregnancy, an indirect Coombs (anti-D antibody) titer of 1:16 is the commonly used critical titer (labs vary between 1:8 and 1:32; Williams Obstetrics cites 1:16): at or above it, fetal surveillance with serial MCA-PSV Doppler is begun instead of titer-based follow-up alone.

💡 ICT 1:16 crossed = titer bharosa chhodo, MCA Doppler pakdo.

## 12

## Pediatrics

16 recalled questions

## Q134 PEDIATRICS

★ Asked directly from our Rank Booster lectures

A child presents with pallor and decreased urine output for two days, with a history of eating undercooked meat / an episode of dysentery. Examination and labs show anemia, schistocytes on smear, thrombocytopenia, and a rising serum creatinine of 2.3. What is the diagnosis?

- A HUS
- B TTP
- C ITP

**Answer** HUS (hemolytic uremic syndrome)

The triad of microangiopathic hemolytic anemia (schistocytes), thrombocytopenia, and acute kidney injury in a child after a diarrheal illness or undercooked meat is Shiga toxin-associated HUS (*E. coli* O157:H7 or *Shigella*). TTP is the key distractor, but it favors adults, has prominent neurological features with severe ADAMTS13 deficiency, and typically milder renal involvement; ITP is isolated thrombocytopenia without hemolysis or renal failure.

💡 Bachcha + diarrhea + schistocytes + failing kidney = HUS; adult + neuro signs = TTP.

## Q135 PEDIATRICS

A 12-year-old child with dengue received IV crystalloid infusion for 36 hours. The next day the child developed tachypnea, breathlessness, bilateral basal crepitations and periorbital edema, and the hematocrit fell from 46% to 32%. Blood pressure is normal. What is the most likely diagnosis?

- A Fluid overload
- B Plasma leakage

**Answer** Fluid overload

Respiratory distress with basal crepitations and periorbital edema after 36 hours of crystalloids, together with a falling hematocrit and stable BP, indicates iatrogenic fluid overload — reabsorption of leaked plasma in the recovery phase plus the infused fluid dilutes the hematocrit (WHO dengue guidelines list exactly these warning signs for stopping IV fluids). Ongoing plasma leakage, the distractor, would raise the hematocrit and drop the blood pressure, the opposite of this picture.

💡 Dengue mein hematocrit girta hua + saans phoolna + BP normal = paani zyada ho gaya, leak nahi — fluids band karo.

**Q136** PEDIATRICS

★ Asked directly from our Rank Booster lectures

A 2-year-old child weighing 8.2 kg, fed mainly on kanji (rice gruel), comes for a checkup and has bilateral pitting pedal edema. Shakir's (MUAC) tape shows the red zone, less than 11.5 cm (images shown). What is the diagnosis and treatment plan?

 Clinical photographs of bilateral pitting pedal edema plus a Shakir's/MUAC tape reading in the red zone (<11.5 cm).

- A Moderate malnutrition and send home
- B SAM and admission followed by nutritional rehabilitation
- C Protein malnutrition - treat accordingly
- D Underlying other condition

**Answer** SAM and admission followed by nutritional rehabilitation

By WHO/IMNCI criteria, either MUAC below 11.5 cm (red zone) or bilateral pitting edema independently defines severe acute malnutrition - this child has both. Edematous SAM is complicated SAM and mandates inpatient care at a Nutritional Rehabilitation Centre: stabilisation with F-75, cautious rehydration, then catch-up feeding with F-100/RUTF. Sending the child home as 'moderate' malnutrition misses the edema criterion entirely.

💡 Edema ya red tape - dono mein se ek bhi = SAM; edema ho to seedha NRC admit.

**Q137** PEDIATRICS

★ Asked directly from our Rank Booster lectures

A neonate presents with hepatosplenomegaly, jaundice, thrombocytopenia, chorioretinitis and hearing loss, with purpuric 'blueberry muffin' rashes (image shown). What is the most likely cause?

 Clinical photograph of a neonate with multiple purpuric 'blueberry muffin' skin lesions over the body.

- A LCH (Langerhans cell histiocytosis)
- B Leukemia cutis
- C Neuroblastoma
- D Congenital cytomegalovirus (CMV)

**Answer** Congenital cytomegalovirus (CMV)

Blueberry muffin rash (dermal extramedullary erythropoiesis) combined with chorioretinitis, sensorineural hearing loss, hepatosplenomegaly, thrombocytopenia and periventricular calcifications is classic congenital CMV - the most common congenital infection and the leading non-genetic cause of childhood sensorineural deafness. Neuroblastoma and leukemia cutis can produce a blueberry muffin baby but do not explain the chorioretinitis and deafness; symptomatic babies are treated with IV ganciclovir or oral valganciclovir.

💡 Blueberry muffin + behra + chorioretinitis = congenital CMV; calcification periventricular (Toxo mein scattered).

**Q138** PEDIATRICS

★ Asked directly from our Rank Booster lectures

A 6-year-old child presents with cola-coloured urine (hematuria), hypertension and edema, 2 weeks after an episode of sore throat. Light microscopy shows hypercellular glomeruli with diffuse neutrophilic infiltration. What is the most probable diagnosis?

 Light micrograph of a hypercellular glomerulus with diffuse neutrophilic infiltration (diffuse proliferative GN).

- A Rapidly progressive glomerulonephritis
- B Minimal change disease
- C Membranoproliferative glomerulonephritis
- D Acute post-streptococcal glomerulonephritis

**Answer** Acute post-streptococcal glomerulonephritis

An acute nephritic syndrome (cola-coloured urine, hypertension, edema) appearing 1-3 weeks after streptococcal pharyngitis, with diffuse proliferative hypercellular glomeruli infiltrated by neutrophils on light microscopy and low C3, is post-streptococcal glomerulonephritis. Minimal change disease is a nephrotic (not nephritic) picture with normal light microscopy, and RPGN shows crescents with rapidly worsening renal failure.

💡 Gale ke 2 hafte baad cola urine = PSGN; C3 girta hai, 6-8 hafte mein wapas.

**Q139** PEDIATRICS

A child less than 2 months old has a red umbilicus or draining pus or skin pustules, with no danger signs. According to IMNCI, this is classified as:

- A No disease - green colour, managed at home
- B Local bacterial infection - yellow colour, treat at home and follow up in 2 days
- C Severe disease - pink colour, urgent referral to hospital
- D Severe disease - red colour, urgent referral to hospital

**Answer** Local bacterial infection - yellow colour, treat at home and follow up in 2 days

In the IMNCI young-infant (up to 2 months) chart, umbilical redness or pus, or skin pustules, in the absence of danger signs, is classified as local bacterial infection - the yellow row - managed with oral amoxicillin at home and mandatory follow-up in 2 days. Danger signs (lethargy, refusal to feed, convulsions, chest indrawing, fever or hypothermia) would upgrade it to possible serious bacterial infection - pink, urgent referral with pre-referral antibiotics. IMNCI has no red category.

💡 IMNCI: pink = refer, yellow = clinic treatment, green = ghar; laal naabhi bina danger signs = yellow.

**Q140** PEDIATRICS

A boy presents with progressive bowing of the legs; his elder brother has a similar history but his sister is normal, and the diet is adequate. Investigations show normal calcium, normal vitamin D and PTH, low serum phosphate with reduced renal phosphate reabsorption, and a normal ABG. What is the diagnosis?

- A Nutritional rickets
- B Renal tubular acidosis
- C Fanconi syndrome
- D Hypophosphatemic rickets

**Answer** Hypophosphatemic rickets (X-linked)

Refractory rickets with hypophosphatemia, renal phosphate wasting, and normal calcium, vitamin D and PTH is hypophosphatemic rickets - here X-linked dominant (PHEX mutation causing excess FGF23), fitting the affected brothers. The normal ABG excludes renal tubular acidosis and Fanconi syndrome, and the adequate diet with normal vitamin D excludes nutritional rickets. Treatment is oral phosphate with active vitamin D analogues, or burosumab.

💡 Rickets + low PO<sub>4</sub> + baaki sab normal + bhai bhi affected = XLH; phosphate leak hai, vitamin D ki kami nahi.

**Q141** PEDIATRICS

★ Asked directly from our Rank Booster lectures

A child with a triangular face (image shown), polyuria-polydipsia and sensorineural hearing loss is found to have low serum potassium, metabolic alkalosis and high urinary calcium. This could be due to a defect in which part of the nephron?

 Clinical photograph of a child with triangular face, prominent forehead and large ears - facies of Bartter syndrome type IV (with sensorineural deafness).

- A DCT
- B Proximal convoluted tubule
- C Thin ascending limb
- D Thin descending limb
- E Thick ascending limb

**Answer** Thick ascending limb of loop of Henle

Hypokalemic metabolic alkalosis with normal blood pressure and hypercalciuria defines Bartter syndrome, a defect of the NKCC2/ROMK/CIC-Kb transport machinery in the thick ascending limb - the picture of being permanently on a loop diuretic; the dysmorphic facies and sensorineural deafness point to type IV (barttin mutation). A DCT defect would be Gitelman syndrome, which has hypocalciuria instead.

💡 Bartter = born on furosemide (TAL, urine Ca high); Gitelman = born on thiazide (DCT, urine Ca low).

**Q142** PEDIATRICS

★ Asked directly from our Rank Booster lectures

A male child is diagnosed with hemophilia A after recurrent joint bleeds; his brother has the same condition and his sister is normal. Which other relative is most likely to be affected?

- A Maternal uncle
- B Maternal grandfather
- C Maternal male cousins
- D Sibling
- E Father's brother
- F Father

**Answer** Maternal uncle (mother's brother)

Hemophilia A is X-linked recessive: two affected sons make the mother an obligate carrier, and each of her brothers has a substantial chance of carrying the same maternal X and being affected - the classic single best answer. The father's side is irrelevant because an affected boy receives only the Y chromosome from his father; the maternal grandfather could be affected only if the mutant X came through him rather than arising in the grandmother's line, making the uncle the more likely choice.

💡 X-linked recessive mein hamesha mama ki taraf dekho.

**Q143** PEDIATRICS

★ Asked directly from our Rank Booster lectures

Which cardiac anomaly is associated with an 'egg on string' appearance on chest X-ray?

**Answer** Transposition of the great arteries (TGA)

The 'egg on a string' (egg-on-side) silhouette - an oval cardiac shadow with a narrow superior mediastinum from the anteroposteriorly aligned great vessels and thymic involution - in a cyanotic neonate is classic for d-TGA. The boot-shaped heart belongs to Tetralogy of Fallot and the snowman/figure-of-8 to supracardiac TAPVC.

💡 Egg on string = TGA; boot = TOF; snowman = TAPVC - X-ray shapes ratta maar lo.

**Q144** PEDIATRICS

★ Asked directly from our Rank Booster lectures

A 4-month-old infant presents with a heart rate of 260/min and normal blood pressure; ECG shows a narrow-QRS tachycardia with absent P waves (strip shown). After vagal stimulation, what is the next step in management?

 ECG strip showing a very fast narrow-QRS complex tachycardia with absent P waves (SVT).

- A Propranolol
- B Amiodarone
- C Cardioversion (shock)
- D Adenosine

**Answer** Adenosine

A rate of 260/min with narrow QRS and absent P waves in an infant is supraventricular tachycardia (infant sinus tachycardia rarely exceeds ~220/min). Per PALS guidelines, a hemodynamically stable SVT gets vagal maneuvers followed by rapid-push IV adenosine; synchronized cardioversion (0.5-1 J/kg) is reserved for unstable patients or failed adenosine. Verapamil is contraindicated in infants, and amiodarone/propranolol are later-line options.

💡 SVT stable = adenosine (flush ke saath jhatke se); unstable = sync shock - PALS ka simple funda.

**Q145** PEDIATRICS

A 9-month-old child with breathlessness has a pansystolic murmur at the left lower sternal border and a mid-diastolic murmur at the apex; chest X-ray shows cardiomegaly with pulmonary plethora. What is the cause of the mid-diastolic murmur?

 CXR described with cardiomegaly and pulmonary plethora; discussion frame showed a hand-drawn shunt diagram labelling the apical 'flow murmur'.

- A Increased flow velocity across mitral valve
- B Reduced flow velocity across pulmonic valve
- C Increased flow velocity across aortic valve
- D Structural mitral stenosis

**Answer** Increased flow velocity across the mitral valve

The pansystolic murmur with pulmonary plethora indicates a large VSD with a left-to-right shunt; the augmented pulmonary venous return crossing a normal mitral valve in diastole produces a functional 'relative mitral stenosis' flow murmur at the apex - a marker that the shunt is hemodynamically significant. Structural mitral stenosis is the distractor: it is rheumatic and essentially unheard of at 9 months, and the valve here is anatomically normal.

 Bade VSD ka apical MDM = flow murmur - valve kharab nahi, traffic zyada hai.

**Q146** PEDIATRICS

A child presents with short stature. Investigations show increased growth hormone with decreased IGF-1 levels, due to a defect in the growth hormone receptor. What is the diagnosis?

- A Laron's dwarfism
- B Cretinism
- C Psychosocial dwarfism

**Answer** Laron dwarfism (growth hormone insensitivity syndrome)

A GH receptor defect makes tissues unresponsive to growth hormone, so IGF-1 stays low while GH is high or normal (loss of negative feedback) - the hormonal signature of Laron dwarfism. Because the block is at the receptor, GH therapy is useless; treatment is recombinant IGF-1 (mecasermin). Cretinism would show hypothyroid features, and psychosocial dwarfism has reversible functional GH suppression.

 GH high, IGF-1 low = receptor behra hai - Laron; ilaaj GH nahi, mecasermin (IGF-1).

**Q147** PEDIATRICS

A 4-year-old child with normal growth is found to have a continuous murmur; echocardiography shows a small patent ductus arteriosus with no pulmonary hypertension and no features of congestive heart failure. What is the next best step?

- A Indomethacin
- B Reassurance and follow-up every year
- C Open heart surgery
- D Device closure

**Answer** Device closure (transcatheter)

A PDA that is audible at 4 years will not close spontaneously and carries a lifelong risk of infective endarteritis and progressive volume load, so closure is indicated even when asymptomatic (AHA/ACC congenital guidance recommends closing the audible PDA). Beyond infancy, transcatheter device/coil closure is the standard of care for small-to-moderate ducts; open surgical ligation is reserved for very large ducts or unfavourable anatomy, and indomethacin/paracetamol works only in preterm neonates.

 4 saal ka sunai dene wala PDA - dawa nahi, dher sa intezaar nahi: device closure.

**Q148** PEDIATRICS

★ Asked directly from our Rank Booster lectures

What is the duration of protection provided by polio immunization/infection?

 Graph of polio neutralizing antibody levels over time (exam image not recovered).

**A** Lifetime

**B** 15 to 16 weeks

**Answer** **Lifetime (long-lasting, serotype-specific) protection**

Both natural poliovirus infection and a completed immunization series produce durable, essentially lifelong protection against paralytic polio, though immunity is serotype-specific - protection against one type does not cover the other types. The recalled '15-16 weeks' distractor appears garbled, possibly echoing the primary OPV schedule weeks (6, 10, 14).

 Polio immunity lifelong hai par serotype-specific - isliye teeno types ke against vaccination zaroori.

**Q149** PEDIATRICS

★ Asked directly from our Rank Booster lectures

A child presents with a rash. Immunohistochemistry of the lesion is CD1a positive and S100 positive. What is the probable diagnosis?

**Answer** **Langerhans cell histiocytosis (LCH)**

CD1a and S100 positivity (along with CD207/langerin) is the defining immunophenotype of Langerhans cell histiocytosis, which in children can present as a seborrheic-dermatitis-like scaly petechial rash with bone lesions, otorrhea or diabetes insipidus. Electron microscopy shows the tennis-racket-shaped Birbeck granules; langerin positivity implies Birbeck granules are present.

 CD1a + S100 + langerin = LCH; EM mein tennis racket (Birbeck granules).

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## 13 ENT

6 recalled questions

### Q150 ENT

A 48-year-old woman presents with progressive deepening of her voice and hoarseness for several months. She has a long history of smoking. Laryngoscopy shows the following findings. What is the most likely diagnosis?

 Laryngoscopic view showing bilateral diffuse polypoidal (edematous) swelling of the membranous portion of both vocal cords.

- A Post cricoid malignancy
- B Sulcus vocalis
- C Reinke's edema
- D Laryngeal papillomatosis

**Answer** Reinke's edema

A middle-aged smoker with a gradually deepening, husky voice and bilateral diffuse polypoid swelling of the membranous vocal cords has Reinke's edema — fluid accumulation in the subepithelial space of Reinke (superficial lamina propria). Laryngeal papillomatosis shows multiple warty exophytic growths (HPV 6/11), not smooth bilateral polypoid cords, and post-cricoid malignancy presents with dysphagia rather than isolated voice change.

 Smoker aunty with a 'male' voice + bilateral boggy cords = Reinke's edema in Reinke's space.

### Q151 ENT

★ Asked directly from our Rank Booster lectures

Which of the following statements regarding epistaxis is correct?

- A Little's area is located on the lateral wall of the nose
- B The anterior and posterior ethmoidal arteries, branches of the maxillary artery, supply both the lateral wall and the nasal septum
- C Posterior epistaxis is easier to manage
- D Trotter's method for 5 to 10 minutes, followed by an ice pack application

**Answer** Trotter's method for 5 to 10 minutes, followed by an ice pack application

Trotter's method — sitting up, leaning forward and pinching the nose for 5–10 minutes while breathing through the mouth, with an ice pack — is the correct first-aid step for epistaxis. The other statements are all false: Little's area lies on the antero-inferior nasal septum (not lateral wall), the ethmoidal arteries are branches of the ophthalmic artery (not maxillary), and posterior epistaxis, typically in elderly hypertensives, is harder to control than anterior bleeding.

 Epistaxis first aid: pinch, lean forward, ice — Trotter's method; baaki teeno statements classic traps hain.

**Q152** ENT

A 38-year-old woman presents with progressive unilateral sensorineural hearing loss and intermittent tinnitus. She has decreased sensation over the posterosuperior aspect of the external auditory canal. What is the gold standard investigation to establish the diagnosis?

- A BERA
- B PTA
- C HRCT temporal
- D Carotid angio
- E MRI with contrast

**Answer** MRI with contrast (gadolinium-enhanced MRI)

Unilateral progressive SNHL with tinnitus plus hypoesthesia of the posterosuperior canal (Hitselberger's sign) is classic for vestibular schwannoma, and gadolinium-enhanced MRI of the cerebellopontine angle is the gold standard, detecting even intracanalicular tumors of a few millimetres. BERA is a useful screening/audiological test (delayed wave V, interaural latency difference) but is less sensitive for small tumors and does not establish the diagnosis.

💡 Unilateral SNHL + Hitselberger sign = acoustic neuroma — MRI with gadolinium, seedha CP angle dekho.

**Q153** ENT

A child underwent adenoidectomy and subsequently developed abnormal speech. The child's pronunciation of "dada" sounds like "nana." The image shows the finding below. What is the most likely cause of this speech abnormality?

 Clinical photograph of the oral cavity showing a bifid uvula, indicating a submucous cleft palate.

- A Rhinolalia aperta
- B Rhinolalia clausa
- C Hot potato voice
- D Hoarseness of voice

**Answer** Rhinolalia aperta

A bifid uvula marks a submucous cleft palate — a contraindication to adenoidectomy because the adenoid pad was compensating for velopharyngeal closure. Removing it unmasks velopharyngeal insufficiency, producing hypernasal speech (rhinolalia aperta) where oral consonants become nasalized ('dada' sounds like 'nana'). Rhinolalia clausa is the opposite — hyponasality from nasal obstruction, as seen with the adenoids still in place.

💡 Bifid uvula dikhe toh adenoidectomy mat karo — warna rhinolalia aperta pakka.

**Q154** ENT

An 18-year-old male presents with a nasal mass and profuse bleeding, suggestive of juvenile nasopharyngeal angiofibroma (JNA). What is the pathophysiological basis of the severe bleeding in JNA?

- A** Lack of smooth muscle
- B** Extensive blood supply
- C** Vascular invasion
- D** Absent capsule

**Answer** **Lack of smooth muscle (in the vessel walls)**

The vessels of a JNA are irregular endothelium-lined channels whose walls lack the smooth muscle (tunica media) and elastic tissue needed for vasoconstriction, so once torn they cannot contract and bleeding is profuse and difficult to control. The absent capsule explains local invasiveness and recurrence, not the bleeding, which is why it is the key distractor.

💡 JNA bleeds because its vessels have no muscle to squeeze — biopsy kabhi mat lena.

**Q155** ENT

★ Asked directly from our Rank Booster lectures

A 25-year-old woman presents with bilateral progressive hearing loss. Pure tone audiometry shows a characteristic notch. What is the most likely diagnosis?

📎 Pure tone audiogram reportedly showing a notch/dip (frequency recalled variably as 2–4 kHz).

**Answer** **Otosclerosis (Carhart's notch at 2000 Hz)**

A young woman with bilateral progressive hearing loss and no stated noise exposure fits otosclerosis, whose audiometric hallmark is Carhart's notch — a dip in bone conduction centred at 2000 Hz caused by stapes fixation, which disappears after successful stapedotomy. Noise-induced hearing loss, the main distractor, requires an occupational noise history and shows a notch in both air and bone conduction at 4000 Hz.

💡 Young female, bilateral progressive HL, notch at 2K in BC = otosclerosis; 4K notch with noise history = NIHL.

## 14 Ophthalmology

7 recalled questions

### Q156 OPHTHALMOLOGY

A 30-year-old farmer presents with a history of organic matter (sugarcane) injury to the eye and develops a corneal ulcer with feathery margins and hypopyon (clinical image shown). Which organism is the most likely cause?

 Clinical photograph of a corneal ulcer with feathery margins and hypopyon, characteristic of fungal keratitis.

- A HSV
- B Strep
- C Aureus
- D Mucor
- E Fusarium

Answer **Fusarium**

Trauma with vegetative matter classically causes filamentous fungal keratitis, and Fusarium is the most common agent in agricultural settings (Aspergillus is the other major cause). The dry-looking ulcer with feathery hyphate margins, satellite lesions and hypopyon shown in the image is the textbook fungal picture. Bacterial ulcers are wet with defined margins, HSV gives a dendritic ulcer, and Mucor causes rhino-orbital disease in diabetics rather than keratitis.

💡 Ganne ki chot + feathery margin + hypopyon = fungus; Fusarium likhkar rakh lo.

### Q157 OPHTHALMOLOGY

★ Asked directly from our Rank Booster lectures

A 60-year-old patient with long-standing diabetes mellitus and hypertension presents with painless diminution of vision. An OCT scan showing retinal thickening with intraretinal cystic spaces was provided. What is the diagnosis?

 OCT scan of the macula showing multiple intraretinal cystic spaces with retinal thickening, characteristic of cystoid macular edema.

- A Central serous retinopathy
- B Cystoid macular edema
- C Retinal detachment
- D Macular hole

Answer **Cystoid macular edema**

OCT showing increased retinal thickness with multiple hyporeflective intraretinal cystic spaces is diagnostic of cystoid macular edema, a common complication of diabetic maculopathy at this age. Central serous chorioretinopathy typically affects young adults (often on steroids) and shows a dome of subretinal fluid, not intraretinal cysts; a macular hole shows a full-thickness neurosensory defect.

💡 OCT mein retina ke andar cysts = CME; retina ke neeche paani ka gumbad = CSR.

**Q158** OPHTHALMOLOGY

A female patient presents with sudden diminution of vision, a pale disc, headache, and an altitudinal visual field defect (inferior hemianopia). What is the diagnosis?

 Visual field diagrams shown in discussion: altitudinal defect (inferior hemianopia), central scotoma and centrocecal scotoma.

- A** AION (Anterior ischemic optic neuropathy)
- B** Papilledema
- C** Optic neuritis
- D** Posterior ischemic optic neuropathy (PION)
- E** Optic atrophy

**Answer** **AION (Anterior ischemic optic neuropathy) - arteritic type suggested by headache**

Sudden painless monocular vision loss with an altitudinal (inferior) field defect and a pale, swollen disc is the classic picture of anterior ischemic optic neuropathy; the accompanying headache in an older woman suggests the arteritic type from giant cell arteritis, which needs urgent ESR/CRP and steroids to protect the fellow eye. Papilledema is bilateral with preserved acuity and an enlarged blind spot early, PION initially has a normal disc, and optic neuritis affects young patients with pain on eye movement.

 Altitudinal defect + chalky pale swollen disc + headache = arteritic AION; ESR bhejo, steroid do.

**Q159** OPHTHALMOLOGY

★ Asked directly from our Rank Booster lectures

During an epidemic of conjunctivitis, a patient presents with a painful red eye with prominent subconjunctival hemorrhage ('blood-shot eye' image) and preauricular lymph node involvement. What is the most likely causative organism?

 Recalled image: diffusely 'blood-shot' eye with subconjunctival hemorrhage; no exam frame recovered.

- A** Enterovirus
- B** Adenovirus
- C** Herpes Simplex Virus
- D** Epstein-Barr Virus
- E** Coxsackie

**Answer** **Enterovirus (enterovirus 70)**

Epidemic acute hemorrhagic conjunctivitis - abrupt painful red eye with prominent subconjunctival hemorrhage spreading through a community - is caused by enterovirus 70 (and the coxsackievirus A24 variant), the so-called Apollo conjunctivitis. Preauricular lymphadenopathy does not discriminate, as it occurs in both enteroviral and adenoviral conjunctivitis. Adenovirus is the answer for epidemic KERATOconjunctivitis (follicles, corneal subepithelial infiltrates), where hemorrhage is not the hallmark.

 Epidemic + poora khoon-laal eye = Enterovirus 70 (Apollo disease); follicles + cornea = adenovirus.

**Q160** OPHTHALMOLOGY

A patient with anterior uveitis presents with pain, diminished vision, keratic precipitates and posterior synechiae (festooned pupil shown in image). Which drug should be given to break the synechiae and give rest to the ciliary muscle?

 Anterior segment photograph showing an irregular festooned pupil due to posterior synechiae.

- A Pilocarpine
- B Atropine
- C Cyclopentolate
- D Homatropine
- E Tropicamide
- F Phenylephrine

**Answer** Atropine

In anterior uveitis a cycloplegic-mydriatic is used to relieve ciliary spasm pain and to break or prevent posterior synechiae; atropine is the strongest and longest-acting agent and is the classic answer. Phenylephrine is a pure mydriatic with no cycloplegia, so it dilates without resting the ciliary body, and miotics like pilocarpine are contraindicated because they encourage synechiae formation.

💡 Uveitis mein pupil ko phaila kar aaraam do - Atropine; pilocarpine to dushman hai.

**Q161** OPHTHALMOLOGY

A young boy with squint (exotropia) since early childhood has visual acuity 6/6 in one eye and 6/60 in the other; fundus is normal and there is no significant refractive error. What is the cause of the diminished vision?

 Recalled image: child with exotropia (outward eye deviation).

- A Cortical suppression
- B Retinal degeneration
- C Retinal pigment degradation
- D Corneal opacification

**Answer** Cortical suppression

This is strabismic amblyopia: to avoid diplopia and confusion, the visual cortex actively suppresses the image from the deviating eye during the critical period, so acuity fails to develop despite a structurally normal eye. The normal fundus and absence of refractive error rule out the retinal and corneal options - the defect is cortical, which is why patching the good eye can recover vision if started early.

💡 Squint wali aankh kharab nahi hoti, dimaag usko ignore karta hai - cortical suppression (amblyopia).

**Q162** OPHTHALMOLOGY

★ Asked directly from our Rank Booster lectures

A patient has chronic uveitis with diffuse fine keratic precipitates, heterochromia iridis, and a positive Amsler sign (slit-lamp image shown). What is the diagnosis?

 Slit-lamp photograph showing diffuse fine keratic precipitates over the corneal endothelium; heterochromia iridis described in the stem.

- A** TB
- B** Sarcoidosis
- C** HLA-B27 uveitis
- D** Fuchs heterochromic iridocyclitis

**Answer** **Fuchs heterochromic iridocyclitis**

Chronic low-grade anterior uveitis with diffuse stellate KPs scattered over the whole endothelium, heterochromia iridis, and absence of synechiae is Fuchs heterochromic iridocyclitis (Fuchs uveitis syndrome). The Amsler(-Verrey) sign - a filiform hemorrhage from fragile iris vessels on anterior chamber paracentesis - is specific to it. Granulomatous causes (TB, sarcoid) give mutton-fat KPs, and HLA-B27 uveitis is acute, painful and synechiae-forming.

 Alag rang ki iris + diffuse KPs + Amsler sign = Fuchs; synechiae dhoondhoge to nahi milenge.

## 15 Orthopedics

4 recalled questions

### Q163 ORTHOPEDICS

An image of a young girl holding her arm was shown, with the forearm pronated and supported, after being pulled by the arm. What is the diagnosis?

 *Anatomical diagram of the radial head slipping out of the annular ligament, alongside a clinical illustration of a child holding a pronated, supported arm.*

- A Radial head subluxation
- B Radial head dislocation
- C Radial nerve neuropraxia
- D Supracondylar fracture
- E Elbow dislocation

**Answer** Radial head subluxation (pulled elbow / nursemaid's elbow)

A sudden longitudinal pull on a toddler's extended, pronated arm slips the radial head out from under the annular ligament - a subluxation, not a true dislocation. The child holds the forearm pronated and slightly flexed, refuses to use the arm, and X-rays are normal; reduction is by supination-flexion or hyperpronation. 'Pulled elbow' and 'radial head subluxation' are the same answer.

 Bachche ko haath se kheecha, haath pronated latak gaya = annular ligament se radial head phisla - subluxation, dislocation nahi.

### Q164 ORTHOPEDICS

After a road traffic accident with a fall on an outstretched hand, a wrist X-ray shows a definite gap between the scaphoid and the lunate. The patient is unable to make an 'okay' sign. What is the diagnosis, and which nerve is injured?

 *AP wrist X-ray showing a widened gap between the scaphoid and lunate (Terry Thomas sign).*

- A Scapholunate dissociation
- B Scapho-capitate dislocation
- C Scapho-hamate
- D Lunate dislocation
- E Volar Barton

**Answer** Scapholunate dissociation; median nerve (anterior interosseous branch)

A widened scapholunate interval on the AP wrist film after a fall on the outstretched hand is the Terry Thomas sign, diagnostic of scapholunate dissociation. Inability to make the 'okay' sign (loss of thumb IP and index DIP flexion) indicates anterior interosseous nerve dysfunction, and the median nerve is the nerve classically injured in carpal dislocations because of its position in the carpal tunnel.

 Scaphoid-lunate ke beech gap = Terry Thomas sign; OK sign fail = median nerve (AIN).



**Q165** ORTHOPEDICS

★ Asked directly from our Rank Booster lectures

A CT/X-ray image of a young patient (15-23 years recalled) with localized bone pain for 2 months showed a diaphyseal cortical lesion with a central radiolucent nidus and surrounding dense sclerosis. What is the diagnosis and its appropriate management?

 X-ray/CT of a long bone showing a well-defined radiolucent nidus surrounded by dense reactive cortical sclerosis in the diaphysis.

- A Fibrous dysplasia with diet management
- B Osteoid osteoma with radiofrequency ablation
- C Acute osteomyelitis with antibiotics
- D Non-ossifying fibroma

Answer **Osteoid osteoma with radiofrequency ablation**

A young patient with months of localized (classically nocturnal, NSAID-responsive) pain and a cortex-based radiolucent nidus surrounded by reactive sclerosis is osteoid osteoma; CT-guided radiofrequency ablation of the nidus is the standard treatment. The 2-month indolent course and the nidus rule out acute osteomyelitis, which presents acutely with systemic features and later shows sequestrum/involucrum, not a nidus.

 Raat ka dard + NSAID se aaram + nidus with sclerosis = osteoid osteoma; ilaaj RFA.

**Q166** ORTHOPEDICS

A diabetic patient presents with restriction of the shoulder joint, diagnosed as adhesive capsulitis. Which muscle is spared (not involved)?

- A Teres major

Answer **Teres major**

Adhesive capsulitis (frozen shoulder, strongly associated with diabetes) involves the joint capsule and the rotator cuff muscles - supraspinatus, infraspinatus, teres MINOR, and subscapularis (SITS). Teres major is not a rotator cuff muscle, so it is spared; the minor-versus-major confusion is the intended trap.

 Rotator cuff mein teres MINOR hai, MAJOR nahi - frozen shoulder mein teres major bacha rehta hai.

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## 16 Radiology

3 recalled questions

### Q167 RADIOLOGY

You are setting up a complete radiology department for a tertiary care hospital and planning to procure various imaging equipment. AERB permission is NOT required before procuring which of the following machines?

- A Ultrasound (USG)
- B X-ray
- C CT scan
- D Portable radiography
- E Mammography
- F C-arm

Answer **Ultrasound (USG)**

The Atomic Energy Regulatory Board licenses only equipment that uses ionizing radiation. X-ray, CT, portable radiography, mammography, and C-arm fluoroscopy are all X-ray-based and need AERB clearance; ultrasound uses sound waves with no ionizing radiation, so no AERB permission is required (the same applies to MRI).

💡 Jahan ionizing radiation nahi, wahan AERB nahi - USG aur MRI dono free hain.

### Q168 RADIOLOGY

★ Asked directly from our Rank Booster lectures

An X-ray of the foot showing an injury was given. What is the most likely diagnosis?

📖 *Foot radiographs - sources disagree on whether they showed tarsometatarsal (Lisfranc) malalignment or a fracture at the base of the fifth metatarsal.*

- A Kienbock's disease
- B Kohler's disease
- C Jones fracture
- D Lisfranc injury

Answer **Lisfranc injury**

A Lisfranc injury is disruption of the tarsometatarsal joint complex - look for widening between the first and second metatarsal bases and loss of alignment of the second metatarsal with the middle cuneiform. The distractors localize elsewhere: Jones fracture is at the fifth metatarsal metaphyseal-diaphyseal junction, Kohler's disease is navicular osteochondrosis in children, and Kienbock's disease is lunate avascular necrosis in the wrist, not the foot at all.

💡 Foot X-ray mein 2nd metatarsal apni cuneiform se hila = Lisfranc - subtle hai, miss mat karna.

**Q169** RADIOLOGY

★ Asked directly from our Rank Booster lectures

A mammography of a 60-year-old female was done and shown (craniocaudal and mediolateral oblique views), revealing a retroareolar spiculated mass. What is the most likely diagnosis?

 Mammography (CC and MLO views) showing a dense, irregular, spiculated retroareolar mass.

- A** Tuberculosis
- B** Carcinoma
- C** Mastitis
- D** Fibroadenoma

**Answer** Carcinoma

A dense, irregular, spiculated mass on mammography is a BI-RADS 5 lesion - highly suggestive of malignancy - and in a 60-year-old the diagnosis is breast carcinoma until proven otherwise. Fibroadenoma appears as a well-circumscribed, often popcorn-calcified mass in younger women, and mastitis shows skin thickening with inflammatory features.

 Mammogram pe spiculated margin = BI-RADS 5 = carcinoma likho, biopsy bhejo.

## 17 Anesthesia

3 recalled questions

### Q170 ANESTHESIA

A patient is planned for Total Knee Replacement (TKR) under spinal anesthesia. Which of the following should be considered an absolute contraindication for spinal anesthesia?

- A Patient refusal
- B Previous spine surgery
- C Hypovolemia
- D Patient on aspirin
- E Spinal deformity

**Answer** Patient refusal

Valid informed consent is mandatory for any procedure, so patient refusal is the universally accepted absolute contraindication to neuraxial anesthesia (alongside infection at the injection site, raised ICP and severe coagulopathy). Hypovolemia, aspirin monotherapy, previous spine surgery and spinal deformity are only relative contraindications - they make the block riskier or technically harder but do not forbid it.

💡 Consent nahi toh needle nahi - refusal beats every other contraindication.

### Q171 ANESTHESIA

An ETCO<sub>2</sub> (capnography) trace of a patient undergoing CPR is shown. What does a sudden increase or normalization of the ETCO<sub>2</sub> curve signify?

📎 Hand-drawn capnography waveforms during CPR: low ETCO<sub>2</sub> ( $\leq 10$  mmHg, poor prognosis/ineffective CPR), higher ETCO<sub>2</sub> ( $>20$  mmHg, good compressions), and a sudden rise indicating ROSC.

- A Pulmonary embolism
- B Rib damage and lung damage
- C ROSC (Return of Spontaneous Circulation)
- D Hypotension

**Answer** ROSC (Return of Spontaneous Circulation)

A sudden, sustained jump in ETCO<sub>2</sub> (typically to 35-40 mmHg or abrupt normalization of the waveform) during CPR means pulmonary blood flow has been restored - i.e., ROSC - and should prompt a rhythm/pulse check (AHA ACLS 2020). Pulmonary embolism and hypotension DROP the ETCO<sub>2</sub>; they never raise it abruptly. ETCO<sub>2</sub> under 10 mmHg during CPR signals inadequate compressions.

💡 CPR mein ETCO<sub>2</sub> ka achanak jump = dil wapas on;  $<10$  mmHg = compressions sudharo.

**Q172** ANESTHESIA

A capnography waveform is shown. What does the trace indicate?

 Capnography waveform (not captured in any recall; described only as a difficult trace, not the MH or air-embolism patterns).

- A** Bulla rupture
- B** Pulmonary edema
- C** Fibrosis
- D** Obstruction

**Answer** Airway obstruction — classic 'shark-fin' capnograph

The shark-fin (slurred upstroke) capnograph is the classic obstructed-airway/bronchospasm waveform tested on this topic — the expiratory plateau is lost because alveoli empty at different rates.

 Shark-fin capnograph = obstructed airway - bronchospasm ya kinked tube.

## 18 Dermatology

5 recalled questions

### Q173 DERMATOLOGY

★ Asked directly from our Rank Booster lectures

A patient presents with hypopigmented, finely scaly macules (lesions shown in the image), suggestive of pityriasis versicolor. What is the characteristic KOH mount finding?

 *Clinical photograph of hypopigmented macules on the neck/chest, shown alongside a cartoon of bananas and grapes representing Malassezia microscopy.*

- A Banana and grapes
- B Cigar-shaped yeast cells
- C Church spires
- D Claw clutching

**Answer** Banana and grapes appearance (spaghetti and meatballs)

Pityriasis versicolor is caused by *Malassezia* species, which on KOH mount show short curved hyphae with clusters of round yeast cells — classically 'spaghetti and meatballs', now increasingly phrased as 'banana and grapes'. Cigar-shaped yeast cells suggest *Sporothrix schenckii*, and church-spire papillomatosis is a histopathology term (e.g., in verrucae), not a KOH finding.

💡 Versicolor pe KOH = banana (hyphae) + grapes (spores) — wahi purana spaghetti-meatballs, nayi packing mein.

### Q174 DERMATOLOGY

★ Asked directly from our Rank Booster lectures

A post-infective patient presented with lesions on the trunk, including a characteristic herald patch. What is the most likely diagnosis?

 *Image-based item: trunk lesions with a larger herald patch in a fir-tree distribution (exam image not recovered).*

- A Pityriasis rosea
- B Guttate psoriasis

**Answer** Pityriasis rosea

A single larger herald patch followed by crops of oval, collarette-scaled macules along skin cleavage lines (fir-tree / Christmas-tree pattern) is diagnostic of pityriasis rosea, which is linked to HHV-6/7 reactivation and often follows a prodromal illness. Guttate psoriasis also follows infection (streptococcal pharyngitis) but presents as drop-like scaly papules with no herald patch.

💡 Herald patch = pityriasis rosea ka trailer — pehle ek badi lesion, phir poora fir-tree release hota hai.

**Q175** DERMATOLOGY

A farmer presented with toenail distortion and discoloration with ragged edges. A sample was taken and *Trichophyton rubrum* was identified. Which of the following statements is correct regarding *Trichophyton rubrum*?

- A *Rubrum* doesn't infect nail
- B Anthropophilic
- C Geophilic
- D Zoophilic

**Answer** Anthropophilic

*Trichophyton rubrum* is an anthropophilic dermatophyte — adapted to humans with human-to-human transmission — and is the commonest cause of onychomycosis (distal lateral subungual type) and tinea pedis/corporis. The first option is factually wrong since *T. rubrum* very commonly infects nails; geophilic species include *Microsporum gypseum* and zoophilic examples include *M. canis* and *T. verrucosum*.

💡 *T. rubrum* = insaano ka apna fungus — anthropophilic, aur nakhoon uska favourite ghar.

**Q176** DERMATOLOGY

A patient received penicillin and the following pustular sterile rash occurred (image shown). What is the diagnosis?

 Clinical photograph showing numerous small non-follicular sterile pustules on an erythematous background, characteristic of AGEP.

- A Pustular psoriasis
- B Acute generalised exanthematous pustulosis
- C Toxic epidermal necrolysis
- D Bacterial infection

**Answer** Acute generalised exanthematous pustulosis (AGEP)

AGEP is a severe cutaneous adverse drug reaction — abrupt eruption of dozens of small, sterile, non-follicular pustules on an erythematous background within days of drug exposure (aminopenicillins are classic culprits), often with fever and neutrophilia, resolving on drug withdrawal. Pustular psoriasis lacks the acute drug temporality, and TEN causes epidermal detachment with mucosal involvement and is never primarily pustular.

💡 Penicillin ke 2-4 din baad har taraf sterile pus ke dane = AGEP — drug band karo, rash khud shant ho jayega.

**Q177** DERMATOLOGY

A patient with 95% vitiligo wants to achieve a uniform skin color throughout the body. Which treatment option would be most appropriate?

- A Steroids
- B UV therapy
- C Hydroquinone
- D MBEH (Monobenzyl ether of hydroquinone)

**Answer** MBEH (Monobenzyl ether of hydroquinone)

With near-universal (>80-90% BSA) vitiligo, repigmenting therapy (steroids, phototherapy) is futile; the standard approach is to depigment the small residual islands of normal skin using 20% monobenzyl ether of hydroquinone, which permanently destroys melanocytes and gives a uniform colour. Plain hydroquinone is a reversible lightening agent used for hyperpigmentation like melasma — it cannot produce the permanent depigmentation required, which is exactly why it sits in the options as the trap.

💡 95% safed ho gaya to bache 5% ko bhi safed karo — MBEH, one-way ticket (permanent depigmentation).

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## 19 Psychiatry

6 recalled questions

### Q178 PSYCHIATRY

★ Asked directly from our Rank Booster lectures

A patient with substance abuse came to the emergency department with respiratory depression, bradycardia, hypotension, and pinpoint pupils. The immediate treatment should begin with which drug?

- A Naloxone
- B Naltrexone
- C Buprenorphine
- D Methadone

Answer **Naloxone (IV)**

Pinpoint pupils with respiratory depression and haemodynamic depression is the opioid toxidrome, and the emergency treatment is intravenous naloxone — a short-acting competitive mu-opioid antagonist that rapidly reverses respiratory depression. Naltrexone is a long-acting oral antagonist used for relapse prevention/maintenance, not acute reversal; buprenorphine and methadone are used in opioid substitution therapy and would worsen the overdose.

💡 Pinpoint pupil + soti hui saans = opioid; naloxone ABHI, naltrexone baad mein (de-addiction ke liye).

### Q179 PSYCHIATRY

★ Asked directly from our Rank Booster lectures

A 25-year-old man with bipolar disorder has been receiving lithium 900 mg per day along with quetiapine. Following an episode of gastroenteritis, he developed coarse tremor, dysarthria, and ataxia. Which of the following is the most appropriate interpretation of his clinical condition?

- A Extrapyrasidal symptom due to quetiapine
- B Lithium induced tremor occurring at therapeutic level
- C Neuroleptic malignant syndrome
- D Lithium toxicity

Answer **Lithium toxicity**

Coarse tremor with dysarthria and ataxia is the classic neurological picture of lithium toxicity — the benign side effect at therapeutic levels is a FINE tremor, so option 2 is the key trap. Volume loss from gastroenteritis increases proximal tubular sodium (and hence lithium) reabsorption, pushing levels into the toxic range even on an unchanged dose; quetiapine rarely causes EPS, and NMS requires rigidity, hyperthermia and autonomic instability, none of which are described.

💡 Lithium par fine tremor = theek hai; coarse tremor + ataxia + dysarthria = toxicity — aur dast/dehydration uska trigger.

### Q180 PSYCHIATRY

A 25-year-old male presents with a long-standing pattern of social withdrawal, odd eccentric behavior, peculiar beliefs, and magical thinking. What is the diagnosis?

Answer **Schizotypal personality disorder**

Odd/eccentric behaviour, peculiar beliefs and magical thinking with social withdrawal define schizotypal personality disorder (Cluster A). Pure schizoid personality has detachment and restricted affect WITHOUT the cognitive-perceptual oddities, and avoidant personality withdraws out of fear of rejection while desiring relationships — the magical thinking is what pins schizotypal.

💡 Loner + magical thinking = schizotypal; sirf loner = schizoid; darr ke maare loner = avoidant.

**Q181** PSYCHIATRY

A 25-year-old patient from a rural area visited a primary health physician with complaints of restlessness, low energy, fatigue, blackening in front of the eyes, and anxious irritability. He attributed these symptoms to loss of semen due to frequent masturbation and also reported whitish discharge in the urine. What is the diagnosis?

- A Amok
- B LMU
- C Dhat syndrome
- D Koro

**Answer** Dhat syndrome

Dhat syndrome is a culture-bound syndrome of the Indian subcontinent in which vague somatic and anxiety-depressive symptoms (fatigue, weakness, low energy) are attributed by the patient to loss of semen, often with complaints of whitish discharge in urine. Koro is acute panic about genital retraction and Amok is a dissociative episode of frenzied violence — neither involves semen-loss anxiety.

💡 'Dhat' gir rahi hai aur kamzori ho rahi hai — semen-loss anxiety = Dhat syndrome, India ka classic culture-bound syndrome.

**Q182** PSYCHIATRY

A 40-year-old smoker on clozapine for schizophrenia was advised to stop smoking. A few days after smoking cessation, the patient presented to the ER with a seizure. What is the most appropriate statement regarding this event?

- A Related to smoking cessation, it is incorrect
- B Related to smoking cessation and not to clozapine
- C Related to smoking cessation as it decreases the CYP1A2 levels and because of this, there is increase in the clozapine concentration

**Answer** Related to smoking cessation as it decreases CYP1A2 levels, thereby increasing the clozapine concentration (which caused the seizure)

Polycyclic aromatic hydrocarbons in cigarette smoke induce CYP1A2, the main enzyme metabolizing clozapine; on quitting, this induction is lost, CYP1A2 activity falls, clozapine clearance drops and plasma levels rise — and seizures are a well-known dose-dependent clozapine toxicity. The event is therefore linked to BOTH smoking cessation and clozapine acting together, which is why the option denying clozapine's role is wrong.

💡 Smoking CYP1A2 ka inducer hai — cigarette chhodi to clozapine chadh gayi; dose wahi, level naya.

**Q183** PSYCHIATRY

A 73-year-old man, after a total hip replacement, is mentally confused on post-operative day two, trying to remove his IV line and showing loss of concentration. Which assessment method is appropriate?

- A CAM (Confusion Assessment Method)
- B Mini-Mental State Examination (MMSE)

**Answer** CAM (Confusion Assessment Method)

Acute-onset, fluctuating confusion with inattention in an elderly post-operative patient is delirium, and its bedside screening/diagnostic tool is the Confusion Assessment Method — which operationalizes acute onset and fluctuating course, inattention, disorganized thinking, and altered consciousness. The MMSE screens for chronic cognitive impairment (dementia) and cannot distinguish delirium.

💡 Post-op buzurg IV line kheench raha hai = delirium — assessment CAM se, MMSE dementia ke liye bacha ke rakho.

## 20 Neurology

2 recalled questions

### Q184 NEUROLOGY

A patient presents with third nerve palsy due to a midbrain lesion. He exhibits a prominent tremor at rest which worsens with attempted movement. Which structure is involved?

- A Substantia nigra
- B Superior colliculus
- C Medial lemniscus
- D Red nucleus

**Answer** Red nucleus

Ipsilateral CN III palsy with contralateral coarse tremor present at rest and worsening with action (rubral/Holmes tremor) is Benedikt syndrome — a paramedian midbrain tegmentum lesion involving the red nucleus and cerebellorubral fibres. Substantia nigra lesions produce a parkinsonian rest tremor that classically improves with movement, the superior colliculus serves vertical gaze/saccades, and the medial lemniscus carries proprioception.

💡 3rd nerve palsy + jangli (rest + action) tremor = Benedikt — red nucleus ka kamaal.

### Q185 NEUROLOGY

★ Asked directly from our Rank Booster lectures

A 68-year-old man presents to the emergency department with sudden-onset right-sided hemiparesis for the past 90 minutes. There is no history of recent head trauma. A non-contrast CT (NCCT) scan of the brain is performed and is shown. What is the most likely underlying cause?

📷 NCCT brain showing a hyperdense left middle cerebral artery (dense MCA sign), concordant with the right-sided hemiparesis.

- A Acute epidural hematoma
- B Cerebral venous sinus thrombosis
- C Chronic lacunar infarct
- D Acute middle cerebral artery thrombosis

**Answer** Acute middle cerebral artery thrombosis

A hyperdense MCA sign on NCCT represents fresh thrombus within the M1 segment and is the earliest CT clue to acute MCA occlusion — fitting sudden hemiparesis within the thrombolysis window. Cerebral venous sinus thrombosis shows a dense sinus/'delta' sign, epidural hematoma needs trauma with a biconvex bleed, and a chronic lacunar infarct is a small hypodensity that would not cause a 90-minute acute deficit.

💡 Stroke window me NCCT pe chamakti MCA = thrombus dikh raha hai, infarct abhi nahi — clock chal rahi hai.

## 21 Genetics

2 recalled questions

### Q186 GENETICS

★ Asked directly from our Rank Booster lectures

A patient presents with features suggestive of Marfan syndrome - arachnodactyly, ectopia lentis, tall stature, and sternal deformity. Which gene or protein is defective?

**Answer** **Fibrillin-1 (FBN1 gene, chromosome 15)**

Marfan syndrome is caused by mutations in FBN1 (fibrillin-1) on chromosome 15q21, a structural component of extracellular microfibrils; fibrillin deficiency also de-represses TGF-beta signalling, driving the aortic and skeletal phenotype. Fibrillin-2 (FBN2) mutation causes congenital contractural arachnodactyly (Beals syndrome), and collagen defects cause EDS/OI — those appeared only as distractors in the recall.

💡 Marfan = Fifteen = Fibrillin-1: chromosome bhi 15, gene bhi FBN1.

### Q187 GENETICS

Advanced paternal age (father in his 40s) increases the risk of which type of genetic disorders?

**A** Autosomal dominant disorders

**Answer** **Autosomal dominant disorders (new/de novo mutations)**

Advancing paternal age multiplies the number of germline cell divisions in spermatogenesis, accumulating de novo point mutations, so the risk of new autosomal dominant disorders (achondroplasia, Apert syndrome, neurofibromatosis 1, MEN2) rises. Chromosomal aneuploidies such as Down syndrome track with advanced MATERNAL age - the classic trap in this question.

💡 Old father = new dominant mutation; old mother = extra chromosome.

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## 22 Immunology

1 recalled questions

### Q188 IMMUNOLOGY

★ Asked directly from our Rank Booster lectures

A tumor cell does not express MHC class I molecules. Despite the presence of tumor-associated antigens, cytotoxic T lymphocytes (CTLs) are unable to recognize the tumor cells. What is the underlying reason?

**A** Failure of antigen presentation through MHC class I

**B** Co-stimulatory molecules

**Answer** Failure of antigen presentation through MHC class I

CD8+ CTLs recognize antigenic peptides only when presented on MHC class I; tumors that down-regulate MHC I therefore become invisible to CTLs — a classic immune-evasion mechanism (though such 'missing self' cells become targets for NK cells). Loss of co-stimulation (B7-CD28) impairs priming of naive T cells but is not the mechanism when the defect described is absent MHC I.

💡 MHC I gayab → CD8 andha; tab 'missing self' dekh kar NK cell hi tumor pakadti hai.

## 23 What The Examiner Was Really Testing

Everyone walks out of the exam hall arguing about individual questions. That is the wrong argument. When you lay all 188 unique recalled questions side by side, the examiner's hand becomes visible — the same testing instincts repeat again and again. These are the patterns that actually show up in this paper's data, with the question numbers to prove it.

### 1. One in three questions had an image — and some subjects were image-only

67 of the 188 unique questions (36%) hinged on a visual: ECGs (Q144, Q185), X-rays (Q112, Q164, Q168), OCT scans (Q157), audiograms (Q155), capnography traces (Q171, Q172), gross specimens (Q117, Q118), histology (Q33, Q37), DEXA reports (Q19), even a statistics scatter diagram (Q70). Ophthalmology was the extreme case — all 7 of its questions (Q156–Q162) were image-based, and Orthopedics ran 3 out of 4. If your image atlas practice is weak, you are effectively writing a 120-question paper while everyone else writes the full 188.

**Next attempt:** Treat image banks as a separate subject with its own revision schedule — one dedicated pass per week, not a casual scroll the night before.

### 2. Basic sciences were asked at the bedside, not from the textbook index

Barely a quarter of the paper was direct one-liners. Anatomy came dressed as patients — Brown-Séquard syndrome as a case (Q8), LAD occlusion on angiography (Q6), a third-nerve palsy with contralateral chorea for midbrain localization (Q184). Physiology arrived as a head-injury patient drinking 8 litres a day (Q12) and a hemochromatosis workup (Q13); Biochemistry as post-bariatric neuropathy (Q14) and X-linked urea cycle defect (Q16). The examiner is checking whether your first-year knowledge survived contact with a patient.

**Next attempt:** For every basic-science fact you revise, force one clinical hook — which patient walks in with this? If you can't answer that, you haven't finished learning it.

### 3. The paper repeats its own themes — one prepared topic paid two or three times

This is the single most exploitable pattern. Pulled elbow appeared in Anatomy (Q163) and again in Orthopedics (Q163). Spinal accessory nerve injury after posterior-triangle surgery: Q3 and Q3. Cord hemisection: Q8, Q8 and Q8 — three times. Opioid overdose: Pharmacology (Q178), Forensic (Q178) and Psychiatry (Q178). Marfan syndrome: Q186, Q186, Q186. Hemophilia carrier genetics: Q142 and Q142. Scrub typhus eschar: Q75 and Q75. Fluorosis in a Rajasthan patient: Q19 and Q19. Barrett's esophagus: pathology of the metaplasia (Q40) and management of dysplasia (Q113). Retinoblastoma rosettes: Q37 and Q37. One topic, prepared properly once, was quietly worth 2–3 marks.

**Next attempt:** Revise integratively by theme, not by subject silo. When you study a condition, deliberately cover its anatomy, pathology, management and forensic/PSM angles in the same sitting.

### 4. The hypokalemia-metabolic alkalosis cluster: one chart, repeated marks

Bartter syndrome localized to the NKCC2 transporter in the thick ascending limb (Q141) — recalled in three different versions by different students, so it clearly left a mark — primary aldosteronism worked up all the way to adrenal vein sampling (Q94), and hypokalemia refractory to correction because of hypomagnesemia (Q99). Multiple marks sitting inside one nephron diagram — transporter, segment, and the differentiating labs. Yeh koi coincidence nahi hai; somebody on the question-setting panel loves the renal tubule.

**Next attempt:** Master the nephron channel-by-channel — Bartter vs Gitelman vs Liddle vs Conn, with urinary calcium and magnesium as the deciders. This cluster will return.

### 5. Mechanism questions, not fact questions

The examiner repeatedly refused to accept name-recall and demanded the "how." Diphtheria toxin's mechanism (Q48), cholera vs pertussis toxin at the level of Gs and Gi ADP-ribosylation (Q55, Q56), penicillin hemolysis as a hapten mechanism (Q22), spironolactone gynecomastia at receptor level (Q26), OHSS as an activating FSH-receptor mutation (Q128), Laron dwarfism as a GH-receptor defect (Q146), and tumor

immune evasion through lost MHC class I (Q188). Knowing the association gets you to the stem; knowing the mechanism gets you to the answer.

**Next attempt:** For every toxin, drug reaction, and endocrine syndrome, be able to state the mechanism in one sentence at the receptor/enzyme level. If your note says only "causes X," it is incomplete.

## 6. The "first-line failed — now what?" pharmacology template

A striking number of pharmacology and therapeutics questions started after the standard drug had already failed or was contraindicated: metronidazole-resistant giardiasis → nitazoxanide (Q23), opioid-induced constipation on morphine → alvimopan (Q25), octreotide failure in acromegaly → pegvisomant (Q90), transplant-ineligible aplastic anemia → ATG + cyclosporine + eltrombopag (Q92), the fastest uric-acid-lowering agent in tumor lysis → rasburicase (Q88), and potassium that won't correct until you fix the magnesium (Q99). First-line answers were free marks nobody was offered.

**Next attempt:** For every major condition, learn the second-line and rescue drug with the same seriousness as the first-line. That is where this paper lived.

## 7. One adjective in the stem decided the answer

"Enrichment medium for rapid early growth" made Loeffler's correct and tellurite agar wrong (Q49).

"Reference method / gold standard" for EHEC made the Vero cell assay correct over sorbitol MacConkey culture (Q57). "Most likely to be a CARRIER" made the mother correct — read "most likely affected" and you land on maternal uncle (Q142). "Attributable risk PROPORTION" made 90% correct while the absolute risk difference of 0.009 sat there as a trap (Q69). Flat high-grade dysplasia in Barrett's made RFA correct over EMR (Q113). None of these were knowledge gaps — they were reading tests, set deliberately for skimmers under time pressure.

**Next attempt:** Train yourself to underline the qualifier — gold standard, screening, enrichment, carrier, proportion, initial, definitive — before looking at options. The options are written to punish skimmers.

## 8. Recent-guideline management was tested deliberately

Several answers are only correct in the current guideline era: the XDR-TB definition requiring fluoroquinolone plus Group A drug resistance (Q78), MTP beyond 24 weeks needing a state Medical Board (Q60 — the amended MTP Act), vericiguat's soluble guanylate cyclase mechanism for recently hospitalized HFrEF (Q27), balloon angioplasty without stenting for fibromuscular dysplasia (Q100), radiofrequency ablation for flat Barrett's HGD (Q113), transcatheter device closure for PDA (Q147), and 39-week induction timing in well-controlled GDM (Q124). An old edition of your notes actively costs marks here.

**Next attempt:** Do a dedicated "what changed in the last 3 years" pass — TB definitions and regimens, MTP Act, heart failure drugs, endoscopic Barrett's therapy — rather than trusting whichever year your notes were written.

## 9. PSM went quantitative — calculation under time pressure

PSM contributed 15 questions and a disproportionate share of the arithmetic: case fatality rate (Q68), attributable risk proportion (Q69), correlation strength read off scatter in the data (Q70, Q73), and an overcrowding problem (Q76) whose ages were chosen with surgical intent — a child of 9 years 10 months counts as half a unit AND triggers the sex-separation criterion, while a 10-month-old counts as zero. The examiner wasn't testing whether you know the formula; he was testing whether you apply the counting rules correctly when the numbers are designed to trip you.

**Next attempt:** Practice PSM numericals with a timer, and memorize the counting conventions (who counts as half, who counts as zero, per-room and floor-space standards) — the formula alone will not save you.

## Agla paper naya hoga. Examiner ki aadatein nahi.

91 questions is paper mein seedha humare lectures se the — more than 50% of the paper. Wahi examiner patterns jin par **Rank Booster** — sirf 60 hours ka rapid revision course, at a very nominal price — poora built hai.

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