FMGE Sample Paper – 1

Complete Full-Length Practice Paper

Duration: 210 Minutes **Maximum Marks: 300**

Instructions

- This paper contains **300 Multiple Choice Questions (Single Correct Answer)** modelled on the complete **Foreign Medical Graduate Examination (FMGE)**.
- Each correct answer carries **+1 mark**. There is **no negative marking** — attempt all questions.
- Questions follow the clinically integrated pattern of the FMGE examination, covering all 19 medical subjects as per the National Medical Commission (NMC) syllabus.
- The paper is organised into four sections: **Section A** (Pre-clinical Sciences, Q1–50), **Section B** (Para-clinical Sciences, Q51–120), **Section C** (Clinical Medicine, Q121–200), and **Section D** (Clinical Surgery & Specialties, Q201–300).
- Use of mobile phones, calculators, or electronic gadgets is strictly prohibited. Rough paper will be provided at the examination centre.
- Click any question number in the Answer Key to navigate to its detailed solution.

Section A — Pre-clinical Sciences (Q 1–50)

Q1. A 42-year-old construction worker presents with pain and paraesthesia along the medial aspect of his forearm and the medial one-and-a-half fingers of the right hand following a crush injury to the upper limb. Sensory testing confirms loss of light touch in that distribution. Which nerve root level is **MOST likely** responsible for this dermatomal pattern?

(A) C8–T1

(B) C6–C7

(C) C5–C6

(D) T2–T3

Q2. A 29-year-old woman undergoes a lymph node biopsy in the posterior triangle of the neck. Two weeks post-operatively she is unable to abduct her right shoulder beyond 90° and the scapula wings when she pushes against a wall. Which nerve was **MOST likely** injured during the procedure?

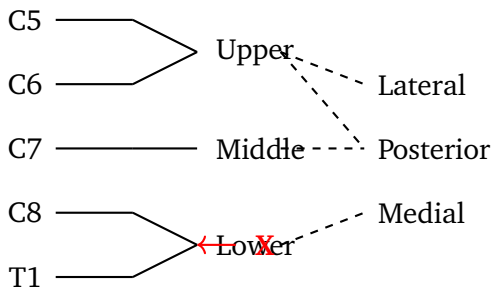
(A) Phrenic nerve

(B) Accessory nerve (CN XI)

(C) Greater auricular nerve

(D) Dorsal scapular nerve

Q3. A 25-year-old motorcyclist is brought to the emergency department after a high-speed collision. He has complete sensory and motor loss in the entire upper limb on the left side. Imaging shows avulsion of nerve roots. The diagram below represents the brachial plexus.



The point marked **X** indicates avulsion of which trunk, and which named terminal branches originate exclusively from the roots contributing to this trunk?

(A) Lower trunk; medial pectoral and medial cutaneous nerves of arm and forearm

(B) Upper trunk; suprascapular and nerve to subclavius

(C) Middle trunk; no named branches arise exclusively from it

(D) Lower trunk; long thoracic and dorsal scapular nerves

Q4. A 67-year-old hypertensive man is brought unconscious to the emergency room. CT scan reveals a haemorrhage in the territory of the lenticulostriate arteries. Which major cerebral artery gives rise to these perforating vessels?

(A) Posterior cerebral artery

(B) Anterior cerebral artery

(C) Middle cerebral artery

(D) Basilar artery

Q5. During a cadaveric dissection, a student identifies the artery running in the posterior interventricular groove. Occlusion of this vessel **MOST commonly** leads to infarction of which region?

(A) Anterior wall of right ventricle

(B) Lateral wall of left ventricle

(C) Anterior two-thirds of the interventricular septum

(D) Posterior one-third of the interventricular septum and posterior wall of the left ventricle

Q6. A 45-year-old patient undergoing elective nephrectomy is found to have a polar renal artery arising directly from the aorta supplying the upper pole of the left kidney. The surgeon inadvertently ligates this vessel. Which of the following statements about renal segmental arteries is **MOST accurate**?

(A) They are end-arteries; ligation causes infarction of the corresponding segment

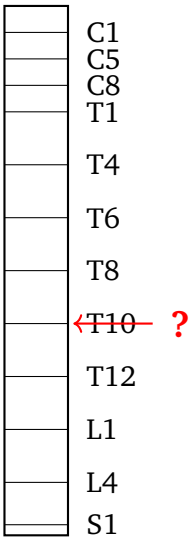
(B) They anastomose freely at the cortex, so ligation is inconsequential

(C) They run only in the renal sinus, not entering the parenchyma

(D) They arise exclusively from the renal vein tributaries

Q7. A 55-year-old man presents with a band-like burning pain around his trunk at the level of the umbilicus following a herpes zoster eruption.

The diagram below shows a schematic of the spinal column with vertebral levels marked.



The arrow marked **?** points to the spinal level that **MOST likely** corresponds to the dermatomal level of the umbilicus. Which level is this?

(A) T6

(B) T8

(C) T10

(D) T12

Q8. A 20-year-old rugby player sustains a severe direct blow to the lateral aspect of his right leg. Four hours later he complains of excruciating pain in the leg that is disproportionate to the injury and worsens on passive dorsiflexion of the toes. Compartment syndrome is suspected. Which compartment of the leg contains the deep peroneal nerve and the anterior tibial artery?

(A) Lateral compartment

(B) Anterior compartment

(C) Superficial posterior compartment

(D) Deep posterior compartment

Q9. A 52-year-old woman is diagnosed with carcinoma of the lateral quadrant of her right breast. During staging workup, sentinel lymph node

biopsy is planned. To which group of lymph nodes does **MOST** (approximately 75%) of the lymphatic drainage of the breast primarily drain?

(A) Internal mammary (parasternal) nodes

(B) Supraclavicular nodes

(C) Deltopectoral (infraclavicular) nodes

(D) Anterior (pectoral) axillary nodes

Q10. A 78-year-old woman sustains a displaced intracapsular fracture of the neck of the femur after a fall. Her orthopaedic surgeon explains that avascular necrosis of the femoral head is a major complication. The primary blood supply to the adult femoral head is derived from which source?

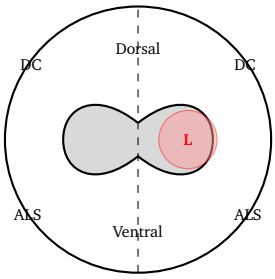
(A) Retinacular branches of the medial circumflex femoral artery

(B) Obturator artery via the ligamentum teres

(C) Lateral circumflex femoral artery

(D) Inferior gluteal artery

Q11. A 34-year-old man presents with loss of pain and temperature sensation on the right side of the body below T6, and loss of fine touch and proprioception on the left side below T6, following a penetrating injury. The diagram below represents a transverse section of the spinal cord.



DC = dorsal columns; ALS = anterolateral system; L = lesion site. This injury pattern is **MOST consistent** with which syndrome?

(A) Central cord syndrome

(B) Anterior cord syndrome

(C) Brown-Séquard syndrome

(D) Posterior cord syndrome

Q12. A 60-year-old man presents with a groin swelling that appears **medial** to the inferior epigastric vessels and passes through the superficial inguinal ring. It is reducible and has an expansile cough impulse. Which of the following best describes this hernia?

(A) Indirect inguinal hernia passing through the deep inguinal ring lateral to the inferior epigastric vessels

(B) Femoral hernia passing through the femoral canal below the inguinal ligament

(C) Spigelian hernia through the linea semilunaris

(D) Direct inguinal hernia through Hesselbach's triangle

Q13. A 35-year-old patient with a base of skull fracture has clinical signs of a lesion at the jugular foramen. Which combination of cranial nerves would **MOST likely** be affected?

(A) CN IX, CN X, CN XI

(B) CN III, CN IV, CN VI

(C) CN V1, CN V2, CN VI

(D) CN VII, CN VIII

Q14. A neonate is found to have malrotation of the midgut during exploratory laparotomy for volvulus. During normal embryological development, the physiological herniation of the midgut through the umbilical ring occurs in the **SIXTH week**. When the gut returns to the abdominal cavity, it undergoes counterclockwise rotation of how many degrees (as viewed from the front)?

(A) 90°

(B) 270°

(C) 180°

(D) 360°

Q15. A pathology resident is examining a renal biopsy specimen under light microscopy. She identifies tall columnar cells with a brush border, abundant mitochondria, and no visible lumen in cross-section due to complex interdigitating basolateral processes. These features are **MOST characteristic** of which segment of the nephron?

(A) Collecting duct

(B) Descending thin limb of loop of Henle

(C) Proximal convoluted tubule

(D) Glomerular parietal epithelium

Q16. A 48-year-old man with cirrhosis develops haematemesis. Upper GI endoscopy reveals oesophageal varices at the lower end of the oesophagus. This is due to a portal-systemic anastomosis between the left gastric (portal) and the oesophageal tributaries of the azygos vein (systemic). Which additional site of clinically important porto-systemic anastomosis can produce *caput medusae*?

(A) Lower rectum between superior and inferior rectal veins

(B) Bare area of liver between portal tributaries and phrenic veins

(C) Retroperitoneum via veins of Retzius

(D) Para-umbilical veins (portal) anastomosing with superficial epigastric veins (systemic) at the falciform ligament

Q17. A 40-year-old man develops sudden onset of complete unilateral facial weakness involving the forehead, eye, cheek, and mouth on the right side following a viral illness. He cannot close his right eye and has hyperacusis. Which of the following **BEST** explains the hyperacusis in this patient?

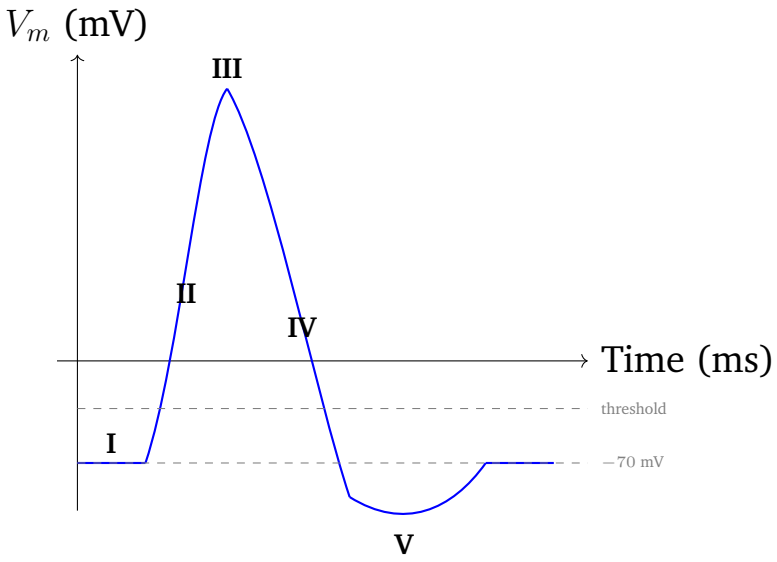
(A) Loss of the greater petrosal nerve branch affecting the lacrimal gland

(B) Paralysis of stapedius muscle, which normally damps loud sounds

(C) Damage to the chorda tympani reducing taste on the anterior two-thirds of the tongue

(D) Involvement of the auriculotemporal branch of the mandibular nerve

Q18. A physiology student is studying a neuronal action potential trace obtained by intracellular recording. The diagram below shows the voltage change over time, with five labelled phases.



During phase **III** (the peak of the action potential), which ion channel state and ion movement **BEST** describe the membrane condition?

(A) Voltage-gated Na^+ channels open; rapid Na^+ influx drives the membrane to $+30$ to $+40$ mV

(B) Voltage-gated K^+ channels open; K^+ efflux causes repolarisation

(C) Na^+–K^+ ATPase actively pumps Na^+ out; restoring resting potential

(D) Voltage-gated Na^+ channels are inactivated; membrane is in absolute refractory period

Q19. A pharmacology researcher applies a drug that selectively blocks voltage-gated K^+ channels at rest in a cardiac myocyte. Which of the following **BEST** describes the immediate effect on the resting membrane potential (RMP)?

(A) RMP becomes more negative (hyperpolarisation) because Na^+ influx increases

(B) RMP becomes less negative (depolarisation) because the outward K^+ current maintaining RMP is lost

(C) RMP remains unchanged because the Na^+–K^+ ATPase compensates immediately

(D) RMP becomes more negative due to increased Cl^- conductance

Q20. A 30-year-old medical student is interpreting a Wiggers diagram in a physiology exam. He notes that at the moment the mitral valve **closes**, the aortic valve is also **closed** and the left ventricular pressure is **rising rapidly**. Which phase of the cardiac cycle does this describe?

(A) Rapid ventricular filling

(B) Isovolumetric relaxation

(C) Isovolumetric contraction

(D) Rapid ejection

Q21. During a cardiac catheterisation study in a 55-year-old woman, the following values are recorded: O_2 consumption = 250 mL/min; pulmonary artery O_2 content = 150 mL/L; pulmonary vein O_2 content = 200 mL/L. Using the Fick principle, what is the cardiac output?

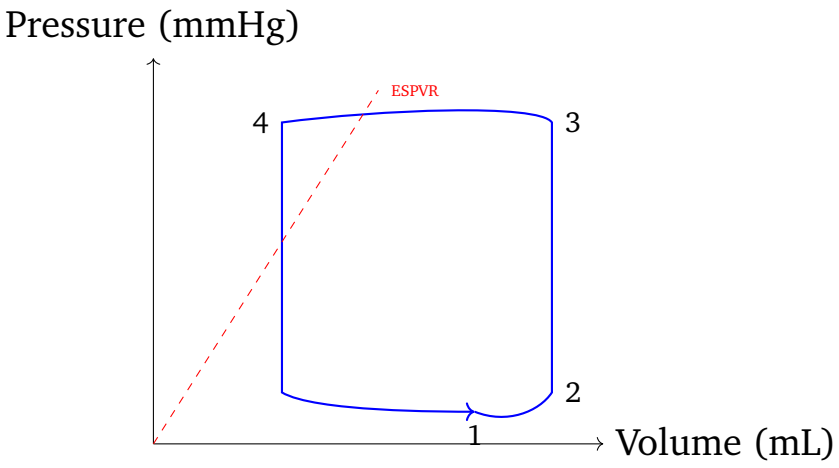
(A) 3.5 L/min

(B) 4.0 L/min

(C) 4.5 L/min

(D) 5.0 L/min

Q22. The diagram below shows a left ventricular pressure-volume (PV) loop for a healthy adult at rest. Four corners are labelled 1–4.



The segment from point **2** to point **3** represents which phase, and which valves are open at this time?

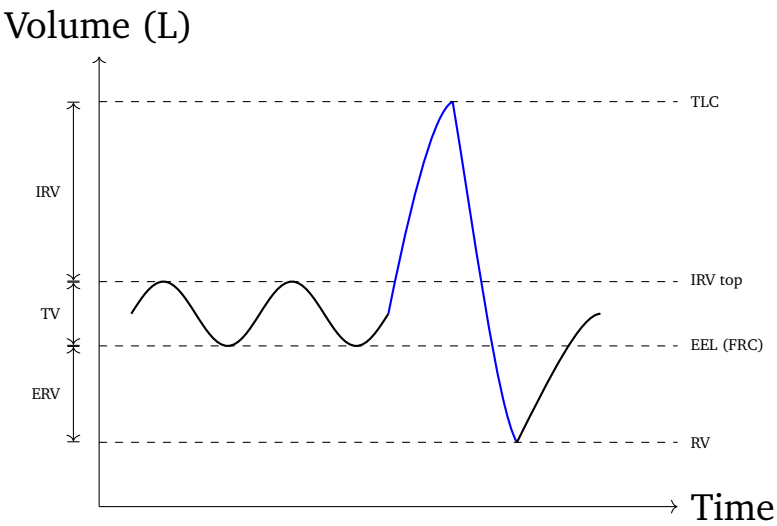
(A) Isovolumetric contraction; both mitral and aortic valves closed

(B) Rapid ejection; aortic valve open, mitral valve closed

(C) Isovolumetric relaxation; both valves closed

(D) Rapid ventricular filling; mitral open, aortic closed

Q23. A 65-year-old long-term smoker performs spirometry. The trace below shows a schematic tidal and forced breathing manoeuvre with lung volumes labelled.



In a patient with obstructive lung disease, which **combination** of spirometric values is **MOST** characteristic?

(A) Decreased TLC, decreased FRC, normal RV

(B) Decreased FEV_1/FVC ratio with decreased TLC

(C) Decreased FEV_1/FVC ratio with increased RV and FRC (air trapping)

(D) Normal FEV_1/FVC ratio with decreased FVC and decreased TLC

Q24. A 50-year-old hypertensive patient is started on an ACE inhibitor. His serum creatinine rises modestly. The mechanism of GFR maintenance in the kidney involves myogenic autoregulation and tubuloglomerular feedback. Which of the following **BEST** explains the rise in creatinine in this patient on ACE inhibition?

(A) Afferent arteriolar vasoconstriction reduces renal plasma flow

(B) Efferent arteriolar dilation reduces glomerular hydrostatic pressure and therefore GFR

(C) Increased aldosterone causes sodium retention, expanding plasma volume and diluting creatinine

(D) Mesangial cell contraction reduces filtration surface area in response to elevated angiotensin II

Q25. A 22-year-old woman presents with glycosuria but a fasting blood glucose of 5.2 mmol/L (normal). Urine glucose is consistently positive. Genetic testing confirms a mutation in the SGLT2 transporter in the proximal tubule. Which physiological concept **BEST** explains why glucose normally does not appear in the urine up to a plasma glucose of approximately 180 mg/dL?

(A) Glucose is freely filtered but entirely reabsorbed by passive diffusion in the loop of Henle

(B) Glucose reabsorption is driven solely by the concentration gradient with no transporter involvement

(C) The tubular transport maximum (T_m) for glucose is not exceeded below approximately 180 mg/dL; all filtered glucose is reabsorbed

(D) The glomerular basement membrane is impermeable to glucose below 180 mg/dL

Q26. A 3-day-old neonate is noted to be jaundiced. Total serum bilirubin is 15 mg/dL, predominantly unconjugated. The neonate is feeding well and has no haemolysis. Which enzyme is responsible for conjugating bilirubin to bilirubin glucuronide in hepatocytes, and why is its activity transiently reduced in neonatal physiological jaundice?

(A) Biliverdin reductase; reduced due to low haem oxygenase activity

(B) Alanine aminotransferase; reduced due to immature hepatic blood flow

(C) Alkaline phosphatase; reduced because the canalicular membrane is underdeveloped

(D) UDP-glucuronosyltransferase (UGT1A1); transiently low expression in the neonatal liver

Q27. A 28-year-old woman is being investigated for infertility. Her menstrual cycle is 28 days. A urine LH kit detects a surge on day 13. Which of the following **BEST** describes the hormonal event that triggers the LH surge?

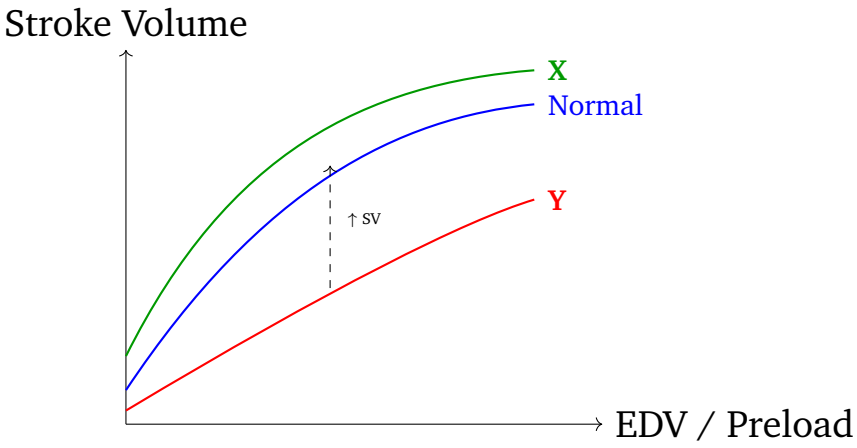
(A) Rising oestradiol from the dominant follicle switches the hypothalamo-pituitary axis from negative to positive feedback, triggering GnRH pulse amplification and the LH surge

(B) A sudden fall in progesterone from the corpus luteum removes negative feedback, allowing FSH to surge

(C) Rising inhibin B from granulosa cells directly stimulates pituitary LH release

(D) A rise in prolactin from the anterior pituitary stimulates ovarian LH receptors to trigger ovulation

Q28. The diagram below shows Frank-Starling curves for the heart under different conditions.



Curve **X** is shifted upward relative to the normal curve. Which physiological or pharmacological state **BEST** explains this shift?

(A) Administration of a beta-blocker reducing heart rate and contractility

(B) Administration of dobutamine, a positive inotrope increasing myocardial contractility

(C) Acute haemorrhage reducing venous return and preload

(D) Cardiac tamponade limiting ventricular filling

Q29. A 22-year-old male marathon runner collapses at the finish line. His rectal temperature is 40.5°C. In the normal temperature regulation response to hyperthermia, which of the following **CORRECTLY** describes the effector responses coordinated by the hypothalamic thermostat?

(A) Cutaneous vasoconstriction to conserve heat; piloerection; shivering thermogenesis

(B) Reduced metabolic rate in skeletal muscle; increased GI absorption of water

(C) Cutaneous vasodilation; increased sweating; reduced skeletal muscle tone

(D) Increased ADH release to reduce urinary free water loss; peripheral vasoconstriction

Q30. A 45-year-old patient of blood group A (subgroup A_1) requires an urgent blood transfusion. Cross-matching is performed. A person with blood group **O** is known as a "universal donor" for red cell transfusions in emergency settings. Which of the following **CORRECTLY** explains why group O red cells cause less haemolytic risk than other ABO-incompatible transfusions?

(A) Group O individuals have the H antigen; anti-H antibodies in recipients destroy O red cells slowly

(B) Group O red cells carry both A and B antigens at low density

(C) Group O plasma contains anti-A and anti-B antibodies that are neutralised by recipient red cells

(D) Group O red cells lack A and B surface antigens, so the recipient's circulating anti-A or anti-B antibodies cannot bind and activate complement on transfused cells

Q31. A neurosurgery resident is asked about the composition and flow of cerebrospinal fluid. A 60-year-old patient with normal pressure hydrocephalus has impaired CSF reabsorption. CSF is primarily produced by which structure, and what is the approximate daily production rate?

(A) Choroid plexus of the lateral, third, and fourth ventricles; approximately 500 mL/day

(B) Ependymal lining of the cerebral cortex; approximately 100 mL/day

(C) Arachnoid granulations; approximately 250 mL/day

(D) Dural venous sinuses; approximately 50 mL/day

Q32. A 70-year-old patient with chronic obstructive pulmonary disease (COPD) has a PaO_2 of 55 mmHg, $PaCO_2$ of 55 mmHg, and a pH of 7.32. In the context of the haemoglobin-oxygen dissociation curve, a **right shift** (decreased O_2 affinity) occurs due to which combination of factors present in this patient?

(A) Decreased CO_2, alkalosis, and decreased temperature

(B) Increased CO_2, acidosis (Bohr effect), and increased 2,3-DPG

(C) Increased CO_2 alone; pH and 2,3-DPG are not relevant to the curve

(D) Decreased 2,3-DPG due to chronic hypoxia shifting the curve leftward

Q33. A 35-year-old man is admitted with polyuria and polydipsia. Water deprivation test shows urine osmolality of 150 mOsm/kg even after 8 hours of fluid deprivation. After administration of desmopressin (ADH analogue), urine osmolality rises to 650 mOsm/kg. Which diagnosis is **MOST consistent**?

(A) Primary (psychogenic) polydipsia

(B) Nephrogenic diabetes insipidus

(C) Central (cranial) diabetes insipidus

(D) Type 2 diabetes mellitus with osmotic diuresis

Q34. A 68-year-old patient with sick sinus syndrome undergoes electrophysiology testing. The pacemaker potential (phase 4 depolarisation) of the sinoatrial node differs from that of a ventricular myocyte primarily because SA node phase 4 is driven by which current?

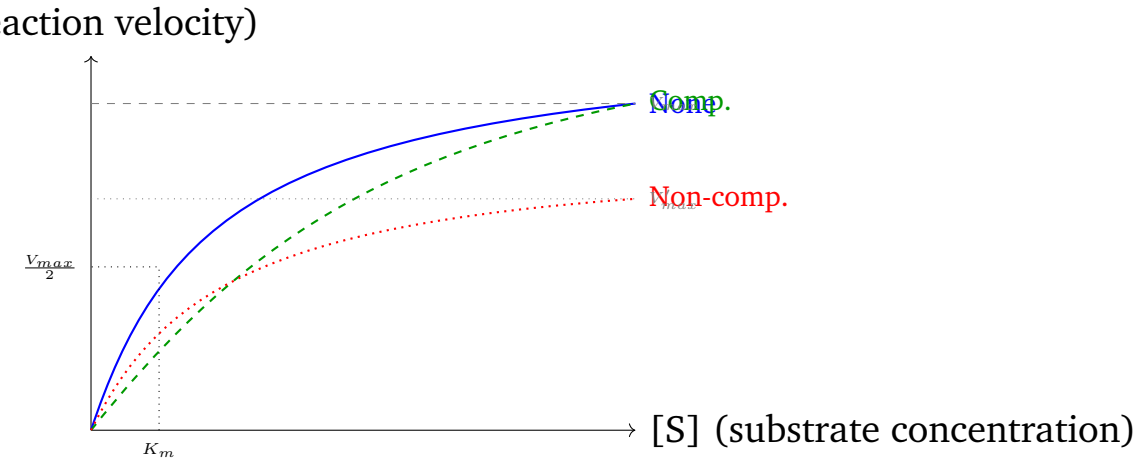
(A) A large inward Na^+ current (fast Na^+ channels, I_{Na}) identical to that seen in ventricular cells

(B) Outward K^+ current (I_K) creating spontaneous depolarisation

(C) Inward Ca^{2+} current through L-type channels alone, without any funny current

(D) The funny current (I_f, a mixed Na^+/K^+ inward current) combined with declining I_K and a T-type Ca^{2+} inward current

Q35. The diagram below shows a Michaelis-Menten velocity curve for an enzyme under three conditions: no inhibitor, competitive inhibitor, and non-competitive inhibitor.



A kinase inhibitor drug acts as a competitive inhibitor of a target enzyme. Adding excess substrate **MOST likely** produces which outcome?

(A) The reaction velocity approaches the same V_{max} as the uninhibited enzyme because competitive inhibition is reversible and overcome by high [S]

(B) The reaction velocity is permanently reduced below V_{max} regardless of [S]

(C) V_{max} is reduced but K_m is unchanged

(D) The enzyme is irreversibly inactivated; adding substrate has no effect

Q36. A 24-year-old biochemistry student is asked to calculate the net ATP yield from the complete aerobic oxidation of one molecule of glucose. She knows that glycolysis generates 2 ATP (net) and 2 NADH in the cytoplasm, the pyruvate dehydrogenase step generates 2 NADH, and the TCA cycle generates 6 NADH, 2 $FADH_2$, and 2 GTP per glucose. Using the P/O ratios of 2.5 for NADH and 1.5 for $FADH_2$, what is the **MOST accurate** total ATP yield per glucose?

(A) 36–38 ATP (classic textbook value using P/O of 3 for NADH, 2 for $FADH_2$)

(B) Approximately 30–32 ATP using modern P/O ratios of 2.5 and 1.5

(C) 18 ATP (counting only substrate-level phosphorylation)

(D) 40 ATP (including energy from membrane potential alone)

Q37. A patient with severe thiamine (vitamin B_1) deficiency presents with neurological symptoms and lactic acidosis. Thiamine pyrophosphate (TPP) is a cofactor for multiple enzymes in intermediary metabolism. Which of the following enzymes is **NOT** dependent on TPP?

(A) Pyruvate dehydrogenase

(B) α-Ketoglutarate dehydrogenase

(C) Pyruvate carboxylase

(D) Transketolase (pentose phosphate pathway)

Q38. A 10-day-old neonate presents with lethargy, vomiting, and hyperammonaemia following protein feeding. Plasma amino acid analysis shows elevated citrulline. Urine orotic acid is increased. Argininosuccinate synthetase deficiency (citrullinaemia type I) is diagnosed. Which of the following **BEST** explains the elevated orotic acid in urea cycle defects?

(A) Decreased arginine synthesis reduces feedback inhibition of ornithine transcarbamylase; carbamoyl phosphate diverts into the pyrimidine synthesis pathway producing orotic acid

(B) Excess orotic acid is produced from arginine catabolism in the liver

(C) Orotic acid accumulates because the urea cycle consumes it as an intermediate

(D) Carbamoyl phosphate accumulates in the mitochondria and spills into the cytosol, where it enters the pyrimidine synthesis pathway and is converted to orotic acid

Q39. A nutritionist explains to a medical student the key differences between fatty acid *synthesis* and fatty acid *oxidation* (β-oxidation). Which of the following statements correctly identifies a difference between these two pathways?

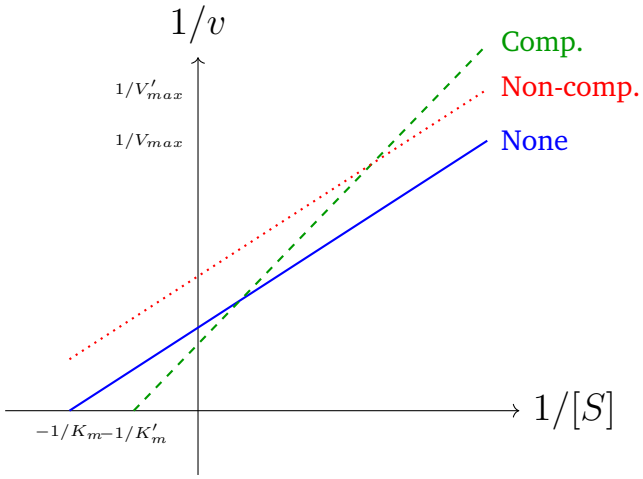
(A) Fatty acid synthesis occurs in the cytoplasm and uses NADPH; β-oxidation occurs in the mitochondrial matrix and generates NADH and $FADH_2$

(B) Both processes occur in the mitochondrial matrix and use the same carrier proteins

(C) Fatty acid synthesis uses FAD and NAD^+ as cofactors; β-oxidation uses NADPH

(D) β-Oxidation is the rate-limiting step for cholesterol biosynthesis

Q40. The Lineweaver-Burk (double-reciprocal) plots below compare uninhibited enzyme kinetics with two inhibitor conditions.



Based on the Lineweaver-Burk plot, non-competitive inhibition is identified when which feature is observed?

(A) The x-intercept shifts to the right (less negative) but the y-intercept is unchanged

(B) Both the x-intercept and y-intercept shift, indicating changes in both K_m and V_{max}

(C) The y-intercept increases (higher $1/V_{max}$) while the x-intercept remains unchanged (same $-1/K_m$), indicating reduced V_{max} with unchanged K_m

(D) The slope decreases and lines converge at the y-axis

Q41. A 55-year-old man with gout is started on allopurinol, which inhibits xanthine oxidase. A rare enzyme deficiency — hypoxanthine-guanine phosphoribosyltransferase (HGPRT) — is responsible for Lesch-Nyhan syndrome. HGPRT is critical for which metabolic pathway?

(A) De novo purine synthesis from PRPP and glutamine

(B) The purine salvage pathway, converting hypoxanthine and guanine to IMP and GMP using PRPP

(C) De novo pyrimidine synthesis via carbamoyl phosphate

(D) The folate cycle for thymidylate synthesis

Q42. A 40-year-old alcoholic man presents with the classic triad of dermatitis, diarrhoea, and dementia (Casal's necklace rash). Pellagra caused by niacin (vitamin B_3) deficiency is diagnosed. Niacin is a precursor of which coenzyme critical for oxidation-reduction reactions in glycolysis and the TCA cycle?

(A) NAD^+ and $NADP^+$

(B) Coenzyme A (CoA)

(C) FAD and FMN

(D) Thiamine pyrophosphate (TPP)

Q43. A 30-year-old woman with a strong family history of colorectal cancer is found to carry a germline mutation in the *MLH1* gene, which encodes a mismatch repair protein. The mismatch repair (MMR) system corrects errors introduced during DNA replication. Which of the following **BEST** describes the biochemical basis of MMR?

(A) MMR removes UV-induced thymine dimers by nucleotide excision

(B) MMR corrects double-strand breaks using homologous recombination with the sister chromatid

(C) MMR recognises and repairs base mismatches and small insertion-deletion loops left after DNA polymerase errors, using strand discrimination signals (e.g., hemi-methylation in bacteria; PCNA-based in humans)

(D) MMR operates only in mitochondrial DNA because nuclear DNA uses base excision repair exclusively

Q44. A 55-year-old woman is found to have a BRCA1 mutation. BRCA1 is classified as a tumour suppressor gene and follows the "two-hit" hypothesis. Which of the following is the **MOST accurate** statement about oncogenes versus tumour suppressor genes?

(A) Oncogenes require mutation of both alleles (loss of function) to promote tumorigenesis

(B) Oncogenes are gain-of-function mutations; one mutant allele is sufficient (dominant); tumour suppressors are loss-of-function and require both alleles to be inactivated

(C) Tumour suppressor genes promote cell growth; their overexpression causes cancer

(D) Both oncogenes and tumour suppressors are always inherited through the germline

Q45. A 55-year-old patient with nephrotic syndrome has a serum albumin of 1.8 g/dL (normal 3.5–5.0 g/dL). He develops bilateral pitting oedema. Besides its oncotic role, albumin performs several important physiological functions. Which of the following is **NOT** a function of albumin?

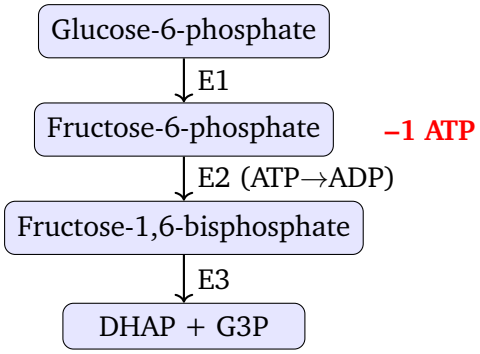
(A) Binding and transport of unconjugated bilirubin

(B) Binding and transport of free fatty acids

(C) Maintenance of plasma colloid osmotic pressure

(D) Synthesis of acute-phase proteins such as C-reactive protein and fibrinogen

Q46. The diagram below shows three consecutive steps of glycolysis, with enzymes labelled E1, E2, and E3.



Enzyme **E2** (which consumes one ATP and is a key regulatory step) is the enzyme that is **MOST significantly** allosterically inhibited by ATP and citrate, and activated by AMP and fructose-2,6-bisphosphate. What is the identity of E2?

(A) Phosphofructokinase-1 (PFK-1)

(B) Hexokinase

(C) Phosphoglucose isomerase

(D) Pyruvate kinase

Q47. A 19-year-old patient with type 1 diabetes presents in diabetic ketoacidosis (DKA). His arterial blood gas shows pH 7.18, HCO_3^- 8 mEq/L, and blood glucose 450 mg/dL. Serum ketones are markedly elevated. The rate-limiting enzyme of ketogenesis in hepatocyte mitochondria is:

(A) Acetyl-CoA carboxylase

(B) Acyl-CoA dehydrogenase

(C) HMG-CoA synthase (mitochondrial)

(D) HMG-CoA lyase

Q48. A 65-year-old housebound woman presents with proximal muscle weakness and bone pain. Her 25-hydroxyvitamin D level is 12 ng/mL (severely deficient). Vitamin D must undergo two hydroxylation steps before becoming biologically active calcitriol. Which of the following **CORRECTLY** describes the sequential hydroxylations?

(A) First hydroxylation at position 1 in the kidney; second hydroxylation at position 25 in the liver

(B) First hydroxylation at position 25 in the liver; second hydroxylation at position 1 in the kidney (by 1α-hydroxylase, stimulated by PTH)

(C) Both hydroxylations occur in the kidney under the control of calcitonin

(D) First hydroxylation at position 25 in the skin; second at position 1 in the liver

Q49. A 28-year-old patient with Leber's hereditary optic neuropathy (LHON) carries a point mutation in mitochondrial DNA encoding a subunit of complex I of the electron transport chain. Complex I (NADH dehydrogenase) performs which of the following reactions?

(A) Transfers electrons from $FADH_2$ to ubiquinone (CoQ) and pumps 4 H^+ across the inner mitochondrial membrane

(B) Transfers electrons from ubiquinol to cytochrome c and pumps 4 H^+

(C) Reduces O_2 to H_2O using electrons from cytochrome c and pumps 2 H^+

(D) Transfers electrons from NADH to ubiquinone (CoQ) and pumps 4 H^+ across the inner mitochondrial membrane per NADH

Q50. A 60-year-old woman with rheumatoid arthritis has a markedly elevated erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP). CRP is an acute-phase protein synthesised by the liver in response to IL-6. Which of the following **BEST** describes the biological function of CRP in innate immunity?

(A) CRP neutralises bacterial endotoxin by competitive binding to TLR-4 on macrophages

(B) CRP activates the adaptive immune response by presenting antigens to T lymphocytes

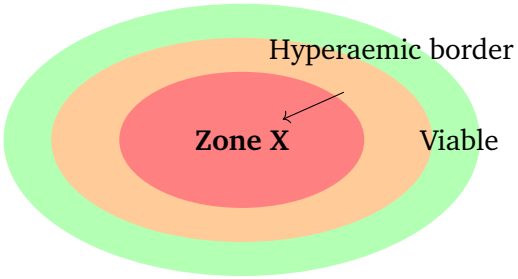
(C) CRP binds phosphocholine on the surface of apoptotic cells and microorganisms, activating the classical complement pathway and facilitating opsonisation

(D) CRP inhibits the acute-phase response by suppressing IL-6 secretion from hepatocytes via negative feedback

Section B — Para-clinical Sciences (Q 51–120)

Q51. A 62-year-old man is brought to the emergency department 3 hours after the onset of crushing chest pain. His ECG shows ST elevation in leads II, III, and aVF. He is resuscitated but dies 18 hours later. At autopsy, the infarcted myocardium shows preserved cellular outlines with loss of

nuclei and cytoplasmic eosinophilia. The diagram below schematically represents the zone of necrosis:



Zone X (the necrotic core) in this setting **MOST likely** represents which type of necrosis?

(A) Coagulative necrosis

(B) Liquefactive necrosis

(C) Caseous necrosis

(D) Fat necrosis

Q52. A pathologist examines a liver biopsy from a 38-year-old woman with chronic hepatitis C infection. On H&E staining, scattered hepatocytes are shrunken, with deeply eosinophilic cytoplasm and pyknotic nuclei, surrounded by a clear halo, without surrounding inflammation. Which of the following **BEST** distinguishes this process from necrosis?

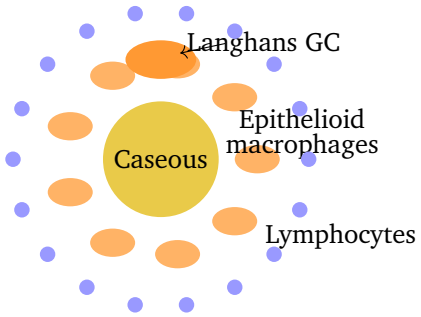
(A) Loss of plasma membrane integrity

(B) Release of lysosomal enzymes

(C) Energy (ATP)-dependent process with caspase activation

(D) Neutrophilic infiltration at the site

Q53. A 34-year-old man presents with a 3-month history of low-grade fever, weight loss, and a productive cough. Chest X-ray shows an upper-lobe cavitary lesion. Sputum AFB smear is positive. Biopsy of the lung lesion reveals the structure depicted below:



The **cell type** that is the hallmark of this lesion and is derived from circulating monocytes is:

(A) Langhans giant cell

(B) Epithelioid macrophage

(C) CD8+ cytotoxic T lymphocyte

(D) Plasma cell

Q54. A 28-year-old man sustains a deep laceration over his forearm that is left unsutured. Two weeks later, the wound shows granulation tissue with fibroblast proliferation, new capillary formation, and deposition of type III collagen. Which of the following statements about the **subsequent** remodeling phase is **CORRECT**?

(A) Type I collagen is replaced by type III collagen

(B) Tensile strength never exceeds 50% of normal skin

(C) Matrix metalloproteinases (MMPs) are inhibited throughout remodeling

(D) Type III collagen is gradually replaced by type I collagen, increasing tensile strength

Q55. A 25-year-old woman develops redness, swelling, and pain in her right hand 24 hours after a thorn puncture. Microscopy of the inflamed tissue shows predominantly neutrophil infiltration. Which of the following mediators is **MOST responsible** for the increased vascular permeability seen within the **first 30 minutes**?

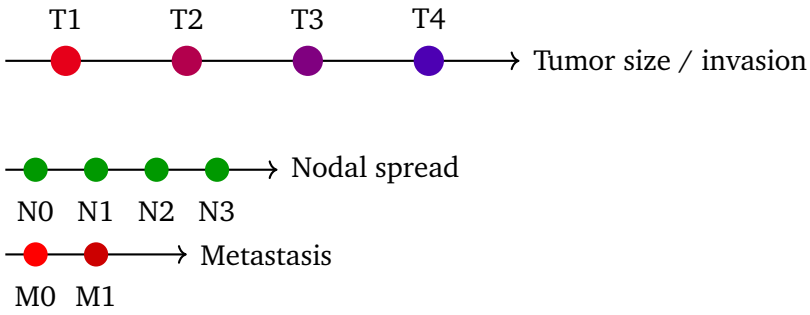
(A) Histamine released from mast cells

(B) Leukotriene B4

(C) Interleukin-1 (IL-1)

(D) Complement C3a only

Q56. A 55-year-old woman presents with a breast lump. Biopsy confirms invasive ductal carcinoma. The diagram below represents the TNM staging concept for breast cancer:



A tumor that has invaded the chest wall (T4), involves 4 axillary lymph nodes (N2), with no distant metastasis (M0) is classified as:

(A) Stage I

(B) Stage II

(C) Stage IIIB

(D) Stage IV

Q57. A 4-year-old child presents with an abdominal mass. Imaging reveals a large renal tumor. Histology shows primitive blastema, epithelial tubules, and stromal cells (triphasic pattern). Molecular analysis reveals loss of both alleles of a tumor suppressor gene on chromosome 11p13. This gene normally functions to:

(A) Activate the RAS signaling pathway

(B) Phosphorylate and activate CDK4

(C) Encode a growth factor receptor with tyrosine kinase activity

(D) Suppress transcription of genes promoting cell proliferation

Q58. A 28-year-old woman with menorrhagia presents with fatigue and pallor. Her CBC shows: Hb 7.8 g/dL, MCV 62 fL, MCH 18 pg, RDW 18% (high). Peripheral smear shows hypochromic microcytic RBCs with pencil cells. Serum ferritin is 4 ng/mL. Which of the following laboratory findings is **ALSO expected**?

(A) Increased total iron-binding capacity (TIBC)

(B) Decreased TIBC

(C) Normal serum iron with increased ferritin

(D) Increased serum iron with decreased TIBC

Q59. A 16-year-old boy presents with mild jaundice, splenomegaly, and a history of similar episodes since childhood. His elder brother has the same condition. CBC shows: Hb 9.5 g/dL, MCV 78 fL, MCHC 38 g/dL (elevated), reticulocyte count 8%. Osmotic fragility test is positive. The **PRIMARY molecular defect** in this condition is:

(A) Deficiency of glucose-6-phosphate dehydrogenase

(B) Defect in spectrin or ankyrin protein of the RBC cytoskeleton

(C) Abnormal hemoglobin chain (HbS)

(D) Acquired complement-mediated RBC destruction

Q60. A 52-year-old man presents with fatigue, left upper quadrant pain, and weight loss. Examination reveals massive splenomegaly. CBC: WBC 120,000/μL with a left shift showing myelocytes, metamyelocytes, and basophilia; Hb 9 g/dL; platelets 800,000/μL. Leukocyte alkaline phosphatase (LAP) score is very low. Cytogenetics reveals t(9;22)(q34;q11). This translocation creates:

(A) A mutant RAS protein with constitutive GTPase activity

(B) An abnormal transcription factor BCL-2/IgH fusion

(C) BCR-ABL fusion protein with constitutive tyrosine kinase activity

(D) Loss of WT1 tumor suppressor gene

Q61. A 22-year-old man presents with painless cervical lymphadenopathy, low-grade fever, night sweats, and 8 kg weight loss over 3 months. CT shows mediastinal involvement. Lymph node biopsy shows large binucleated cells with prominent ”owl-eye” nucleoli in a background of lymphocytes, plasma cells, and eosinophils. These large cells are:

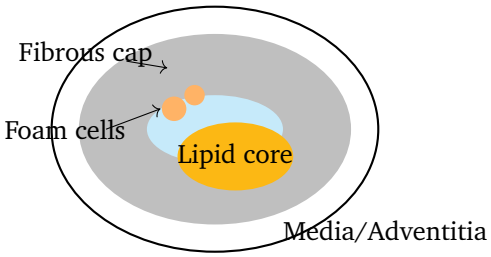
(A) Reed-Sternberg cells of Hodgkin lymphoma

(B) Burkitt lymphoma cells with starry-sky pattern

(C) Follicular center B-cells with BCL-2 overexpression

(D) Reed-Sternberg variant cells of non-Hodgkin lymphoma

Q62. A 58-year-old man with a 30-year history of smoking and hypertension undergoes coronary angiography showing 80% stenosis of the left anterior descending artery. The cross-section of an atherosclerotic plaque is shown schematically below:



Foam cells in the atheromatous plaque are derived **PRIMARILY** from:

(A) Smooth muscle cells only

(B) Endothelial cells that undergo mesenchymal transition

(C) Platelets that aggregate at the lesion site

(D) Macrophages that ingest oxidized LDL

Q63. A 65-year-old man is admitted with an anterior STEMI and is successfully thrombolysed. On day 5, he develops sudden severe hypotension and a new harsh pansystolic murmur at the left sternal border with a thrill. Echocardiography reveals a left-to-right shunt. The **MOST likely** complication responsible is:

(A) Free wall rupture with tamponade

(B) Rupture of the interventricular septum

(C) Acute mitral regurgitation due to papillary muscle rupture

(D) Left ventricular aneurysm

Q64. A 7-year-old boy develops periorbital puffiness and frothy urine 2 weeks after a sore throat. Urinalysis: protein 3+ (4 g/24 h), hematuria, RBC casts, and C3 complement is low. Renal biopsy by electron microscopy shows subepithelial "humps." This pattern is **MOST consistent** with:

(A) Minimal change disease

(B) IgA nephropathy (Berger's disease)

(C) Post-streptococcal (diffuse proliferative) glomerulonephritis

(D) Membranous nephropathy

Q65. A 4-year-old girl presents with generalized edema, heavy proteinuria (10 g/day), hypoalbuminemia (serum albumin 1.8 g/dL), and hyperlipidemia. Urine microscopy shows oval fat bodies. Renal biopsy by light microscopy is essentially normal; electron microscopy shows diffuse effacement of podocyte foot processes. The **drug of choice** for initial treatment is:

(A) Prednisolone (corticosteroids)

(B) Cyclophosphamide

(C) Captopril (ACE inhibitor)

(D) Furosemide alone

Q66. A 48-year-old man with a 20-year history of heavy alcohol use presents with jaundice, tender hepatomegaly, and fever. Labs: AST 280 U/L, ALT 110 U/L (AST:ALT ratio $>$2:1), GGT markedly elevated, bilirubin 5.2 mg/dL. Liver biopsy shows hepatocyte ballooning, Mallory-Denk bodies, neutrophil infiltration, and pericellular fibrosis. The intracellular inclusions (Mallory-Denk bodies) are composed of:

(A) Accumulated glycogen granules

(B) Fat vacuoles from triglyceride accumulation

(C) Periodic acid-Schiff (PAS)-positive granules of α-1-antitrypsin

(D) Aggregated intermediate filaments (cytokeratin 8/18)

Q67. A 32-year-old man is found to have HBsAg positive, HBeAg positive, anti-HBc IgM positive, and high HBV DNA. He is asymptomatic but has elevated ALT. Which of the following indicates **HIGH infectivity**?

(A) Anti-HBs positivity

(B) HBeAg positivity

(C) Anti-HBe positivity

(D) Anti-HBc IgG alone (with negative HBsAg)

Q68. A 55-year-old man with hepatitis C-related cirrhosis presents with sudden-onset hematemesis. Endoscopy reveals bleeding esophageal varices. The pathophysiological basis of variceal formation in cirrhosis is:

(A) Decreased hepatic arterial pressure

(B) Hypoalbuminemia causing reduced oncotic pressure

(C) Portal hypertension due to increased intrahepatic vascular resistance

(D) Hypercoagulable state causing portal vein thrombosis only

Q69. A 70-year-old man with diabetes is admitted with a 4-day history of productive cough, fever, and right-sided chest pain. CXR shows consolidation of the right lower lobe. His sputum Gram stain shows Gram-positive diplococci. The **MOST common** organism causing community-acquired lobar pneumonia in adults is:

(A) Streptococcus pneumoniae

(B) Klebsiella pneumoniae

(C) Staphylococcus aureus

(D) Haemophilus influenzae

Q70. A 42-year-old HIV-positive man (CD4 count 80 cells/μL) presents with fever, night sweats, and diffuse bilateral pulmonary infiltrates. AFB smear of BAL is positive. Which feature of TB pathology is **LEAST likely** to be seen in this severely immunocompromised patient?

(A) Numerous AFB organisms

(B) Widespread caseation

(C) Miliary pattern of dissemination

(D) Well-formed epithelioid granulomas with Langhans giant cells

Q71. A 62-year-old heavy smoker presents with a 3-cm hilar mass, hemoptysis, and hypercalcemia. No PTH-related protein elevation is confirmed by lab. Bronchoscopy biopsy shows large polygonal cells with intercellular bridges and keratin pearls. The **MOST likely** diagnosis is:

(A) Small cell (oat cell) carcinoma

(B) Squamous cell carcinoma of the lung

(C) Adenocarcinoma of the lung

(D) Large cell carcinoma

Q72. A 55-year-old man with a PSA of 28 ng/mL undergoes prostate biopsy showing adenocarcinoma. The PSA is used as a **tumor marker**. Regarding tumor markers, which of the following statements is **CORRECT**?

(A) AFP is the most specific marker for prostate carcinoma

(B) CEA is specific for colorectal carcinoma and is used for screening

(C) Tumor markers are useful for monitoring treatment response and detecting recurrence

(D) CA-125 is highly specific for ovarian cancer and is used for population-based screening

Q73. A 35-year-old man with anaphylactic shock is brought to the emergency department with blood pressure 60/30 mmHg, heart rate 130 bpm, and severe bronchospasm. The **drug of choice** for anaphylactic shock that acts on α_1, β_1, and β_2 receptors is:

(A) Epinephrine (adrenaline) 0.5 mg IM

(B) Norepinephrine IV infusion

(C) Phenylephrine IV

(D) Dopamine 5 μg/kg/min IV

Q74. A 28-year-old farmer is brought unconscious with excessive secretions, miosis, bradycardia (HR 42 bpm), bronchospasm, and muscle fasciculations. His family reports he was spraying pesticides. Initial management includes atropine and pralidoxime. Pralidoxime is **MOST effective** when given:

(A) After 72 hours, when the clinical picture is clear

(B) Only after all atropine requirements are met

(C) As a sole agent without atropine

(D) Within 24–48 hours before ”aging” of acetylcholinesterase occurs

Q75. A 25-year-old woman with a history of generalized tonic-clonic seizures is started on valproic acid. She becomes pregnant. The **MOST significant teratogenic risk** associated with valproate use in the **first trimester** is:

(A) Neonatal hypoglycemia

(B) Neural tube defects (spina bifida)

(C) Cleft palate alone

(D) Neonatal hemorrhage due to vitamin K deficiency

Q76. A 38-year-old man with schizophrenia has been on haloperidol for 6 months. He now presents with slow, writhing involuntary movements of his tongue and face that persist even after the drug is reduced. This adverse effect is known as:

(A) Akathisia

(B) Acute dystonia

(C) Tardive dyskinesia

(D) Neuroleptic malignant syndrome

Q77. A 42-year-old woman with major depressive disorder is started on fluoxetine. She is also taking tramadol for chronic pain. Three weeks later she develops agitation, hyperthermia, myoclonus, and diaphoresis. This life-threatening interaction is caused by:

(A) Serotonin syndrome due to excess serotonergic activity

(B) Neuroleptic malignant syndrome from dopamine blockade

(C) Anticholinergic toxidrome from muscarinic blockade

(D) Sympathomimetic crisis from norepinephrine excess

Q78. A 55-year-old man with cancer pain is on long-term morphine. He presents with severe respiratory depression (RR 6/min), miosis, and unconsciousness after accidentally receiving an extra dose. The **drug of choice** for reversal is:

(A) Nalbuphine

(B) Buprenorphine

(C) Pentazocine

(D) Naloxone IV

Q79. A 45-year-old man requires general anaesthesia for an elective cholecystectomy. Induction is achieved with propofol. Which of the following **BEST** describes propofol's mechanism of action?

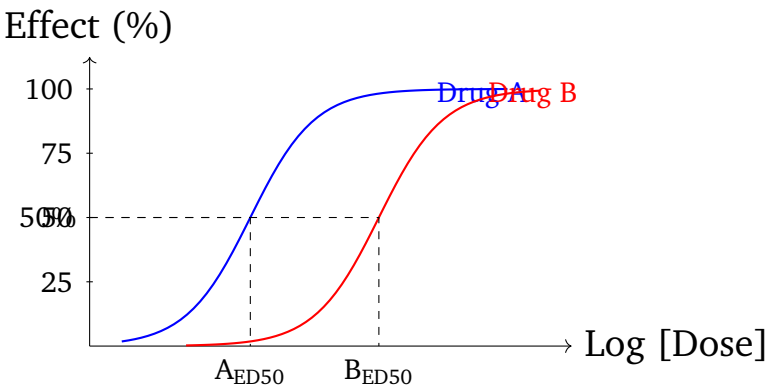
(A) Blockade of NMDA glutamate receptors

(B) Potentiation of $GABA_A$ receptor-mediated chloride influx

(C) Inhibition of voltage-gated sodium channels

(D) Agonism at μ-opioid receptors

Q80. A pharmacologist plots full dose-response curves for two drugs acting on the same receptor. The graph below shows Drug A and Drug B:



From the graph, Drug A compared to Drug B has:

(A) Greater efficacy and greater potency

(B) Same efficacy but lesser potency

(C) Greater potency but the same maximum efficacy

(D) Lesser potency and lesser efficacy

Q81. A 52-year-old man with hypertension and benign prostatic hyperplasia (BPH) is started on a single drug that will treat both conditions simultaneously. The mechanism of this drug is:

(A) Blockade of β_1 adrenergic receptors reducing cardiac output

(B) Inhibition of ACE preventing angiotensin II formation

(C) Blockade of calcium channels in vascular smooth muscle

(D) Blockade of α_1 adrenergic receptors causing vasodilation and relaxing bladder neck

Q82. A 68-year-old man with a previous MI develops ventricular tachycardia (VT). He is hemodynamically stable. The drug of choice for hemodynamically stable VT that acts by blocking fast sodium channels (Class IA/IB) is:

(A) Amiodarone IV

(B) Verapamil IV

(C) Digoxin

(D) Adenosine IV

Q83. A 60-year-old man with heart failure with reduced ejection fraction (HFrEF, EF 30%) is on furosemide, enalapril, and carvedilol. He develops worsening edema despite optimal doses. Which of the following **ADDITIONAL** drug has been shown to reduce **mortality** in this setting?

(A) Digoxin

(B) Spironolactone (aldosterone antagonist)

(C) Hydralazine alone

(D) Amlodipine

Q84. A 70-year-old woman with non-valvular atrial fibrillation requires anticoagulation for stroke prevention. She has normal renal function. She is started on rivaroxaban. The mechanism of rivaroxaban is:

(A) Inhibition of thrombin (Factor IIa) directly

(B) Inhibition of vitamin K-dependent clotting factor synthesis

(C) Direct inhibition of Factor Xa

(D) Activation of antithrombin III to inhibit multiple clotting factors

Q85. A 5-year-old boy is prescribed amoxicillin for acute otitis media. His mother reports he had a rash (urticaria) with amoxicillin 1 year ago. Which of the following is the **MOST appropriate** alternative?

(A) Ampicillin (another aminopenicillin)

(B) Cephalexin (first-generation cephalosporin)

(C) Piperacillin-tazobactam

(D) Azithromycin (macrolide)

Q86. A 65-year-old man with hospital-acquired pneumonia is started on gentamicin. After 7 days, he develops rising serum creatinine and reports difficulty hearing. Which of the following monitoring parameters is **MOST important** for aminoglycoside therapy?

(A) Trough serum levels to assess nephrotoxicity risk and audiometry

(B) Liver function tests for hepatotoxicity

(C) Platelet count for thrombocytopenia

(D) INR for anticoagulant interaction

Q87. A 55-year-old woman with a urinary tract infection is treated with ciprofloxacin. She concurrently takes antacids containing magnesium. The pharmacokinetic concern is:

(A) Ciprofloxacin increases the renal excretion of magnesium

(B) Magnesium induces CYP450 enzymes, increasing ciprofloxacin metabolism

(C) Divalent cations chelate ciprofloxacin in the gut, markedly reducing absorption

(D) Ciprofloxacin is contraindicated only because of cardiac QTc prolongation with Mg

Q88. A 35-year-old HIV-positive patient (CD4 count 50 cells/μL) develops headache, fever, and neck stiffness. CSF India ink preparation reveals encapsulated yeast. He is started on amphotericin B deoxycholate. The mechanism of amphotericin B is:

(A) Inhibition of beta-glucan synthase (cell wall synthesis)

(B) Binding to ergosterol in fungal cell membrane causing pore formation

(C) Inhibition of lanosterol 14α-demethylase (CYP51)

(D) Inhibition of fungal DNA synthesis

Q89. A 32-year-old man newly diagnosed with HIV (CD4 230 cells/μL, viral load 120,000 copies/mL) is started on tenofovir + emtricitabine + dolutegravir. Dolutegravir belongs to which drug class and acts by:

(A) Nucleoside reverse transcriptase inhibitor (NRTI) — inhibits viral RNA-dependent DNA polymerase

(B) Protease inhibitor — prevents cleavage of viral polyprotein

(C) Non-nucleoside reverse transcriptase inhibitor (NNRTI) — binds allosteric site of reverse transcriptase

(D) Integrase strand transfer inhibitor (INSTI) — prevents integration of viral DNA into host genome

Q90. A 28-year-old man is started on HRZE (isoniazid, rifampicin, ethambutol, pyrazinamide) for pulmonary TB. After 2 months, he develops peripheral neuropathy. The **drug responsible** and the **mechanism** are:

(A) Rifampicin — inhibits mycobacterial RNA polymerase causing neuronal toxicity

(B) Ethambutol — inhibits arabinosyl transferase causing optic neuritis, not peripheral neuropathy

(C) Isoniazid — inhibits pyridoxine (vitamin B6) metabolism causing peripheral neuropathy

(D) Pyrazinamide — inhibits fatty acid synthesis causing peripheral neuropathy

Q91. A 48-year-old woman with rheumatoid arthritis on long-term diclofenac develops epigastric pain and melena. Endoscopy shows a gastric ulcer. The **MOST important mechanism** by which NSAIDs cause GI injury is:

(A) Inhibition of COX-1 reducing prostaglandin E2 synthesis, impairing mucosal protection

(B) Direct topical acid injury to gastric mucosa when taken on an empty stomach

(C) Selective COX-2 inhibition increasing thromboxane A2

(D) Inhibition of histamine H2 receptors in gastric parietal cells

Q92. A 35-year-old woman with breast cancer (ER+/PR+, HER2-) receives tamoxifen as adjuvant therapy. The **mechanism of action** of tamoxifen is:

(A) Aromatase inhibition preventing peripheral conversion of androgens to estrogens

(B) Competitive antagonism at estrogen receptors in breast tissue (SERM)

(C) Direct cytotoxic alkylation of tumor cell DNA

(D) Inhibition of topoisomerase II causing double-strand DNA breaks

Q93. A 72-year-old man with insomnia is prescribed a benzodiazepine. Due to his age and risk of accumulation, the **MOST appropriate** short-acting benzodiazepine with **no active metabolites** is:

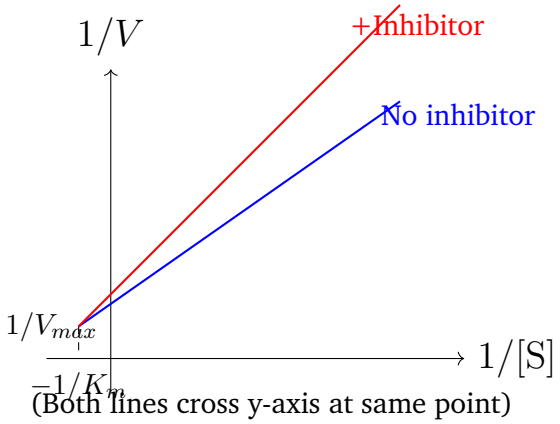
(A) Diazepam

(B) Chlordiazepoxide

(C) Flurazepam

(D) Lorazepam

Q94. A pharmacologist studies the effect of a reversible competitive inhibitor on an enzyme. The Lineweaver-Burk (double-reciprocal) plot is shown:



(Both lines cross y-axis at same point)

Which of the following changes is **characteristic** of competitive inhibition as shown?

(A) Decreased Vmax, unchanged Km

(B) Decreased Vmax, increased Km

(C) Unchanged Vmax, increased apparent Km

(D) Unchanged Vmax, decreased Km

Q95. A 12-year-old boy presents with a painful, fluctuant swelling over his right forearm following a minor abrasion one week ago. Pus culture

shows Gram-positive cocci in clusters, coagulase-positive, beta-hemolytic on blood agar. The organism produces a toxin that destroys leukocytes. This toxin is:

(A) Panton-Valentine leukocidin (PVL)

(B) Toxic shock syndrome toxin-1 (TSST-1)

(C) Exfoliatin (Epidermolytic toxin)

(D) Streptolysin O

Q96. A 3-day-old neonate has not passed meconium and has difficulty feeding. He was born at home; the umbilical cord was cut with unsterilized scissors. He develops muscle rigidity and spasms triggered by light stimulation. The **MOST likely** causative organism is:

(A) Clostridium perfringens

(B) Clostridium difficile

(C) Bacillus anthracis

(D) Clostridium tetani

Q97. A 5-year-old child from a flood-affected area presents with sudden-onset profuse watery ”rice water” diarrhea without blood or mucus, and severe dehydration. Stool culture on TCBS agar shows yellow colonies. The organism produces a toxin that acts by:

(A) Inhibiting protein synthesis at the 60S ribosomal subunit

(B) Permanently activating adenylate cyclase via ADP-ribosylation of Gs, increasing cAMP and causing secretory diarrhea

(C) Disrupting tight junctions causing paracellular leakage

(D) Producing a cytotoxin causing bloody diarrhea

Q98. A 25-year-old man returns from a trip to Southeast Asia with a 10-day history of stepwise rising fever, relative bradycardia, coated tongue, rose spots on abdomen, and splenomegaly. Blood culture at 1st week is **MOST likely** to grow:

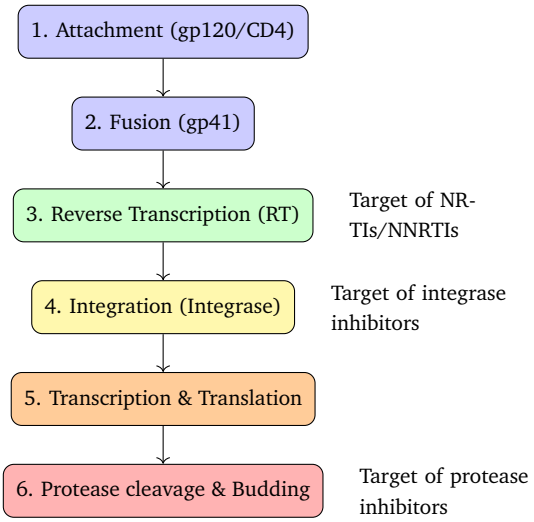
(A) Shigella dysenteriae

(B) Vibrio cholerae

(C) Salmonella typhi

(D) Escherichia coli O157:H7

Q99. A 30-year-old man tests positive for HIV. The schematic below represents key steps in the HIV replication cycle:



The **FIRST** identifiable serological marker to become positive after HIV infection (within 2 weeks, during the ”window period” of antibody testing) is:

(A) HIV p24 antigen

(B) Anti-HIV IgG antibodies

(C) Anti-HIV IgM antibodies

(D) HIV RNA viral load (by 4th generation ELISA)

Q100. A 35-year-old woman is found to be HBsAg positive on routine screening. Further testing: HBeAg negative, anti-HBe positive, HBV DNA 180 IU/mL (low). She has no symptoms and normal liver function. Her serology indicates:

(A) Active hepatitis B with high infectivity

(B) Acute hepatitis B infection

(C) Occult hepatitis B infection

(D) Inactive HBsAg carrier state (low replication phase)

Q101. A 28-year-old man with HIV (CD4 count 40 cells/μL) presents with 3 weeks of progressive headache and mild neck stiffness. CSF shows: opening pressure 320 mmH_2O, lymphocytic pleocytosis, low glucose, elevated protein. India ink stain shows encapsulated budding yeast. The **BEST confirmatory test** for this infection is:

(A) Sabouraud dextrose agar culture at 37°C

(B) CSF cryptococcal antigen (CrAg) detection

(C) Galactomannan antigen assay

(D) Potassium hydroxide (KOH) preparation of sputum

Q102. A 7-year-old boy presents with sore throat, fever, and cervical lymphadenopathy. On examination his tonsils are erythematous with exudates and his tongue is red and bumpy ("strawberry tongue"). He also has a diffuse erythematous rash sparing the face. The organism responsible elaborates which toxin causing the rash?

(A) Leukocidin

(B) Streptolysin S

(C) Erythrogenic (pyrogenic) exotoxin (Streptococcal pyogenic exotoxin)

(D) Streptokinase

Q103. A 22-year-old man returns from sub-Saharan Africa with fever occurring every 48 hours, rigors, and severe headache. Peripheral blood smear shows ring forms within RBCs and banana-shaped gametocytes. Which species of Plasmodium is **responsible** and what is the **serious complication** it can cause?

(A) P. falciparum — cerebral malaria due to cytoadherence of parasitized RBCs

(B) P. vivax — relapse due to hypnozoites in liver

(C) P. malariae — quartan nephrotic syndrome

(D) P. ovale — mild disease with relapses

Q104. A 30-year-old man from a rural area presents with 2 weeks of bloody mucoid diarrhea and right upper quadrant pain. Stool microscopy shows trophozoites with ingested RBCs. Ultrasonography reveals a single 8 cm hepatic lesion with ”anchovy sauce” fluid. The **drug of choice** is:

(A) Chloroquine alone

(B) Paromomycin (luminal agent) alone

(C) Albendazole

(D) Metronidazole followed by a luminal agent (paromomycin or diloxanide furoate)

Q105. The diagram below categorizes the four Gell and Coombs hypersensitivity types:

Type	Mediator	Example
Type I (Immediate	IgE + Mast cells	Anaphylaxis asthma
Type II (Cytotoxic	IgG IgM + complem	Hemolytic anemia
Type III (Immune complex)	IgG complexes + complement	SLE serum sickness
Type IV (Delayed)	T lymphocytes (no A	TB contact dermatitis

A 45-year-old nurse develops an intensely pruritic, vesicular rash on her hands 48 hours after wearing latex gloves. The **hypersensitivity type** responsible is:

(A) Type I (IgE-mediated)

(B) Type II (cytotoxic)

(C) Type IV (delayed-type / cell-mediated)

(D) Type III (immune complex-mediated)

Q106. A 19-year-old student presents with sudden high fever (40°C), severe retro-orbital pain, myalgia, and a petechial rash on the trunk. CBC:

WBC 2,800/μL, platelets 48,000/μL. NS1 antigen test is positive on day 3. What is the **MOST important pathophysiological mechanism** of thrombocytopenia in dengue?

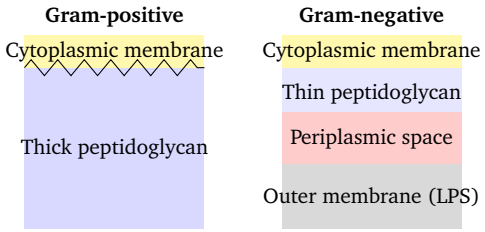
(A) Disseminated intravascular coagulation (DIC) consuming platelets

(B) Bone marrow suppression and platelet destruction by virus/immune mechanisms

(C) Hypersplenism from dengue hepatitis

(D) Autoimmune antibody (anti-platelet IgG) formation only

Q107. The schematic below shows the cell wall architecture of Gram-positive and Gram-negative bacteria:



Which of the following is a **unique component** of the Gram-negative outer membrane **NOT found** in Gram-positive bacteria?

(A) Teichoic acid

(B) Peptidoglycan

(C) Lipopolysaccharide (LPS / endotoxin)

(D) N-acetylmuramic acid

Q108. A 14-year-old boy from a rural farming community presents with iron deficiency anemia and hypoalbuminemia. He is found to have eosinophilia (22%) and a positive stool examination showing oval eggs with thin shells. The **mechanism of anemia** in hookworm infection is:

(A) Intravascular hemolysis of infected RBCs

(B) B12 deficiency due to ileal colonization and binding

(C) Bone marrow suppression by helminthic toxins

(D) Chronic intestinal blood loss from worm feeding on intestinal villi plus blood sucking

Q109. A body is discovered in a room. On examination at 2 PM, the body is found to have well-established rigor mortis in all muscle groups (jaw, neck, trunk, limbs) and greenish discoloration of the right iliac fossa. Body temperature is 28°C (room temperature 22°C). Approximately when did death **MOST likely** occur?

(A) 12–24 hours before discovery

(B) 2–4 hours before discovery

(C) 36–48 hours before discovery

(D) Over 5 days before discovery

Q110. A 28-year-old woman is found hanging in her apartment. At autopsy, the ligature mark is oblique, incomplete (interrupted), and situated above the thyroid cartilage. There is no fracture of cervical vertebrae. Petechiae (Tardieu spots) are seen in the conjunctivae. The cause of death is:

(A) Judicial (homicidal) hanging — fracture-dislocation of C2

(B) Suicidal hanging — asphyxia due to venous obstruction and airway compression

(C) Manual strangulation — bilateral carotid pressure

(D) Garroting — ligature mark is horizontal and complete

Q111. A 35-year-old farmer is rushed to the ER after ingesting a pesticide. He has profuse salivation, lacrimation, urination, diarrhea, bradycardia (HR 40 bpm), and severe bronchospasm. His pupils are pin-point. The **antidote** that addresses the central and peripheral muscarinic features is:

(A) Pralidoxime (PAM) alone

(B) Diazepam for seizure control only

(C) Atropine (large repeated doses titrated to drying of secretions)

(D) N-acetylcysteine

Q112. A 40-year-old man sustains burns in a house fire. At autopsy, the epidermis and part of dermis are destroyed with blistering; the skin beneath the blister is red and moist. Vital reaction (inflammatory response) is present. Which **degree of burn** does this represent?

(A) Second degree (partial thickness) burn — antemortem

(B) First degree (superficial/erythema) burn — antemortem

(C) Third degree (full thickness) burn — antemortem

(D) Charring — postmortem artefact

Q113. A family of four is found unresponsive in a closed room with a kerosene heater in winter. Postmortem examination shows cherry-red discoloration of skin, blood, and viscera. COHb level is 60%. CO toxicity kills by:

(A) Forming metHb which cannot carry oxygen

(B) Direct cardiac toxicity by binding to cardiac myosin

(C) Inhibiting cytochrome c oxidase and forming carboxymyoglobin causing left-shift of HbO2 curve only

(D) Binding to Hb with 240× greater affinity than O2 AND inhibiting mitochondrial cytochrome oxidase, causing cellular hypoxia

Q114. A 25-year-old man is brought to the ER with a stab wound to the left chest. He is unconscious. No relatives are present. The treating physician needs to perform an emergency surgery. The **CORRECT** medicolegal principle governing this situation in India is:

(A) Surgery cannot be performed without written consent from the next of kin

(B) Emergency surgery can proceed under the doctrine of implied consent to save life

(C) The doctor must wait for police clearance before any intervention

(D) Written consent from a magistrate is mandatory before any emergency procedure

Q115. A new screening test for tuberculosis is evaluated in a study. The results are presented in the 2×2 table below:

	Disease+	Disease-
Test+	TP = 90	FP = 20
Test-	FN = 10	TN = 180

All disease+: 100 All disease-: 200

The **sensitivity** and **specificity** of this test are, respectively:

(A) Sensitivity 81.8%, Specificity 94.7%

(B) Sensitivity 94.7%, Specificity 81.8%

(C) Sensitivity 90%, Specificity 90%

(D) Sensitivity 80%, Specificity 75%

Q116. In a cohort study of 10,000 smokers and 10,000 non-smokers followed for 20 years, 400 smokers and 80 non-smokers developed lung cancer. The **relative risk (RR)** of lung cancer in smokers compared to non-smokers is:

(A) 2.0

(B) 5.0

(C) 8.0

(D) 10.0

Q117. A 6-week-old infant is brought for immunization as per the National Immunization Schedule (NIS) of India. According to the current NIS, which of the following vaccines is **NOT** given at 6 weeks of age?

(A) OPV (Oral Polio Vaccine)

(B) Pentavalent vaccine (DPT + Hep B + Hib)

(C) Rotavirus vaccine

(D) MMR (Measles-Mumps-Rubella)

Q118. Following heavy monsoon flooding, an outbreak of jaundice with nausea, abdominal pain, and dark urine occurs in a village. The disease has an incubation period of 15–45 days, is self-limiting, and does NOT lead to chronic infection. The **MOST likely** causative agent is:

(A) Hepatitis B virus

(B) Hepatitis A virus

(C) Hepatitis C virus

(D) Hepatitis E virus

Q119. In a malaria-endemic district, the district health officer implements "source reduction" as the primary vector control strategy. This refers to:

(A) Indoor residual spraying (IRS) with DDT or pyrethroids

(B) Use of long-lasting insecticidal nets (LLINs)

(C) Elimination of mosquito breeding sites (stagnant water, draining swamps)

(D) Biological control using Bacillus thuringiensis israelensis (Bti)

Q120. The **MOST sensitive indicator** of the health status of a community and the quality of obstetric and pediatric care is:

(A) Infant mortality rate (IMR)

(B) Crude birth rate (CBR)

(C) Total fertility rate (TFR)

(D) Maternal mortality ratio (MMR)

Section C — Clinical Medicine (Q 121–200)

Q121. During an outbreak of typhoid fever in a residential school, 120 students were exposed to a common water source. Of these, 30 developed typhoid fever. A subsequent investigation revealed that among the 30 primary cases, 10 had family contacts (total 50 family contacts), and 5 of those family contacts developed typhoid fever. The **secondary attack rate** among family contacts is:

(A) 10%

(B) 25%

(C) 4.2%

(D) 16.7%

Q122. A new rapid diagnostic test for pulmonary tuberculosis is evaluated against sputum culture (gold standard) in 400 patients. The results are shown in the contingency table below:

Test	Culture +	Culture –
Test +	140 (TP)	20 (FP)
Test –	10 (FN)	230 (TN)

The **specificity** of this test is:

(A) 93.3%

(B) 92.0%

(C) 87.5%

(D) 96.0%

Q123. In a cross-sectional survey of a district with a population of 50,000, a total of 2,500 individuals were found to have hypertension at a single point in time. During the previous year, 500 new cases of hypertension were diagnosed in the same population. Which of the following correctly states the **point prevalence** of hypertension?

(A) 1% (500/50,000)

(B) 0.5% (250/50,000)

(C) 5% (2,500/50,000)

(D) 2% (1,000/50,000)

Q124. In a cohort study examining the association between cigarette smoking and lung cancer, the following data were obtained over 10 years:

Group	Cancer (+)	No Cancer (–)
Smokers (1000)	100	900
Non-smokers (1000)	20	980

The **relative risk** (RR) of developing lung cancer in smokers compared to non-smokers is:

(A) 2.5

(B) 3.0

(C) 4.0

(D) 5.0

Q125. A randomised controlled trial on use of aspirin in secondary prevention of myocardial infarction shows that the event rate in the treatment group was 8% and the event rate in the placebo group was 12% over 5 years. The **number needed to treat** (NNT) to prevent one additional myocardial infarction is:

(A) 25

(B) 20

(C) 50

(D) 100

Q126. A health worker in a primary health centre is preparing to administer vaccines to infants. The cold chain temperature for storage of most EPI vaccines (BCG, OPV, DPT, Measles) in an ice-lined refrigerator (ILR) at the district level is:

(A) $+2^{\circ}$C to $+4^{\circ}$C

(B) $+2^{\circ}$C to $+8^{\circ}$C

(C) -15°C to -25°C

(D) 0°C to $+2^{\circ}$C

Q127. A 10-year-old child from a rural area presents with brown discoloration of teeth (dental fluorosis) and skeletal deformities. On investigation, the community water supply shows a fluoride level of 3.5 mg/L. As per WHO and Indian standards, the permissible limit of fluoride in drinking water is:

(A) 0.5 mg/L

(B) 0.8 mg/L

(C) 1.0 mg/L

(D) 1.5 mg/L

Q128. During a malaria surveillance programme in an endemic district, an entomologist collects adult mosquitoes from 100 houses and identifies 200 female *Anopheles* mosquitoes, of which 10 are found to be carrying *Plasmodium falciparum* sporozoites. The **sporozoite rate** and the **most appropriate vector control measure** for *Anopheles culicifacies* (the primary vector) in the indoor resting setting is:

(A) 5%; larviciding with temephos

(B) 5%; biological control with *Gambusia* fish

(C) 10%; aerial spraying of malathion

(D) 5%; indoor residual spraying (IRS) with DDT or synthetic pyrethroid

Q129. A 35-year-old male labourer presents with cough for 3 weeks, evening fever, and haemoptysis. Sputum AFB smear is positive (2+). He is registered under Nikshay and started on Category I DOTS regimen. The **intensive phase** of Category I treatment under the National TB Elimination Programme (NTEP) consists of:

(A) 2 months of HRZE (Isoniazid, Rifampicin, Pyrazinamide, Ethambutol)

(B) 3 months of HRZ

(C) 1 month of HRZES

(D) 2 months of HR only

Q130. The infant mortality rate (IMR) is one of the most sensitive indicators of the health status of a community. The formula for IMR per 1000 live births in a given year uses:

(A) Deaths under 1 year / Total population $\times$ 1000

(B) Deaths under 1 year / Total live births $\times$ 1000

(C) Deaths under 5 years / Total live births $\times$ 1000

(D) Deaths under 28 days / Total live births $\times$ 1000

Q131. A 58-year-old male with a history of hypertension and diabetes presents to the emergency department with severe crushing chest pain radiating to the left arm for 90 minutes, diaphoresis, and nausea. Blood pressure is 100/70 mmHg, heart rate 98 bpm. ECG shows ST elevation of 3 mm in leads II, III, and aVF with reciprocal ST depression in leads I and aVL. Troponin I is 4.2 ng/mL (normal $<$0.04). The **MOST appropriate immediate management** is:

(A) Aspirin + Clopidogrel + IV heparin + transfer for primary PCI within 90 minutes

(B) Aspirin + IV morphine + IV furosemide + thrombolysis with streptokinase

(C) Aspirin + Clopidogrel + IV heparin + primary PCI within 120 minutes if door-to-balloon cannot be achieved within 90 min, else thrombolysis

(D) IV metoprolol + aspirin + atorvastatin + conservative management

Q132. A 70-year-old woman with known ischaemic cardiomyopathy presents with worsening breathlessness on minimal exertion (NYHA class III), bilateral pitting pedal oedema up to the knee, and orthopnoea. Chest X-ray

shows cardiomegaly, upper lobe diversion, and Kerley B lines. Echocardiography reveals EF 30%, dilated left ventricle. BNP is 1200 pg/mL. Which of the following medications has been shown to **reduce mortality** in this patient and is the **drug of choice**?

(A) Digoxin

(B) Loop diuretic (furosemide)

(C) Nitrates

(D) Sacubitril/Valsartan (ARNi)

Q133. A 65-year-old male hypertensive presents with palpitations and breathlessness for 6 hours. The ECG strip below is obtained:

Irregularly irregular rhythm, no distinct P waves

His $CHADS_2$-VASc score is 4. The **MOST appropriate long-term management** is:

(A) Rate control with metoprolol + anticoagulation with rivaroxaban

(B) Rhythm control with amiodarone only, no anticoagulation needed

(C) Aspirin alone for stroke prophylaxis

(D) DC cardioversion immediately without anticoagulation

Q134. A 32-year-old woman with a history of recurrent sore throat in childhood presents with progressive exertional dyspnoea and occasional haemoptysis. On auscultation, the apex is tapping, there is a loud S1, an opening snap 60 ms after A2, and a low-pitched mid-diastolic rumble at the apex. ECG shows P-mitrale. Echocardiography reveals a mitral valve area of 0.9 cm^2 with a mean gradient of 14 mmHg. The **MOST appropriate intervention** is:

(A) Long-term diuretic therapy with furosemide

(B) Balloon mitral valvotomy (BMV/PTMC)

(C) Mitral valve replacement with a mechanical prosthesis

(D) Beta-blocker therapy to control heart rate

Q135. A 55-year-old male with a 10-year history of hypertension presents for review. Current BP is 162/98 mmHg on two readings 15 minutes apart. He has microalbuminuria (urine ACR 45 mg/g), serum creatinine 1.4 mg/dL, eGFR 58 mL/min/1.73m^2, and no other comorbidities. The **drug class** that is **MOST beneficial** as first-line therapy in this patient with hypertension and early CKD:

(A) Thiazide diuretic (hydrochlorothiazide)

(B) Calcium channel blocker (amlodipine)

(C) ACE inhibitor (ramipril)

(D) Beta-blocker (atenolol)

Q136. A 68-year-old retired teacher presents with 5-day history of productive cough with rusty sputum, right-sided pleuritic chest pain, fever (39.2°C), and rigors. Examination reveals dullness to percussion, bronchial breath sounds, and increased vocal resonance over the right lower zone. CXR shows right lower lobe consolidation. WBC is 18,000/mm^3 (neutrophilia), CRP 140 mg/L. Blood cultures are pending. CURB-65 score is 2. The **MOST likely causative organism** and **MOST appropriate initial antibiotic** are:

(A) *Staphylococcus aureus*; vancomycin IV

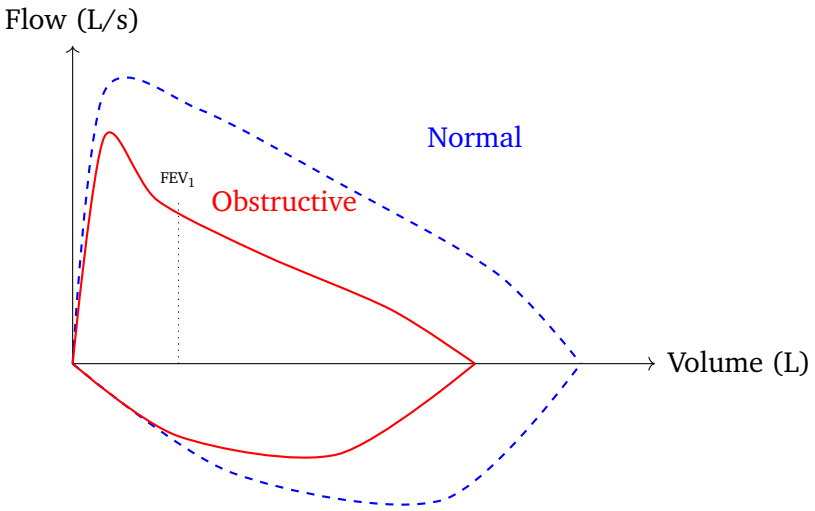
(B) *Klebsiella pneumoniae*; cefepime IV

(C) *Legionella pneumophila*; clarithromycin + rifampicin

(D) *Streptococcus pneumoniae*; amoxicillin-clavulanate or co-amoxiclav

Q137. A 62-year-old male ex-smoker (40 pack-years) presents with progressive breathlessness, chronic cough with whitish sputum, and two hospitalizations in the past year. Spirometry shows FEV_1/FVC ratio of 0.58

(post-bronchodilator), FEV_1 = 42% predicted. The spirometry pattern is shown:



According to GOLD guidelines, this patient is classified as GOLD stage:

(A) GOLD 3 (Severe): FEV_1 30–49% predicted

(B) GOLD 2 (Moderate): FEV_1 50–79% predicted

(C) GOLD 4 (Very Severe): FEV_1 <30% predicted

(D) GOLD 1 (Mild): FEV_1 ≥80% predicted

Q138. A 45-year-old male presents with progressive breathlessness and right-sided dullness on percussion. Pleural fluid aspiration yields straw-coloured fluid. Investigations: Pleural fluid protein 4.8 g/dL, serum protein 6.5 g/dL; pleural fluid LDH 350 IU/L, serum LDH 280 IU/L; pleural fluid glucose 65 mg/dL. Applying **Light's criteria**, this effusion is:

(A) Transudate — likely due to congestive heart failure

(B) Exudate — pleural fluid protein/serum protein ratio >0.5

(C) Transudate — pleural fluid LDH/serum LDH <0.6

(D) Exudate — pleural fluid glucose <60 mg/dL indicates malignancy

Q139. A 19-year-old college student with known bronchial asthma presents to the emergency department with acute severe attack: respiratory rate 28/min, SpO_2 90% on room air, unable to complete sentences, PEFR

40% of predicted, using accessory muscles. She is already on low-dose ICS + SABA PRN. The **MOST appropriate immediate treatment** in the emergency department is:

(A) Double the dose of inhaled corticosteroid and discharge with spacer

(B) Start magnesium sulphate IV and discharge after 30 minutes

(C) Nebulised salbutamol + ipratropium, systemic corticosteroid, controlled oxygen (SpO_2 target 93–95%), monitor for deterioration

(D) Intubate immediately and start mechanical ventilation

Q140. A 42-year-old male presents with a 3-month history of epigastric pain that wakes him from sleep at 2 am, relieved by eating or antacids. Upper GI endoscopy reveals a 1.2 cm duodenal ulcer (D1). Rapid urease test (RUT) is positive. The **MOST appropriate treatment** is:

(A) Omeprazole alone for 8 weeks

(B) H_2 blocker (ranitidine) for 4 weeks

(C) Sucralfate + antacids for 6 weeks

(D) Triple therapy: Omeprazole + Clarithromycin + Amoxicillin for 14 days

Q141. A 26-year-old male presents with 8-month history of intermittent right lower quadrant colicky pain, non-bloody diarrhoea (5–6 stools/day), weight loss of 7 kg, and perianal fistula. Colonoscopy shows skip lesions, cobblestone appearance of mucosa, and strictures in the terminal ileum. Biopsy shows non-caseating granulomas. Which of the following is the **MOST likely diagnosis**?

(A) Crohn's disease

(B) Ulcerative colitis

(C) Intestinal tuberculosis

(D) Ischaemic colitis

Q142. A 50-year-old male with known alcoholic liver disease presents with massive haematemesis. Examination reveals spider naevi, palmar erythema, splenomegaly, and ascites. Serum bilirubin is 3.5 mg/dL, albumin 2.4 g/dL, prothrombin time prolonged by 5 seconds; grade II hepatic encephalopathy; moderate ascites. The Child-Pugh score places him in class:

(A) Child A (score 5–6), low surgical risk

(B) Child C (score $\geq$10), high surgical risk

(C) Child B (score 7–9), intermediate risk

(D) Cannot be calculated without LFT values

Q143. A 38-year-old obese male with a history of alcohol binge presents with severe epigastric pain radiating to the back, nausea, and vomiting. Serum amylase is 1100 U/L (normal $<$100), lipase 2200 U/L. CT abdomen with contrast (CECT) is performed 48 hours after admission. Which of the following CT findings indicates **severe acute pancreatitis** with a high likelihood of pancreatic necrosis?

(A) Pancreatic oedema only (CT severity index 2)

(B) Peripancreatic fat stranding (CTSI 3)

(C) $>$30% non-enhancement of pancreatic parenchyma with peripancreatic fluid collections (CTSI $\geq$7)

(D) Mild peri-pancreatic inflammation only (CTSI 4)

Q144. A 60-year-old male undergoes emergency laparotomy for perforated viscus. Post-operatively he develops oliguria ($<$0.5 mL/kg/hr for 8 hours), serum creatinine rises from baseline 0.9 mg/dL to 3.6 mg/dL. Urine sodium is 8 mEq/L, serum sodium 140 mEq/L, urine creatinine 340 mg/dL, serum creatinine 3.6 mg/dL. The **MOST likely cause** and **fractional excretion of sodium (FENa)** are:

(A) Intra-renal AKI (acute tubular necrosis); FENa $>$2%

(B) Post-renal AKI (obstruction); FENa $>$2%

(C) Intra-renal AKI (acute tubular necrosis); FENa <1%

(D) Pre-renal AKI (hypovolaemia); FENa <1%

Q145. A 55-year-old diabetic female is found to have serum creatinine 4.8 mg/dL, eGFR 14 mL/min/1.73m^2, serum potassium 5.9 mEq/L, bicarbonate 16 mEq/L, Hb 7.8 g/dL, MCV 82 fL, serum ferritin 28 ng/mL, transferrin saturation 12%. The **MOST appropriate treatment** for her anaemia is:

(A) IV iron supplementation followed by erythropoiesis-stimulating agent (ESA) if Hb remains <10 g/dL

(B) Oral iron only

(C) Blood transfusion immediately

(D) ESA alone without iron

Q146. A 28-year-old woman with SLE (ANA+, anti-dsDNA titre 1:640) develops worsening proteinuria (3.5 g/24 hours), serum albumin 2.1 g/dL, creatinine 1.8 mg/dL, and bilateral pitting pedal oedema. Renal biopsy shows diffuse proliferative glomerulonephritis with subendothelial immune deposits (wire loop lesions). This corresponds to:

(A) ISN/RPS Class II (mesangial proliferative lupus nephritis)

(B) ISN/RPS Class IV (diffuse proliferative lupus nephritis) — treat with high-dose steroids + cyclophosphamide or mycophenolate mofetil

(C) ISN/RPS Class V (membranous lupus nephritis)

(D) ISN/RPS Class III (focal proliferative lupus nephritis)

Q147. A 72-year-old hypertensive male with known atrial fibrillation (not on anticoagulation) presents 1.5 hours after sudden onset of left-sided weakness (arm > leg), left facial droop, and expressive aphasia. BP 168/95 mmHg, blood glucose 110 mg/dL, INR 1.0. Non-contrast CT brain shows no haemorrhage, no early ischaemic changes. The **MOST appropriate treatment** is:

(A) Aspirin 300 mg immediately + clopidogrel

(B) Immediate heparin infusion

(C) IV alteplase (tPA) 0.9 mg/kg within the 4.5-hour window + anticoagulation after 24 hours

(D) Supportive care only; thrombolysis contraindicated in AF

Q148. A 19-year-old college student presents with 48-hour history of severe headache, neck stiffness, photophobia, and non-blanching purpuric rash on the trunk and limbs. Temperature 39.8°C. CSF analysis: opening pressure 300 mmH_2O, appearance turbid, WBC 4000/mm^3 (90% polymorphs), protein 280 mg/dL, glucose 28 mg/dL (blood glucose 90 mg/dL), Gram stain shows Gram-negative diplococci. The **MOST likely organism** and immediate treatment are:

(A) *Streptococcus pneumoniae*; cefotaxime + dexamethasone

(B) *Listeria monocytogenes*; ampicillin + gentamicin

(C) *Cryptococcus neoformans*; amphotericin B + flucytosine

(D) *Neisseria meningitidis*; IV benzylpenicillin or ceftriaxone + contact prophylaxis with rifampicin

Q149. A 16-year-old girl has daily episodes of brief (<30 seconds) staring spells during which she is unresponsive, followed by immediate return to normal activity without post-ictal confusion. EEG shows 3 Hz generalised spike-and-wave discharges. She is currently not on any medication. The **drug of choice** for this seizure type is:

(A) Ethosuximide

(B) Carbamazepine

(C) Phenytoin

(D) Levetiracetam

Q150. A 68-year-old retired engineer presents with a 3-year history of resting tremor in the right hand (pill-rolling), bradykinesia, rigidity, and shuffling gait with festination. Examination reveals masked facies, micro-

graphia, and positive pull test. Cognition is intact. The **MOST appropriate initial pharmacological treatment** is:

(A) Donepezil

(B) Levodopa/Carbidopa

(C) Propranolol

(D) Haloperidol

Q151. A 22-year-old male with Type 1 DM presents with 2-day history of vomiting, abdominal pain, polyuria, and confusion. Examination: Kussmaul breathing, fruity odour of breath, BP 90/60 mmHg, HR 120 bpm. Investigations: blood glucose 520 mg/dL, pH 7.12, bicarbonate 8 mEq/L, anion gap 24 mEq/L, serum ketones 4+, serum potassium 5.8 mEq/L. The **MOST critical initial step** in management (before insulin) is:

(A) Start insulin infusion at 0.1 U/kg/hr immediately

(B) Administer sodium bicarbonate to correct acidosis

(C) IV 0.9% normal saline fluid resuscitation (1–1.5 L in first hour)

(D) Give IV potassium 40 mEq/hr immediately

Q152. A 38-year-old female presents with 6-month history of weight gain (8 kg), constipation, cold intolerance, menorrhagia, dry skin, and delayed ankle reflex relaxation. TSH is 42 mIU/L (normal 0.4–4.0), free T4 is 0.4 ng/dL (normal 0.8–1.8), anti-TPO antibody 320 IU/mL (positive). The **treatment of choice** and **target TSH** during replacement therapy are:

(A) Liothyronine (T3) alone; target TSH 0.1–0.5 mIU/L

(B) Carbimazole; target TSH $<$0.1 mIU/L

(C) Propylthiouracil (PTU) + iodine

(D) Levothyroxine (T4); target TSH 0.5–2.5 mIU/L

Q153. A 35-year-old male presents with 6-month history of progressive fatigue, weight loss, hyperpigmentation of skin creases, buccal mucosa and surgical scars, postural hypotension (BP 100/60 supine, 80/50 standing), hyponatraemia (Na 128 mEq/L), and hyperkalaemia (K 5.8 mEq/L). Short

synacthen test (ACTH stimulation) shows a peak cortisol of 12 mcg/dL at 30 minutes (normal >18). The **MOST likely diagnosis** and **treatment** are:

(A) Primary adrenal insufficiency (Addison's disease); hydrocortisone + fludrocortisone

(B) Secondary adrenal insufficiency (pituitary); hydrocortisone only (no mineralocorticoid needed)

(C) Cushing's syndrome; metyrapone

(D) Conn's syndrome; spironolactone

Q154. A 25-year-old pregnant woman (G2P1) at 28 weeks gestation has Hb 8.2 g/dL, MCV 65 fL, MCH 19 pg, serum ferritin 6 ng/mL, TIBC 480 mcg/dL. Peripheral smear shows microcytic hypochromic red cells with pencil cells and occasional target cells. Her previous child had thalassaemia trait. Mentzer index (MCV/RBC count) = 12. The **MOST likely diagnosis** and the **confirmatory test** are:

(A) Anaemia of chronic disease; serum EPO level

(B) Iron deficiency anaemia; serum ferritin + TIBC (already diagnostic)

(C) Thalassaemia trait; HPLC for haemoglobin analysis

(D) Sideroblastic anaemia; bone marrow biopsy

Q155. A 22-year-old female presents with photosensitive butterfly-shaped facial rash, oral ulcers, polyarthralgia, and haematuria. Investigations: ANA 1:640 (homogeneous pattern), anti-dsDNA positive, complement C3 low, urine protein 2.0 g/24 hr, serum creatinine 1.6 mg/dL, haemoglobin 9.4 g/dL (Coombs positive). Direct Coombs test is positive. She fulfils SLICC 2012 criteria. The **haematological feature** in this patient that contributes to the SLICC score is:

(A) Thrombocytopenia (<100,000)

(B) Leukopenia (<4000)

(C) Autoimmune haemolytic anaemia (Coombs positive)

(D) Lymphopenia (<1000)

Q156. A 40-year-old female with an 8-year history of seropositive rheumatoid arthritis (RF+, anti-CCP 320 U/mL) has failed methotrexate (20 mg/week) + leflunomide combination at 6 months. She has active disease (DAS28 5.8, swollen joint count 12/28, CRP 42 mg/L). The **NEXT step** in management according to EULAR guidelines is:

(A) Add hydroxychloroquine to the existing regimen

(B) Switch to sulfasalazine + hydroxychloroquine triple therapy

(C) Increase methotrexate to maximum dose (25 mg/week) and wait another 6 months

(D) Add a biologic DMARD (TNF-alpha inhibitor such as etanercept or adalimumab) or JAK inhibitor (tofacitinib)

Q157. A 52-year-old male presents with an acute attack of severe pain, swelling, and erythema of the right first metatarsophalangeal joint (podagra) for 24 hours. Serum uric acid is 9.8 mg/dL. Synovial fluid analysis reveals negatively birefringent needle-shaped crystals under polarised light. He has a history of two previous similar attacks and has tophi over both Achilles tendons. The **MOST appropriate acute treatment** followed by long-term urate-lowering therapy (ULT) target is:

(A) NSAIDs (indomethacin) or colchicine for acute attack; allopurinol for ULT with target serum uric acid <6 mg/dL

(B) Aspirin for acute attack; febuxostat for ULT

(C) IV methylprednisolone; no ULT needed if only two attacks

(D) Probenecid alone; no NSAID needed in acute phase

Q158. A 30-year-old male HIV-positive patient (CD4 count 180 cells/mm^3) presents with 3-week cough, night sweats, fever, and weight loss. CXR shows bilateral infiltrates and a right upper lobe cavity. Sputum AFB smear is negative $\times 3$. The **MOST appropriate NEXT diagnostic test** to confirm TB is:

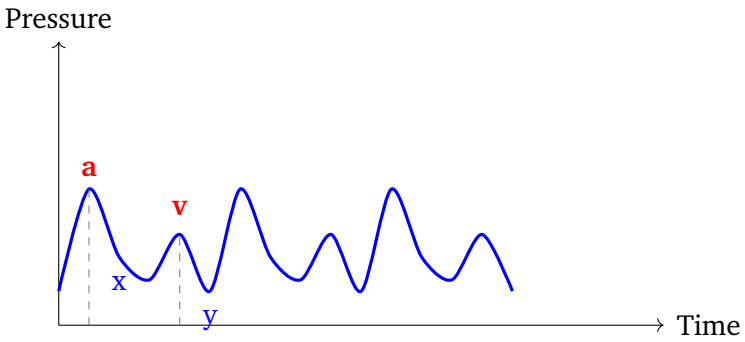
(A) Tuberculin skin test (Mantoux)

(B) Sputum GeneXpert MTB/RIF assay

(C) IGRA (Interferon-Gamma Release Assay) blood test

(D) Bronchoscopy with BAL for fungal culture only

Q159. A 60-year-old patient is assessed for elevated JVP. The JVP waveform is shown below:



In a patient with **tricuspid regurgitation**, the JVP waveform characteristically shows:

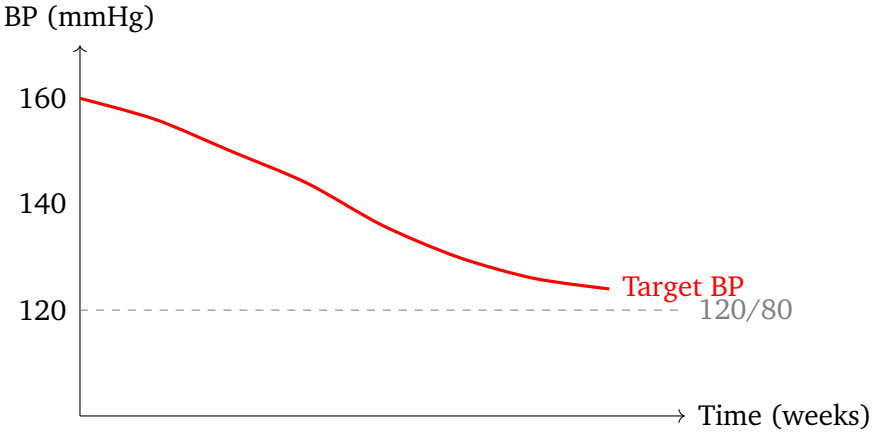
(A) Absent 'a' wave

(B) Cannon 'a' waves

(C) Prominent 'cv' wave with obliteration of 'x' descent

(D) Prominent 'y' descent with normal 'a' wave (Friedrich's sign)

Q160. A 48-year-old male of African origin with hypertension (BP 158/96) and no other comorbidities is started on a calcium channel blocker. The mechanism of action of **amlodipine** in lowering blood pressure is:



(A) Blocks beta-1 receptors, reducing cardiac output

(B) Inhibits ACE, reducing angiotensin II production

(C) Blocks alpha-1 receptors, causing vasodilatation

(D) Blocks L-type voltage-gated calcium channels in vascular smooth muscle, causing vasodilatation and reduced peripheral vascular resistance

Q161. A 21-year-old male college student is brought by his family with a 6-month history of social withdrawal, talking to himself, believing that his neighbours are conspiring to harm him (persecutory delusions), and hearing voices commenting on his actions (third-person auditory hallucinations). He is not on any medications and has no history of drug use. His IQ is normal. The **MOST likely diagnosis** and **drug of choice** for first-episode psychosis are:

(A) Schizophrenia; atypical antipsychotic (risperidone or olanzapine)

(B) Bipolar disorder with psychotic features; lithium + haloperidol

(C) Delusional disorder; psychotherapy alone

(D) Brief psychotic disorder; observe and discharge

Q162. A 30-year-old female has a history of two previous depressive episodes and now presents with a 2-week history of decreased need for sleep (3 hours and feeling rested), grandiose beliefs (believes she is a famous actress), pressured speech, flight of ideas, disinhibited behaviour, and spending large sums of money recklessly. She is not distressed. The **MOST appropriate treatment** for acute mania is:

(A) SSRIs (fluoxetine)

(B) Lithium or valproate + an antipsychotic (olanzapine/risperidone) for acute mania

(C) Benzodiazepine alone for 4 weeks

(D) ECT as first-line treatment

Q163. A 45-year-old female schoolteacher presents with 8-week history of persistent low mood, anhedonia (loss of interest in activities she previously enjoyed), early morning awakening, diurnal variation (worse in the morning), psychomotor retardation, poor concentration, and passive suicidal ideation ("life is not worth living") without a specific plan. PHQ-9 score is 18. The **MOST appropriate initial treatment** for moderate-to-severe MDD is:

(A) CBT alone (no medication needed for moderate severity)

(B) Benzodiazepine for anxiety component first, then reassess

(C) SSRI (sertraline or escitalopram) + CBT combination; assess suicide risk and consider safety planning

(D) Electroconvulsive therapy (ECT) immediately

Q164. A 28-year-old male presents with intrusive thoughts about contamination (ego-dystonic), spending 3 hours a day washing his hands (compulsive ritual) to neutralise anxiety. He recognises these thoughts as irrational. He has tried to resist but cannot. This causes significant occupational impairment. The **defence mechanism** that BEST describes the ritual of handwashing (undoing the contamination thought) is:

(A) Repression

(B) Projection

(C) Displacement

(D) Undoing

Q165. A 40-year-old male chronic heavy drinker is admitted after abruptly stopping alcohol 36 hours ago. He presents with tremor, profuse sweating, tachycardia (HR 118 bpm), BP 158/96 mmHg, confusion, agitation, and generalised tonic-clonic seizure. The **MOST appropriate pharmacological treatment** for alcohol withdrawal and prevention of Wernicke's encephalopathy is:

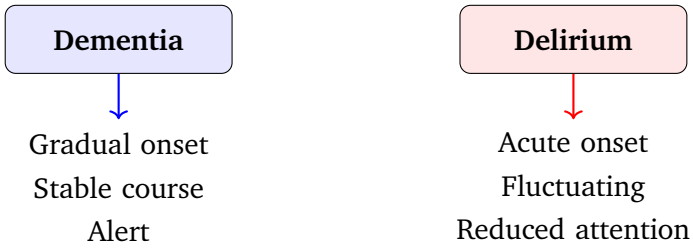
(A) IV thiamine (before glucose) + benzodiazepines (chlordiazepoxide or diazepam) using symptom-triggered or fixed-schedule regimen

(B) Haloperidol + IV glucose only

(C) Naltrexone for craving reduction immediately

(D) Disulfiram starting within 24 hours of last drink

Q166. An 80-year-old male with known moderate Alzheimer's disease is admitted to hospital following a hip fracture. On the 2nd post-operative day, his family notices he is more confused, agitated at night (sundowning), with fluctuating consciousness, inattention, and visual hallucinations (seeing people in the room). His pre-admission cognitive baseline was mild-to-moderate decline. The **feature MOST consistent with superimposed delirium** (rather than dementia progression) is:



(A) Gradually progressive memory loss over 2 years

(B) Acute onset with fluctuating consciousness and inattention

(C) Presence of language difficulties (aphasia)

(D) Personality change over years

Q167. A 55-year-old male, recently separated, unemployed, with a history of two previous suicide attempts (one involving a serious method), presents with hopelessness, a specific plan (he has saved up medications), and states he has "nothing to live for." He drinks heavily (1 bottle/day). Based on **established suicide risk factors**, the **MOST appropriate immediate action** is:

(A) Provide outpatient counselling and safety contract; send home with family

(B) Prescribe an antidepressant and review in 1 week

(C) Admit to inpatient psychiatric unit for observation and safety (voluntary or involuntary); remove access to means; initiate treatment

(D) Refer for CBT; no immediate hospitalisation needed

Q168. A 32-year-old IT professional presents with recurrent episodes of sudden-onset palpitations, chest tightness, shortness of breath, dizziness, trembling, and an overwhelming fear of dying that peaks within 10 minutes and then gradually subsides. Between episodes, he worries constantly about having another attack and has started avoiding public transport. Cardiac evaluation including Holter monitoring is normal. The **MOST likely diagnosis** and first-line pharmacotherapy are:

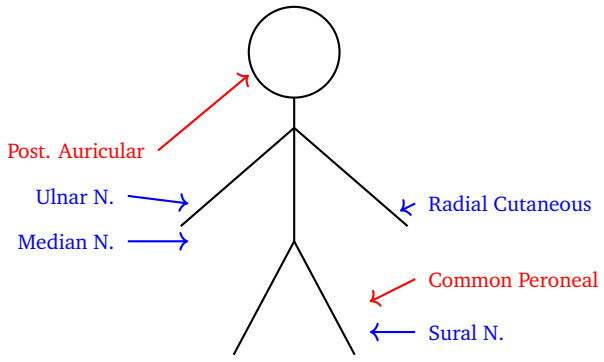
(A) Generalised anxiety disorder; buspirone

(B) Social phobia; beta-blockers (atenolol)

(C) Specific phobia; systematic desensitisation

(D) Panic disorder; SSRI (paroxetine or escitalopram) + CBT (panic-focused)

Q169. A 30-year-old male from rural Bihar presents with hypo-pigmented anaesthetic patches over the trunk, right ulnar nerve thickening with clawing of the 4th and 5th fingers (ulnar claw hand), and right common peroneal nerve thickening causing foot drop. Skin biopsy shows foamy macrophages loaded with acid-fast bacilli. The diagram below shows the commonly enlarged nerves in leprosy:



Commonly thickened nerves (red = most common)

The leprosy type in this patient (foamy macrophages, high bacillary load) is:

(A) Lepromatous leprosy (LL) — multibacillary, poor cell-mediated immunity

(B) Tuberculoid leprosy (TT) — paucibacillary, strong CMI

(C) Borderline lepromatous (BL)

(D) Pure neuritic leprosy

Q170. A 35-year-old male presents with well-defined erythematous plaques with silvery-white scales over the elbows, knees, and scalp for the past 2 years. Auspitz sign is positive. Nail examination shows pitting, onycholysis, and oil-drop sign. PASI score is 18. The **FIRST-LINE treatment** for extensive plaque psoriasis (PASI >10) in an outpatient setting is:

(A) Topical betamethasone alone indefinitely

(B) Systemic methotrexate (MTX) 10–25 mg/week + folic acid 5 mg/week; monitor LFTs

(C) Biologics (adalimumab) as first-line

(D) Cyclosporine A without prior phototherapy

Q171. A 45-year-old female presents with painful oral erosions for 3 months, followed by the development of flaccid blisters over the trunk and back that rupture easily leaving raw erosive areas. Nikolsky sign is positive. Biopsy shows suprabasal acantholysis. Direct immunofluorescence (DIF) reveals net-like (fishnet) IgG and C3 deposits around epidermal cells. The **MOST likely diagnosis** and **autoantibody** are:

(A) Bullous pemphigoid; anti-BP180 (anti-BPAG2) antibody

(B) Dermatitis herpetiformis; IgA anti-endomysial antibody

(C) Pemphigus vulgaris; anti-desmoglein 3 (anti-Dsg3) IgG antibody

(D) Linear IgA bullous dermatosis; IgA antibody

Q172. A 28-year-old male started on carbamazepine for epilepsy 3 weeks ago presents with fever, painful red erosions of the mouth, eyes (conjunctival injection), and genitalia, with extensive skin detachment. Nikolsky sign is positive. BSA skin detachment is approximately 35%. This is:

(A) Stevens-Johnson Syndrome (SJS) — BSA detachment $<10\%$

(B) Erythema multiforme major — target lesions, $<10\%$ BSA

(C) Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS)

(D) Toxic Epidermal Necrolysis (TEN) — BSA detachment $>30\%$; treat in burns ICU, withdraw causative drug, supportive care; consider IVIG or cyclosporine

Q173. A 7-year-old child presents with intensely pruritic lesions, worse at night, involving the web spaces of fingers, wrists, axillae, and genitalia. Several family members have similar complaints. Dermoscopy reveals a triangular dark structure at the end of a burrow ("delta wing jet" sign of the mite). The **drug of choice** and correct treatment protocol are:

(A) Permethrin 5% cream applied from neck down; leave for 8–12 hours; repeat after 1 week; treat all household contacts simultaneously

(B) Topical ivermectin 1% only; repeat daily for 5 days

(C) Benzyl benzoate 25% in children without dilution

(D) Oral doxycycline for 14 days

Q174. A 25-year-old male presents with a painless indurated ulcer (chancre) on the glans penis with bilateral non-tender inguinal lymphadenopathy. He gives history of unprotected sexual contact 3 weeks ago. VDRL is reactive (1:32), FTA-ABS is positive. Which of the following is the **drug of choice** for primary syphilis?

(A) Azithromycin 1 g single dose

(B) Benzathine penicillin G 2.4 million units IM single dose

(C) Doxycycline 100 mg BD for 14 days (only if penicillin allergy)

(D) Ceftriaxone 1 g IM daily for 10 days

Q175. A 20-year-old female student presents with sharply demarcated chalk-white depigmented patches over the face, dorsum of hands, and around

the mouth, progressively enlarging over 2 years. Wood's lamp examination shows bright white fluorescence. She is otherwise healthy. The **pathogenesis** of vitiligo involves:

(A) IgE-mediated mast cell degranulation

(B) Type II hypersensitivity (antibody-dependent cytotoxicity)

(C) Autoimmune destruction of melanocytes by CD8+ T cells and autoantibodies against tyrosinase

(D) Infectious destruction of melanocytes by a spirochaete

Q176. A 4-year-old boy presents with recurrent itchy, erythematous, oozing patches over the cheeks, antecubital fossae, and popliteal fossae, present since infancy. His mother has bronchial asthma. IgE is elevated (480 IU/mL). Skin prick test positive to house dust mite. The **MOST appropriate stepwise treatment** for moderate atopic dermatitis in a child, according to international guidelines (NICE/ETFAD):

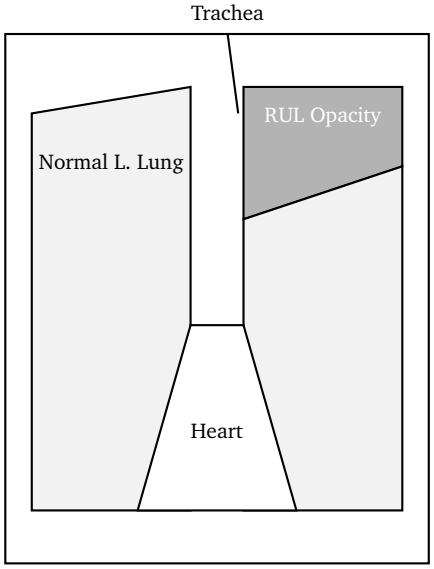
(A) Systemic corticosteroids (prednisolone) as first-line

(B) Topical tacrolimus (calcineurin inhibitor) only; avoid moisturisers

(C) Oral antihistamine alone

(D) Regular emollients (moisturisers) + mid-potency topical corticosteroid for flares; topical calcineurin inhibitor (tacrolimus/pimecrolimus) for face and flexures

Q177. A 55-year-old male smoker presents with breathlessness and a chest X-ray as schematically shown below:



Trachea deviated to right (towards collapse)

The CXR pattern (opacification of right upper zone, elevated right hilum, tracheal shift to the right) is MOST consistent with:

(A) Right upper lobe collapse (atelectasis)

(B) Right-sided pleural effusion

(C) Right upper lobe consolidation (pneumonia)

(D) Right-sided tension pneumothorax

Q178. A 65-year-old male presents with sudden onset headache ("worst headache of my life") and neck stiffness. CT brain (non-contrast) is performed within 2 hours of onset. The **MOST sensitive CT finding** in the first 6 hours of subarachnoid haemorrhage (SAH) is:

(A) Hypodense (dark) area in the basal cisterns

(B) Hyperdense (bright white) blood in the basal cisterns and subarachnoid spaces

(C) Midline shift to the left

(D) Ring-enhancing lesion with mass effect

Q179. A 50-year-old female with known seafood allergy is scheduled for a contrast-enhanced CT scan of the abdomen. The radiologist prescribes pre-medication. Which of the following is the **MOST appropriate pre-medication protocol** to reduce the risk of contrast reaction?

(A) Antihistamine (chlorpheniramine) alone 1 hour before the procedure

(B) IV adrenaline (epinephrine) prophylactically before contrast injection

(C) Prednisolone 50 mg orally 13, 7, and 1 hours before the procedure + diphenhydramine 50 mg IM 1 hour before (ACR protocol)

(D) Avoid contrast entirely; MRI without contrast is equivalent

Q180. A radiology department is reviewing annual radiation dose limits for staff. According to the **Atomic Energy Regulatory Board (AERB)** guidelines for radiation workers in India, the annual effective dose limit for occupational exposure (averaged over 5 consecutive years) is:

(A) 5 mSv/year

(B) 10 mSv/year

(C) 15 mSv/year

(D) 20 mSv/year

Q181. A 22-year-old tall, thin male presents with sudden onset right-sided chest pain and dyspnoea while at rest. He has no prior lung disease. CXR shows a visible pleural line with absent lung markings lateral to it on the right side; the right lung edge is 3 cm from the chest wall at the level of the hilum. Trachea is central, BP is normal. The **MOST appropriate management** for this **primary spontaneous pneumothorax** is:

(A) Needle aspiration or intercostal drain (ICD) if the pneumothorax is >2 cm (BTS guidelines) — drain for moderate/large

(B) Immediate thoracotomy

(C) 100% oxygen only; observe for 4 hours and discharge

(D) Bilateral ICD insertion empirically

Q182. A 45-year-old obese female presents with recurrent right upper quadrant colicky pain after fatty meals, nausea, and vomiting. Ultrasound of the

abdomen shows hyperechoic foci in the gallbladder with posterior acoustic shadowing and movement with change of position. Gallbladder wall is 2 mm (normal). CBD diameter is 4 mm. The **MOST likely diagnosis** and the **investigation of choice** to confirm CBD stones (if bilirubin rises) are:

(A) Gallbladder carcinoma; CT abdomen with contrast

(B) Cholelithiasis (gallstones); MRCP (Magnetic Resonance Cholangiopancreatography) to evaluate CBD

(C) Acute cholecystitis; HIDA scan

(D) Porcelain gallbladder; plain X-ray abdomen

Q183. A 68-year-old male with controlled hypertension (BP 136/84 on amlodipine), Type 2 DM (HbA1c 7.4%, on metformin), BMI 32, and no limitations in daily activities (able to climb 2 flights of stairs) is scheduled for elective laparoscopic cholecystectomy. His ECG shows normal sinus rhythm. His ASA Physical Status Classification is:

(A) ASA I — no systemic disease

(B) ASA II — mild systemic disease, no functional limitation

(C) ASA III — severe systemic disease with functional limitation

(D) ASA IV — constant threat to life

Q184. A 55-year-old patient receives rocuronium 1.2 mg/kg for rapid sequence intubation during emergency surgery. At end of surgery (60 minutes later), train-of-four (TOF) ratio is 0.2 (incomplete reversal). The **MOST appropriate agent** to reverse residual neuromuscular blockade from rocuronium is:

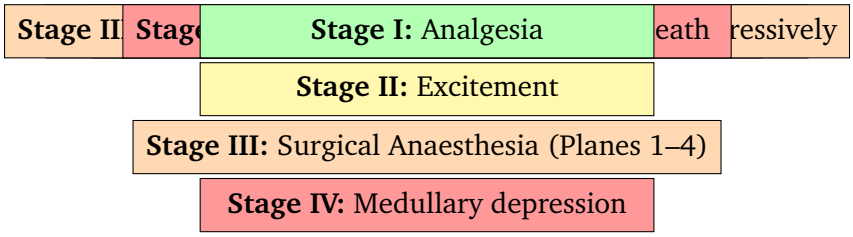
(A) Neostigmine + glycopyrrolate (anticholinesterase reversal)

(B) Edrophonium + atropine

(C) Atropine alone

(D) Sugammadex 4 mg/kg IV (encapsulates rocuronium; most complete reversal)

Q185. A patient undergoing inhalational induction with sevoflurane is observed to have the following signs: loss of consciousness, regular breathing, eyelash reflex absent, eyelid reflex present, pupils equal and mid-size, no response to verbal command, but still has laryngeal and pharyngeal reflexes intact (at risk of laryngospasm). According to **Guedel's stages of anaesthesia**, this corresponds to:



(A) Stage III, Plane 1 (surgical anaesthesia, laryngeal and pharyngeal reflexes still present)

(B) Stage I (analgesia — patient still conscious)

(C) Stage II (excitement — irregular breathing, risk of laryngospasm)

(D) Stage IV (medullary depression — respiratory arrest)

Q186. A 40-year-old patient is being maintained on isoflurane for general anaesthesia. The concept of **Minimum Alveolar Concentration (MAC)** is used to compare potency of inhalational agents. Which of the following statements about MAC is **CORRECT**?

(A) MAC is the concentration that prevents movement in 95% of patients (EC_{95})

(B) MAC is the end-tidal concentration (in % atm) that prevents movement in 50% of patients in response to a standard surgical incision; higher MAC = lower potency

(C) MAC is independent of age, opioid premedication, and body temperature

(D) MAC of nitrous oxide is less than 1% (very potent)

Q187. A 28-year-old primigravida undergoes spinal anaesthesia (intrathecal bupivacaine 2.5 mL of 0.5% heavy) for elective caesarean section. Twenty

minutes after the block, she develops severe hypotension (BP 80/50 mmHg), nausea, and bradycardia (HR 52 bpm). The **MOST likely cause** and **immediate treatment** are:

(A) High spinal block causing diaphragm paralysis; intubation immediately

(B) Total spinal anaesthesia; 100% oxygen

(C) Sympathetic blockade causing aortocaval compression + vasodilation; left lateral tilt + IV ephedrine or phenylephrine + IV fluid bolus

(D) Post-dural puncture headache (PDPH); blood patch immediately

Q188. A 24-year-old male receives succinylcholine + halothane for induction of anaesthesia for an emergency appendicectomy. Within 20 minutes, he develops masseter muscle rigidity, temperature rapidly rising (40.5°C), tachycardia (HR 145 bpm), muscle rigidity, and mixed respiratory and metabolic acidosis on ABG. Serum CK is markedly elevated (12,000 IU/L). The **MOST likely diagnosis** and **specific treatment** are:

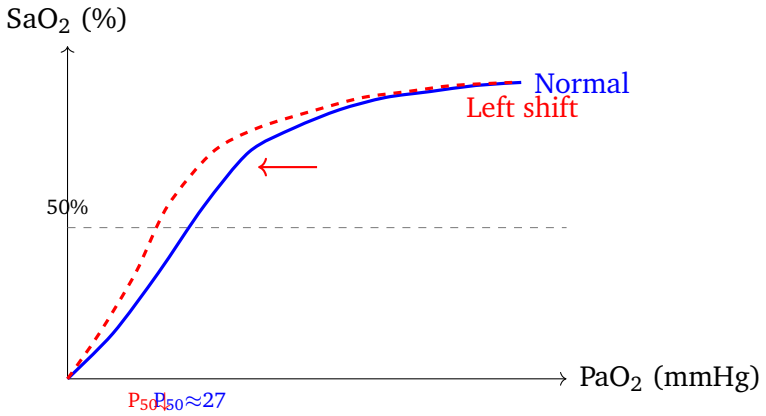
(A) Neuroleptic malignant syndrome; bromocriptine + dantrolene

(B) Thyroid storm; propylthiouracil + beta-blocker

(C) Serotonin syndrome; cyproheptadine

(D) Malignant hyperthermia; dantrolene sodium 2.5 mg/kg IV (up to 10 mg/kg) + stop triggering agents + cooling measures + correct acidosis

Q189. A 32-year-old mountaineer at high altitude develops hyperventilation with a resulting respiratory alkalosis (pH 7.52, $PaCO_2$ 28 mmHg). The effect on the oxygen-haemoglobin dissociation curve is shown below:



The **effect** of respiratory alkalosis on the oxygen-haemoglobin dissociation curve and **tissue oxygen delivery** is:

(A) Left shift of the curve; haemoglobin binds oxygen more avidly, P_{50} decreases, tissue oxygen unloading is *reduced* (Bohr effect)

(B) Right shift; haemoglobin releases oxygen more readily, P_{50} increases

(C) No shift; P_{50} unchanged by pH changes

(D) Left shift; tissue oxygenation is improved (increased oxygen release)

Q190. A 65-year-old male with severe COPD (FEV_1 38% predicted) is scheduled for transurethral resection of the prostate (TURP). The anaesthetist prefers neuraxial anaesthesia. Comparing **spinal** and **epidural** anaesthesia for this patient, which statement is **CORRECT**?

(A) Epidural anaesthesia has a faster onset than spinal

(B) Spinal anaesthesia has a faster, more predictable onset and denser block; epidural allows continuous dosing via catheter and titration; both avoid general anaesthesia and its risks in COPD

(C) Spinal anaesthesia is contraindicated with INR <1.2

(D) Epidural is always preferred over spinal for TURP because of TURP syndrome risk

Q191. A 32-year-old female (non-smoker, history of motion sickness) undergoes laparoscopic surgery under general anaesthesia with volatile agent and opioid analgesia. Post-operatively, she develops severe nausea and

vomiting (PONV) despite standard prophylaxis with ondansetron. Her Apfel score is 4 (all four risk factors present). The **drug mechanism** of **ondansetron** and a **rescue antiemetic** with a different mechanism are:

(A) Ondansetron: D2 receptor antagonist; rescue: granisetron

(B) Ondansetron: NK1 receptor antagonist; rescue: aprepitant

(C) Ondansetron: 5-HT_3 receptor antagonist; rescue: haloperidol (D2 antagonist) or dexamethasone

(D) Ondansetron: H1 receptor antagonist; rescue: cyclizine

Q192. A 65-year-old diabetic male with CKD (eGFR 35 mL/min/1.73m^2, serum creatinine 2.1 mg/dL) requires an urgent contrast-enhanced CT scan. The **MOST important preventive measure** against contrast-induced nephropathy (CIN) is:

(A) Administer acetylcysteine 600 mg BD for 2 days (only)

(B) Stop metformin 48 hours before (only measure needed)

(C) Use high-osmolar contrast (ionic) media as it is more stable

(D) Pre-procedure IV isotonic saline hydration (1 mL/kg/hr for 3–12 hours before and after), use iso-osmolar or low-osmolar non-ionic contrast, minimise contrast volume, hold metformin

Q193. A 35-year-old male presents with sudden-onset severe epigastric pain that has now shifted to the right iliac fossa over 12 hours. He has rebound tenderness and guarding at McBurney's point. Temperature is 38.2°C, WBC 16,500/mm^3 (neutrophilia). Rovsing's sign is positive. Alvarado score is 8. The **MOST likely diagnosis** and **treatment** are:

(A) Acute appendicitis; laparoscopic appendicectomy

(B) Perforated peptic ulcer; laparotomy

(C) Acute mesenteric ischaemia; CT angiography

(D) Renal colic; IV analgesia + ureteric stenting

Q194. A 2-year-old child from a food-insecure household presents with bilateral pitting pedal oedema, sparse depigmented hair that plucks easily (flag sign), flaky-paint dermatosis, hepatomegaly, and a miserable expression (not interested in surroundings). His weight-for-age Z-score is –3. Serum albumin is 1.8 g/dL. The **MOST likely diagnosis** and **distinguishing feature from marasmus** are:

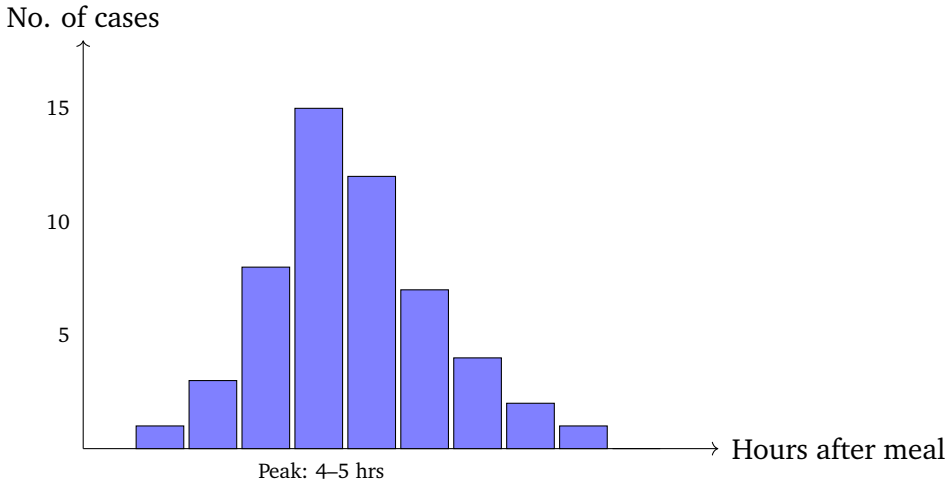
(A) Marasmus; absent oedema, severe wasting (bag of bones appearance)

(B) Kwashiorkor; presence of nutritional oedema, hypoalbuminaemia, adequate calorie intake with protein deficiency

(C) Marasmic kwashiorkor; the child is too thin to have kwashiorkor

(D) Nutritional rickets; bowing of legs is the hallmark

Q195. During a foodborne outbreak in a wedding function attended by 300 guests, cases of acute gastroenteritis (vomiting, diarrhoea, abdominal cramps) were reported. The epidemic curve (bar chart of cases by hour of onset) is shown below:



The epidemic curve pattern (sharp single peak, rapid rise and fall within hours) is **MOST consistent with**:

(A) Propagated person-to-person epidemic

(B) Continuous common-source epidemic (prolonged exposure)

(C) Point-source common-vehicle epidemic (single-exposure event)

(D) Mixed epidemic pattern

Q196. In the Universal Immunisation Programme (UIP) of India, which of the following correctly states the **vaccine given at birth**?

(A) BCG + OPV0 + Hepatitis B (birth dose) + Vitamin K

(B) OPV0 + DPT only

(C) BCG + Rotavirus vaccine

(D) BCG + OPV0 + Hepatitis B birth dose (the three vaccines given at birth under UIP)

Q197. A 5-year-old male child presents with recurrent episodes of haemarthrosis (joint bleeds), particularly involving the right knee, and prolonged bleeding after minor cuts. His parents are first cousins. PT is normal (13 seconds), aPTT is prolonged (72 seconds, control 32 seconds). Platelet count and bleeding time are normal. Factor VIII activity is 2% (severely reduced). The **MOST likely diagnosis** and **specific treatment** are:

(A) Haemophilia A; recombinant Factor VIII concentrate

(B) Haemophilia B; recombinant Factor IX concentrate

(C) von Willebrand disease; DDAVP (desmopressin)

(D) ITP (immune thrombocytopenic purpura); IV immunoglobulin

Q198. A 28-year-old sexually active female presents with 3-day history of burning micturition, frequency, urgency, and suprapubic pain. She has no fever and no flank pain. Urinalysis shows leucocytes 3+, nitrites positive, no casts. Urine culture is pending. She has no prior UTI in the past 6 months and no anatomical abnormality. The **MOST appropriate treatment** is:

(A) IV ceftriaxone for 14 days (complicated UTI)

(B) Nitrofurantoin 100 mg BD for 5 days OR trimethoprim-sulfamethoxazole for 3 days (uncomplicated lower UTI)

(C) Ciprofloxacin for 14 days (treat as pyelonephritis)

(D) No treatment; self-limiting condition; increase fluid intake only

Q199. A 35-year-old female presents with new-onset relapsing-remitting neurological symptoms including transient visual loss (optic neuritis), upper limb weakness, and sensory disturbances. An MRI brain and spine with gadolinium is ordered. The **MOST specific MRI finding** that supports a diagnosis of multiple sclerosis (MS) is:

(A) Single enhancing lesion in the cerebellum

(B) Diffuse cortical atrophy

(C) Multiple periventricular T2/FLAIR hyperintense lesions (Dawson's fingers) with lesions disseminated in space and time; one or more Gd-enhancing lesions

(D) Basal ganglia T1 hyperintense lesions

Q200. A 50-year-old female undergoes open abdominal hysterectomy under general anaesthesia. On Day 1 post-operatively, she reports pain score 8/10. The anaesthetist plans **multimodal analgesia**. Which of the following regimens **BEST** represents the multimodal approach to reduce opioid requirements (opioid-sparing) and side effects?

(A) IV morphine PCA (patient-controlled analgesia) alone

(B) IV paracetamol alone (regular 4-hourly)

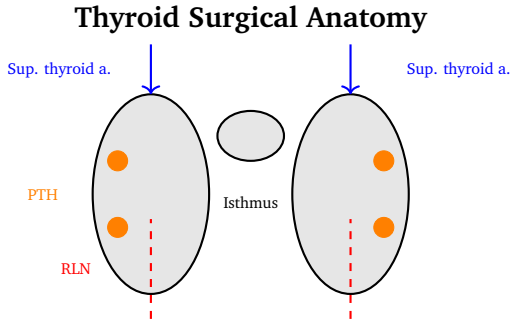
(C) IM diclofenac PRN only

(D) Regular paracetamol IV + NSAID (ketorolac or diclofenac) + epidural or local anaesthetic wound infiltration + low-dose opioid PRN for breakthrough pain

Section D — Clinical Surgery & Specialties (Q 201–300)

Q201. A 38-year-old woman presents with a midline anterior neck swelling that has been enlarging over 6 months. On examination, the swelling moves upward on swallowing. She has no voice change, dysphagia, or pressure

symptoms. Serum TSH is normal. The surgeon plans a total thyroidectomy. During surgery, the structure **MOST at risk** of injury that would cause hoarseness of voice is:



(A) Recurrent laryngeal nerve

(B) External branch of superior laryngeal nerve

(C) Internal branch of superior laryngeal nerve

(D) Superior thyroid artery

Q202. A 52-year-old man presents with a painless, hard thyroid nodule. FNAC shows cells with ground-glass nuclei, nuclear grooves, and psammoma bodies. Family history is negative. Serum calcitonin is normal. Which is the **MOST likely** histological type of thyroid carcinoma?

(A) Follicular carcinoma

(B) Papillary carcinoma

(C) Medullary carcinoma

(D) Anaplastic carcinoma

Q203. A 48-year-old woman is diagnosed with invasive ductal carcinoma of the right breast. Tumour size is 4 cm, 4 ipsilateral axillary lymph nodes are positive for metastasis, and chest CT shows no distant metastases. According to TNM staging, what is the **MOST likely** stage?

(A) Stage IIA

(B) Stage IIB

(C) Stage IIIA

(D) Stage IIIB

Q204. A 40-year-old woman presents with a 2 cm discrete, mobile, firm lump in the left breast. FNAC is performed. The cytology report shows Grade C5 findings. This grading is **MOST consistent** with:

(A) Benign — fibroadenoma

(B) Atypical cells, possibly malignant

(C) Suspicious of malignancy

(D) Definite malignancy

Q205. A 55-year-old woman undergoes modified radical mastectomy for carcinoma of the right breast. Two weeks post-operatively she develops painless, fluctuant, non-erythematous swelling in the axilla. The **MOST likely** complication is:

(A) Seroma

(B) Wound haematoma

(C) Wound dehiscence

(D) Lymphangitis

Q206. A 22-year-old man presents with a 24-hour history of periumbilical pain migrating to the right iliac fossa, nausea, and low-grade fever (37.8°C). On examination: Rovsing's sign positive, McBurney's point tenderness, and guarding. WBC is 14,500/μL with a left shift. The **MOST appropriate NEXT step** in management is:

(A) CT abdomen and pelvis with contrast

(B) Observation and serial abdominal examinations

(C) Emergency appendicectomy

(D) Colonoscopy

Q207. A 65-year-old man presents with colicky abdominal pain, absolute constipation, abdominal distension, and vomiting for 3 days. He had a previous laparotomy for peptic ulcer perforation 10 years ago. X-ray abdomen

supine shows multiple air-fluid levels with central ladder-pattern loops. The **MOST common cause** of this presentation is:

(A) Carcinoma of the colon

(B) Femoral hernia

(C) Adhesions from prior surgery

(D) Volvulus of the sigmoid colon

Q208. A 58-year-old man with no family history of colorectal cancer presents for routine check-up. He is asymptomatic. According to current screening guidelines for average-risk individuals, the **MOST appropriate** primary screening method to recommend at his age is:

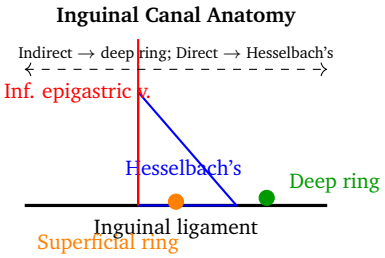
(A) Annual digital rectal examination alone

(B) Flexible sigmoidoscopy every 5 years

(C) Annual faecal occult blood test (FOBT)

(D) Colonoscopy every 10 years

Q209. A 35-year-old man presents with a right inguinal bulge appearing on straining. On examination, the swelling appears medial to the inferior epigastric vessels and does not descend into the scrotum. The deep inguinal ring is unoccupied. This hernia type passes through which anatomical structure?



(A) Deep inguinal ring

(B) Femoral canal

(C) Hesselbach's triangle (posterior wall of inguinal canal)

(D) Superficial inguinal ring only

Q210. A 60-year-old multiparous woman presents with a small, tense, irreducible lump in the upper medial thigh just below and lateral to the pubic tubercle. It appeared 2 days ago and is associated with severe colicky abdominal pain and vomiting. The **MOST likely** diagnosis is:

(A) Inguinal lymphadenopathy

(B) Strangulated femoral hernia

(C) Obturator hernia

(D) Saphena varix

Q211. A 30-year-old man presents with sudden onset severe generalised abdominal pain, board-like rigidity, and absent bowel sounds. He gives a 6-year history of epigastric pain relieved by antacids. Erect chest X-ray shows free air under both hemidiaphragms. The **MOST appropriate** immediate management is:

(A) IV proton pump inhibitor and nasogastric decompression

(B) H. pylori eradication therapy and close observation

(C) Emergency laparotomy with peritoneal lavage

(D) Endoscopy under sedation to identify the source

Q212. A 45-year-old obese woman presents with right upper quadrant pain radiating to the right shoulder tip, nausea, and fever (38.5°C) for 18 hours. On examination: Murphy's sign positive. LFT shows ALP 280 U/L, bilirubin 1.8 mg/dL, ALT 65 U/L. WBC 13,200/μL. The **BEST initial investigation** to confirm the diagnosis is:

(A) CT abdomen with contrast

(B) HIDA (hepatobiliary iminodiacetic acid) scan

(C) MRCP (magnetic resonance cholangiopancreatography)

(D) Ultrasound of the abdomen

Q213. A 55-year-old woman with known gallstones presents with Charcot's triad: right upper quadrant pain, jaundice, and fever with rigors. Total bilirubin is 8.2 mg/dL; ALP is 520 U/L; GGTP is elevated. Ultrasound

shows dilated CBD (12 mm). The **MOST appropriate NEXT step** in management is:

(A) ERCP with sphincterotomy and stone extraction

(B) Urgent open cholecystectomy

(C) Percutaneous transhepatic cholangiography (PTC)

(D) MRCP for further delineation only

Q214. A 48-year-old alcoholic man presents with haematemesis. On examination: splenomegaly, ascites, caput medusae, and jaundice. Upper GI endoscopy reveals large oesophageal varices (Grade III) with active bleeding. The **MOST effective immediate pharmacological measure** to control variceal bleeding is:

(A) Propranolol

(B) Terlipressin (vasopressin analogue)

(C) Spironolactone

(D) Omeprazole IV infusion

Q215. A 62-year-old man presents with painless progressive jaundice, significant weight loss (8 kg in 3 months), and a palpable, non-tender, distended gallbladder. Total bilirubin is 18 mg/dL; ALP is 900 U/L. CA 19-9 is elevated. CT scan shows a mass at the head of pancreas with dilated biliary tree. This clinical picture (palpable gallbladder + painless jaundice) is described by which eponymous sign?

(A) Charcot's sign

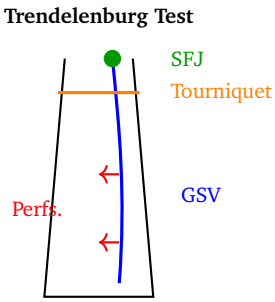
(B) Grey Turner's sign

(C) Courvoisier's sign

(D) Trousseau's sign

Q216. A 42-year-old man presents with dilated, tortuous superficial veins in the left lower leg, aching, heaviness, and occasional swelling. The Trendelenburg test (tourniquet test) is positive when the tourniquet is placed

at the saphenofemoral junction level. The **MOST likely** primary site of incompetence is:



(A) Short saphenous vein at saphenopopliteal junction

(B) Perforator veins of the lower leg (Cockett's perforators)

(C) Deep femoral vein valvular incompetence

(D) Saphenofemoral junction (SFJ) incompetence

Q217. A 55-year-old woman who underwent total hip replacement 5 days ago develops swelling, redness, warmth, and pitting oedema of the left calf. Homan's sign is equivocal. Her Wells score is 3 (high probability). The **BEST diagnostic investigation** to confirm deep vein thrombosis is:

(A) Compression duplex ultrasonography

(B) D-dimer assay

(C) Venography

(D) CT pulmonary angiography

Q218. A 65-year-old diabetic smoker presents with calf pain on walking 200 metres, relieved by rest. ABI (ankle-brachial index) is 0.55. On examination: absent popliteal pulse bilaterally, dependent rubor. This symptom is classified under which Fontaine stage?

(A) Fontaine Stage I — asymptomatic

(B) Fontaine Stage IIb — claudication at < 200 m

(C) Fontaine Stage III — rest pain

(D) Fontaine Stage IV — ulceration/gangrene

Q219. A 72-year-old hypertensive man presents with sudden severe tearing abdominal and back pain. On examination: pulsatile expansile midline abdominal mass, BP 90/60 mmHg, HR 120/min, pallor. The **MOST appropriate immediate management** is:

(A) CT angiography of the aorta urgently

(B) IV fluids resuscitation and urgent vascular surgery review

(C) Emergency open surgical repair

(D) Endovascular aortic repair (EVAR) electively

Q220. A 60-year-old obese diabetic man who underwent elective colectomy develops a serosanguinous discharge from his midline laparotomy wound on post-operative day 7. On examination, bowel loops are visible through the wound edges. This complication is described as:

(A) Wound seroma

(B) Wound haematoma

(C) Anastomotic leak

(D) Burst abdomen (complete wound dehiscence with evisceration)

Q221. A 55-year-old man with operable rectal carcinoma undergoes pre-operative staging. MRI pelvis shows the tumour is 5 cm from the anal verge, invades muscularis propria but not beyond, with one positive mesorectal lymph node, and no distant metastases. According to TNM staging, this is **MOST likely**:

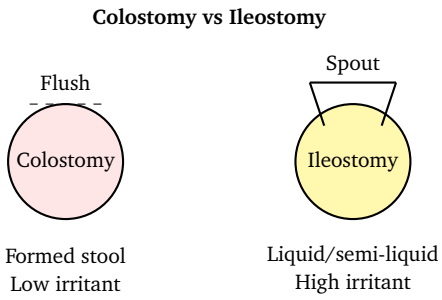
(A) T2N1M0 — Stage IIIA

(B) T3N0M0 — Stage IIA

(C) T4aN1M0 — Stage IIIC

(D) T2N0M0 — Stage I

Q222. A 50-year-old man undergoes Hartmann's procedure for perforated sigmoid diverticulitis. The resultant stoma is a sigmoid colostomy. Compared to an ileostomy, which of the following features is **MOST characteristic** of a colostomy?



(A) Raised spout (to protect skin from caustic effluent)

(B) Flush with the skin, producing formed stool

(C) Higher output (>500 mL/day) with severe electrolyte imbalance

(D) Located in the right iliac fossa typically

Q223. A 26-year-old primigravida at 10 weeks of gestation attends her first antenatal visit. Routine blood investigations are performed. Which of the following is **NOT** routinely offered at the first antenatal visit?

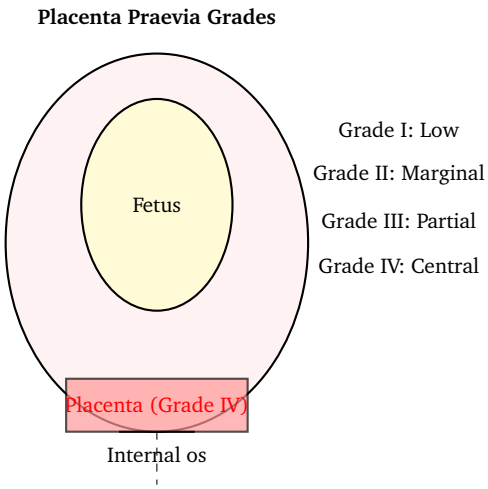
(A) Blood group and Rh typing

(B) VDRL for syphilis screening

(C) Glucose challenge test (GCT) for gestational diabetes

(D) HIV testing with counselling

Q224. A 32-year-old woman at 34 weeks of gestation presents with sudden onset of painless, bright red vaginal bleeding. There is no uterine tenderness. FHR tracing is reassuring. On USS, placenta is seen covering the internal os completely. The **MOST appropriate** immediate management is:



(A) Immediate digital vaginal examination to assess cervix

(B) Induction of labour with oxytocin

(C) Tocolysis with nifedipine and close monitoring

(D) Hospitalisation, bed rest, IV access, crossmatch, and plan for LSCS

Q225. A 28-year-old primigravida at 36 weeks presents with severe headache, visual disturbances, and epigastric pain. BP is 170/115 mmHg. Urinalysis shows 3+ proteinuria. Platelet count is 75,000/μL; ALT 120 U/L; LDH 800 U/L. The **MOST appropriate** immediate treatment to prevent seizures is:

(A) Magnesium sulphate IV

(B) Oral phenobarbitone

(C) IV diazepam

(D) Oral nifedipine alone

Q226. A 30-year-old woman at 26 weeks of gestation undergoes a 75 g oral glucose tolerance test (OGTT) as part of routine screening. Her fasting plasma glucose is 4.8 mmol/L, 1-hour value is 10.5 mmol/L, and 2-hour value is 8.8 mmol/L. According to IADPSG criteria, gestational diabetes mellitus is diagnosed when:

(A) Fasting $\geq$5.1 mmol/L or 1-hour $\geq$10.0 mmol/L or 2-hour $\geq$8.5 mmol/L

(B) Fasting $\geq$7.0 mmol/L or 2-hour $\geq$11.1 mmol/L

(C) 1-hour $\geq$11.1 mmol/L alone

(D) Any random glucose $\geq$11.1 mmol/L only

Q227. A 24-year-old woman with a history of PID presents with sudden severe right iliac fossa pain, vaginal bleeding (spotting), and syncope. LMP was 6 weeks ago. BP 80/50 mmHg, HR 130/min. Urine βhCG is positive. Pelvic USS shows no intrauterine pregnancy and free fluid in the pouch of Douglas. The **MOST appropriate NEXT step** is:

(A) Administer methotrexate IM

(B) Serial βhCG monitoring over 48 hours

(C) Emergency laparotomy/laparoscopy

(D) Diagnostic laparoscopy under elective conditions

Q228. A 38-year-old woman at 41 weeks of gestation is scheduled for induction of labour. Bishop score is assessed. Which of the following cervical parameters is **NOT** included in the Bishop score?

Bishop Score Components

Parameter	0	1	2	3
Dilation	Closed	1-2	3-4	5+
Effacement	0-30	40-50	60-70	80+
Station	-3	-2	-1/0	+1/2
Consistency	Firm	Med	Soft	–
Position	Post	Mid	Ant	–

(A) Cervical dilation

(B) Fetal station

(C) Cervical effacement

(D) Fetal heart rate pattern

Q229. A 25-year-old primipara delivers a 3.8 kg baby vaginally. Twenty minutes after delivery, she has not delivered the placenta, and then develops

torrential vaginal bleeding. Estimated blood loss is 900 mL. On examination: uterus is soft and boggy. BP 90/60 mmHg. The **MOST common cause** of primary postpartum haemorrhage is:

(A) Uterine atony

(B) Retained placenta

(C) Vaginal lacerations

(D) Coagulopathy

Q230. A 22-year-old primigravida at term is in labour for 20 hours. Despite adequate uterine contractions, there is no progress of descent. On examination: cervix is fully dilated; fetal head is at 0 station; a Bandl's ring is palpable. The **MOST appropriate NEXT step** is:

(A) Administer additional oxytocin

(B) Emergency caesarean section

(C) Vacuum-assisted delivery

(D) Symphysiotomy

Q231. A 22-year-old woman presents with oligomenorrhoea, acne, hirsutism, and difficulty conceiving for 2 years. BMI is 28 kg/m^2. USS shows bilateral ovarian enlargement with 12 follicles each measuring 2–9 mm arranged peripherally (necklace sign). LH:FSH ratio is 2.5. According to Rotterdam criteria, PCOS is diagnosed when **at least two of three features** are present. Which set **MOST accurately** reflects these criteria?

(A) Elevated LH, low FSH, and PCO morphology on USS

(B) Oligomenorrhoea alone with high androgens

(C) Oligo-anovulation, hyperandrogenism (clinical/biochemical), and PCO morphology on USS

(D) Infertility, obesity, and acne

Q232. A 35-year-old woman presents with heavy menstrual bleeding (HMB), dysmenorrhoea, and urinary frequency. On bimanual examination, the

uterus is enlarged (14 weeks size), irregular, and non-tender. USS shows multiple intramural and subserosal myomas. Haemoglobin is 8.2 g/dL. The fibroid subtype **MOST likely** responsible for her heavy menstrual bleeding is:

(A) Subserosal fibroid

(B) Pedunculated fibroid

(C) Cervical fibroid

(D) Submucosal fibroid

Q233. A 28-year-old woman presents with progressive dysmenorrhoea starting 2–3 days before menstruation, deep dyspareunia, and secondary infertility. On laparoscopy, powder-burn lesions (black deposits) are seen on the uterosacral ligaments and ovary. There is a 4 cm endometrioma in the left ovary. The **MOST accurate diagnostic tool** for endometriosis is:

(A) Diagnostic laparoscopy with histological confirmation

(B) Serum CA-125 level

(C) MRI of the pelvis

(D) Transvaginal ultrasound

Q234. A 30-year-old sexually active woman attends for routine cervical screening. She has never had an abnormal smear. Current Indian/WHO guidelines for cervical cancer screening recommend which of the following as **preferred** primary screening method?

(A) Annual Pap smear from age 18

(B) HPV DNA testing (co-testing or primary HPV test) every 5 years from age 25–30

(C) Colposcopy every 2 years

(D) VIA (visual inspection with acetic acid) annually

Q235. A 55-year-old postmenopausal woman presents with abdominal distension, bilateral adnexal masses, and ascites. CA-125 is 620 U/mL. CT

abdomen-pelvis shows peritoneal deposits and omental caking. The **MOST common** histological type of ovarian carcinoma is:

(A) Mucinous cystadenocarcinoma

(B) Endometrioid carcinoma

(C) High-grade serous carcinoma

(D) Clear cell carcinoma

Q236. A 42-year-old woman presents with irregular, heavy menstrual bleeding occurring at unpredictable intervals. There is no pelvic mass. Endometrial biopsy shows simple hyperplasia without atypia. The PALM-COEIN classification of abnormal uterine bleeding (AUB) categorises this patient's cause under which structural category?

(A) Polyp (AUB-P)

(B) Adenomyosis (AUB-A)

(C) Leiomyoma (AUB-L)

(D) Endometrial (AUB-E)

Q237. A 32-year-old woman who is breastfeeding her 8-week-old infant wishes to start contraception. She has a history of deep vein thrombosis. She prefers a highly effective, reversible, non-hormonal method that does not affect lactation. The **BEST** contraceptive option for her is:

(A) Copper intrauterine device (Cu-IUD)

(B) Combined oral contraceptive pill

(C) Levonorgestrel IUS (Mirena)

(D) Depot medroxyprogesterone acetate injection

Q238. A couple presents with 2 years of primary infertility. Semen analysis: volume 2.5 mL, count 10 million/mL, motility 30

(A) Azoospermia

(B) Oligoasthenoteratozoospermia (OAT syndrome)

(C) Asthenospermia only

(D) Normospermia with female factor infertility

Q239. A 29-year-old woman in active labour at 40 weeks has continuous electronic fetal monitoring. The CTG shows: baseline FHR 130 bpm, moderate variability (6–15 bpm), accelerations present, and variable decelerations (lasting 30–60 seconds, recovering to baseline). This CTG tracing is classified as:

(A) Normal/reassuring — no action needed

(B) Pathological — emergency delivery required

(C) Suspicious/non-reassuring — requires further evaluation

(D) Sinusoidal — immediate delivery

Q240. A term neonate is delivered by emergency LSCS for fetal distress. At birth: no cry, floppy, cyanotic, HR 60 bpm, irregular gasping respirations. APGAR at 1 minute is 2. After drying, warming, and clearing the airway, what is the **MOST appropriate NEXT step**?

(A) Administer epinephrine IV immediately

(B) Perform cardiac compressions immediately

(C) Start positive pressure ventilation (PPV) with 100

(D) Intubate and give surfactant

Q241. A 34-year-old woman at 32 weeks develops tonic-clonic convulsions at home. She was known to have BP 150/100 mmHg and 2+ proteinuria at her last visit. She is brought to hospital. BP now is 180/120 mmHg. The immediate priority is:

(A) Control seizures with IV magnesium sulphate and stabilise

(B) Emergency lower segment caesarean section immediately

(C) IV diazepam and observe for 24 hours

(D) Administer corticosteroids for fetal lung maturity first

Q242. A 30-year-old primigravida at 33 weeks presents with epigastric pain, vomiting, and malaise. BP 155/105 mmHg. Investigations: haemoglobin 9.8 g/dL, platelet 62,000/μL, LDH 1,020 U/L, AST 185 U/L, peripheral smear shows schistocytes. This presentation is **MOST consistent** with:

(A) Acute fatty liver of pregnancy

(B) HELLP syndrome

(C) ITP (idiopathic thrombocytopenic purpura)

(D) Haemolytic uraemic syndrome

Q243. A 24-year-old primigravida (O negative blood group) delivers a healthy Rh-positive baby. Which of the following is the **MOST appropriate** prophylactic measure to prevent Rh sensitisation in future pregnancies?

(A) Anti-D immunoglobulin 50 μg within 72 hours of delivery

(B) Anti-D immunoglobulin 300 μg within 72 hours of delivery

(C) Blood transfusion to the mother

(D) No action required as first pregnancy is usually safe

Q244. A 22-year-old primigravida is in active labour. Cervical dilation is 5 cm with intact membranes. Uterine contractions are 3 in 10 minutes, each lasting 40 seconds. After 4 hours, repeat examination shows cervical dilation at 6 cm. Fetal descent has not changed. This pattern is **MOST consistent** with:

(A) Normal progress — Friedman's curve shows this is acceptable

(B) Latent phase prolongation

(C) Precipitate labour

(D) Active phase arrest (failure to progress)

Q245. A 15-month-old child is brought by parents concerned about developmental delay. On assessment: the child can walk independently, says 2–3 words with meaning, uses a pincer grasp, and waves bye-bye. Which developmental milestone is **appropriate** for 15 months?

(A) Walking independently

(B) Saying full sentences of 4–5 words

(C) Drawing a triangle

(D) Riding a tricycle

Q246. A term neonate (day 3 of life) has visible jaundice. Total serum bilirubin is 14 mg/dL; direct bilirubin 0.5 mg/dL. Baby is breastfeeding well, urine is clear yellow, stools are yellow. Blood group: mother O positive, baby A positive. DCT (direct Coombs test) is negative. The **MOST likely diagnosis** is:

(A) ABO incompatibility haemolytic disease

(B) Physiological jaundice of newborn

(C) Biliary atresia

(D) Breast milk jaundice

Q247. A 2-year-old child from a rural area has weight 7 kg (expected 12 kg for age). He has generalised oedema, moon face, flaky paint dermatosis, and flag sign in hair. Serum albumin is 1.8 g/dL. This clinical picture is **MOST consistent** with:

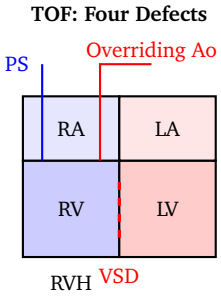
(A) Marasmus

(B) Marasmic-kwashiorkor

(C) Kwashiorkor

(D) Iron deficiency anaemia

Q248. A 4-year-old child is brought with exertional dyspnoea and episodic cyanotic spells (hypercyanotic spells) that are relieved by squatting. On examination: systolic thrill at left sternal border, single S2, and an ejection systolic murmur. CXR shows boot-shaped heart (coeur en sabot) with decreased pulmonary vascular markings. The **MOST likely diagnosis** is:



(A) Ventricular septal defect (VSD) — isolated

(B) Transposition of great arteries (TGA)

(C) Atrial septal defect (ASD) — large

(D) Tetralogy of Fallot (TOF)

Q249. A 6-week-old healthy infant is brought for immunisation. According to the Universal Immunisation Programme (UIP) of India, which vaccines are due at **6 weeks of age**?

(A) OPV-1, Pentavalent-1 (DPT+HepB+Hib), RVV-1, PCV-1, fIPV-1

(B) OPV-0, BCG, Hepatitis B birth dose

(C) MMR-1 and Varicella

(D) OPV-3, Pentavalent-3, RVV-3

Q250. A 14-month-old child presents with 5 days of watery diarrhoea (8–10 stools/day). On examination: sunken eyes, reduced skin turgor, dry mucous membranes, HR 140/min, but no shock. The child is crying but consolable. This degree of dehydration is classified as **moderate**. The **RECOMMENDED treatment** according to WHO IMCI guidelines is:

WHO ORS Composition (Low-Osmolarity)	
Sodium chloride:	2.6 g/L
Trisodium citrate:	2.9 g/L
Potassium chloride:	1.5 g/L
Glucose anhydrous:	13.5 g/L
Osmolarity:	245 mOsm/L

(A) IV Ringer's lactate 100 mL/kg over 3 hours (Plan C)

(B) ORS 75 mL/kg over 4 hours in a health facility (Plan B)

(C) ORS at home, no monitoring needed (Plan A)

(D) Normal saline 20 mL/kg bolus stat then ORS

Q251. A 2.5-year-old child develops a generalised tonic-clonic seizure lasting 2 minutes associated with fever of 39.5°C. There is no neck stiffness or focal deficit. The seizure stops spontaneously. This is the first episode. The **MOST appropriate NEXT step** in management is:

(A) Lumbar puncture immediately to rule out meningitis

(B) Start long-term anticonvulsant therapy (phenobarbitone)

(C) Paracetamol for fever control, reassurance, and parental education

(D) MRI brain to exclude a structural cause

Q252. A 4-year-old unvaccinated child presents with high fever, coryza, conjunctivitis, and Koplik's spots on the buccal mucosa. Three days later, a maculopapular rash appears behind the ears and spreads downward. The **MOST serious preventable complication** of measles in malnourished children is:

(A) Otitis media

(B) Febrile convulsions

(C) Parotitis

(D) Pneumonia and/or encephalitis

Q253. A 6-month-old unvaccinated infant presents with a 3-week history of paroxysmal cough followed by a high-pitched inspiratory whoop, post-tussive vomiting, and apnoeic spells. WBC is 35,000/μL with 80

(A) Azithromycin

(B) Amoxicillin

(C) Co-trimoxazole

(D) Cephalexin

Q254. A 6-month-old infant presents with poor feeding, failure to thrive, recurrent chest infections, and excessive sweating during feeds. On auscultation: harsh pansystolic murmur at the lower left sternal border, grade 4/6, with a mid-diastolic murmur at the apex. CXR shows cardiomegaly and increased pulmonary vascular markings. The **MOST likely diagnosis** is:

(A) ASD (atrial septal defect) — Ostium secundum

(B) Large VSD (ventricular septal defect) with left-to-right shunt

(C) PDA (patent ductus arteriosus)

(D) Pulmonary stenosis

Q255. A 2-day-old neonate presents with lethargy, poor feeding, respiratory distress, and temperature instability (hypothermia 35.8°C). Mother had prolonged rupture of membranes (28 hours). WBC is 3,200/μL (leukopenia), C-reactive protein 48 mg/L, blood culture is pending. The **MOST common causative organism** of early-onset neonatal sepsis in India is:

(A) Staphylococcus aureus

(B) Listeria monocytogenes

(C) Klebsiella pneumoniae

(D) Group B Streptococcus (GBS)

Q256. A 5-year-old partially immunised child presents with low-grade fever, sore throat, and a thick, greyish-white membrane on the tonsils that bleeds on attempted removal. Bull neck appearance is noted. The child develops stridor. The **MOST dangerous complication** in the acute phase of diphtheria is:

(A) Parotitis

(B) Otitis media

(C) Peritonsillar abscess

(D) Laryngeal obstruction and myocarditis

Q257. A 18-month-old child from a slum area presents with severe wasting, loose skin folds, prominent ribs, and an alert but anxious facial expression — "old man's face". Weight is 55

(A) Grade III malnutrition (severe) — marasmus

(B) Grade II malnutrition (moderate) — kwashiorkor

(C) Grade I malnutrition (mild) — marasmic-kwashiorkor

(D) Normal nutritional status

Q258. A preterm neonate (30 weeks gestation) at day 5 of life has increasing oxygen requirements and a continuous machine-like murmur best heard at the left upper sternal border/infraclavicular area. Echocardiography confirms PDA with significant left-to-right shunting. The **drug of choice** for pharmacological closure in a preterm neonate is:

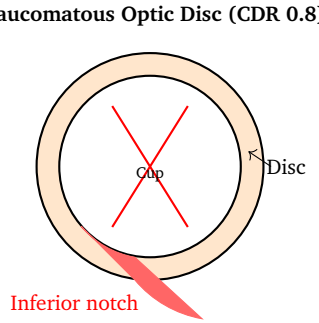
(A) Furosemide

(B) Indomethacin or Ibuprofen (COX inhibitor)

(C) Digoxin

(D) Sildenafil

Q259. A 60-year-old man presents with gradual painless loss of peripheral vision. IOP by Goldmann applanation tonometry is 26 mmHg bilaterally. Gonioscopy shows open angles. Fundus examination shows cup-disc ratio 0.8 bilaterally with inferior notching of the neuroretinal rim. Visual field testing shows arcuate scotoma. The **MOST likely diagnosis** is:



(A) Acute angle-closure glaucoma

(B) Ocular hypertension without glaucoma

(C) Primary open-angle glaucoma (POAG)

(D) Secondary glaucoma from uveitis

Q260. A 65-year-old man undergoes uncomplicated phacoemulsification with posterior chamber IOL implantation. Two months post-operatively he develops gradual painless blurring of vision. Slit-lamp examination shows posterior capsule opacity (PCO) — wrinkled posterior capsule with Elschnig's pearls. The **MOST appropriate treatment** is:

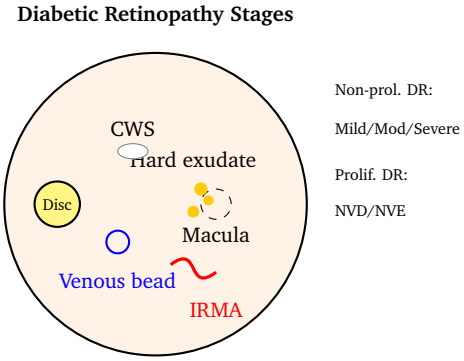
(A) Repeat cataract surgery

(B) Topical corticosteroid eye drops for 6 weeks

(C) Anterior vitrectomy

(D) Nd:YAG laser posterior capsulotomy

Q261. A 50-year-old man with 15-year history of Type 2 DM presents with blurring of central vision. Fundoscopy shows: cotton wool spots, hard exudates within one disc diameter of the fovea, intraretinal microvascular abnormalities (IRMA), and venous beading — but no new vessels. This is classified as:



(A) Severe non-proliferative diabetic retinopathy (NPDR)

(B) Mild NPDR

(C) Moderate NPDR

(D) Proliferative diabetic retinopathy (PDR)

Q262. A 45-year-old myope presents with sudden onset of flashing lights (photopsia) and floaters followed by a dark curtain-like shadow progressing

from the periphery toward the centre of vision. Visual acuity is 6/60 in the affected eye. Fundoscopy shows a grey, billowing retina with a break at the superotemporal quadrant. The **MOST likely type** of retinal detachment is:

(A) Exudative (secondary) retinal detachment

(B) Rhegmatogenous retinal detachment

(C) Tractional retinal detachment

(D) Retinoschisis

Q263. A 3-year-old child from a tribal area presents with difficulty seeing in dim light. On examination, foamy, pearly-white triangular plaques are seen on the nasal and temporal conjunctiva bilaterally (Bitot's spots). Cornea appears dry and lustreless. This condition is caused by deficiency of:

(A) Vitamin D

(B) Vitamin C

(C) Vitamin A

(D) Vitamin B12

Q264. A 30-year-old contact lens wearer presents with a painful red eye, photophobia, and severe purulent discharge. Slit lamp shows a central corneal ulcer with dense stromal infiltrate and a hypopyon (2 mm). Corneal scraping Gram stain shows Gram-negative rods. The **MOST appropriate treatment** is:

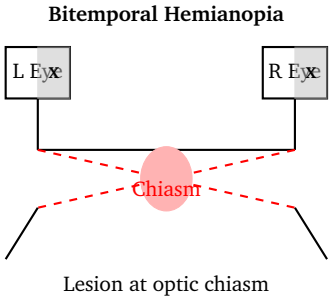
(A) Topical antifungal (natamycin) hourly

(B) Topical acyclovir ointment 5 times daily

(C) Oral acyclovir and topical steroids

(D) Intensive topical fluoroquinolone (ciprofloxacin/moxifloxacin) hourly

Q265. A 55-year-old woman with a pituitary macroadenoma on MRI develops progressive vision loss. Visual field testing (Goldmann perimetry) shows bitemporal hemianopia. The **anatomical location** of the lesion causing this defect is:



(A) Optic chiasm (compression by pituitary tumour)

(B) Right optic nerve

(C) Right optic tract

(D) Left occipital cortex

Q266. A 28-year-old man with HLA-B27 positive ankylosing spondylitis presents with a painful, red eye with photophobia, lacrimation, and decreased vision. Slit-lamp examination: ciliary injection, keratic precipitates (fine, non-granulomatous), and posterior synechiae. The **MOST likely diagnosis** is:

(A) Acute conjunctivitis — bacterial

(B) Anterior uveitis (iridocyclitis)

(C) Posterior uveitis (choroiditis)

(D) Scleritis

Q267. A 4-year-old child is brought with the left eye turning inward (esotropia) noted by parents. On the cover-uncover test, when the right eye is covered, the left eye (deviated eye) makes an outward movement to fix on the target. When the cover is removed, the left eye drifts inward again. The right eye does not move when the left is covered. This finding indicates:

(A) Paralytic (non-comitant) squint

(B) Intermittent squint of the right eye

(C) Unilateral concomitant squint (manifest esotropia of the left eye)

(D) Pseudosquint (epicanthal folds only)

Q268. A 72-year-old woman presents with progressive central vision loss and distortion (metamorphopsia) in her right eye. On fundoscopy: multiple drusen deposits in the macular area, and an area of subretinal neovascular membrane with subretinal haemorrhage. OCT shows fluid under the neuroretinal layer. This is classified as:

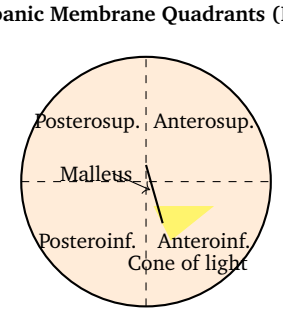
(A) Dry (atrophic) ARMD — geographic atrophy

(B) Central serous retinopathy

(C) Macular hole

(D) Wet (neovascular/exudative) ARMD

Q269. A 5-year-old child presents with right earache, fever (39°C), and decreased hearing for 3 days after an upper respiratory tract infection. On otoscopy: right tympanic membrane is bulging, red, and opaque with loss of cone of light and landmarks. The **MOST appropriate** initial treatment for acute suppurative otitis media is:



(A) Amoxicillin for 5–7 days plus analgesics

(B) Myringotomy and grommet insertion

(C) Topical antibiotic ear drops only

(D) Immediate mastoidectomy

Q270. A 45-year-old man presents with progressive hearing loss in the right ear following a road traffic accident with head injury. On tuning fork tests (512 Hz): Rinne's test is AC < BC (Rinne's negative) on the right; Weber's test lateralises to the right ear. This pattern is **MOST consistent** with:

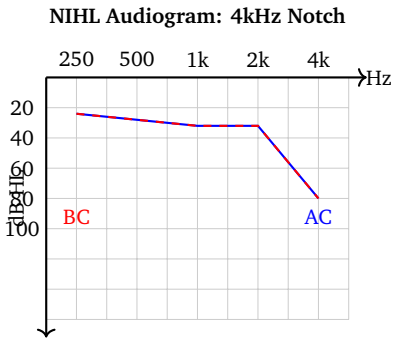
(A) Sensorineural hearing loss in the right ear

(B) Conductive hearing loss in the right ear

(C) Sensorineural hearing loss in the left ear

(D) Mixed hearing loss bilaterally

Q271. An audiogram of a 55-year-old man who works in a noisy factory shows: Air conduction thresholds elevated bilaterally (worst at 4000 Hz — a notch pattern), bone conduction thresholds mirror image of air conduction with no air-bone gap. Speech discrimination is poor. The **MOST likely diagnosis** is:



(A) Otosclerosis

(B) Age-related hearing loss (presbycusis)

(C) Noise-induced hearing loss (NIHL)

(D) Meniere's disease

Q272. A 35-year-old man presents with a 10-year history of foul-smelling, scanty, unilateral ear discharge. He now develops headache and swelling behind the right ear (mastoid). On otoscopy: retraction pocket in the posterior superior quadrant with white pearly mass and a marginal perforation. CT mastoid shows bone erosion. The **MOST likely complication** of untreated cholesteatoma is:

(A) Serous otitis media

(B) Eustachian tube dysfunction

(C) Tympanosclerosis

(D) Intracranial extension — meningitis or brain abscess

Q273. A 38-year-old man with known bronchial asthma and aspirin sensitivity presents with bilateral nasal blockage, anosmia, and clear rhinorrhoea for 2 years. Anterior rhinoscopy reveals pale, gelatinous masses in both nasal cavities originating from the middle meatus. The **drug of choice** for nasal polyps is:

(A) Topical intranasal corticosteroids (e.g., mometasone)

(B) Systemic antihistamines

(C) Antibiotic (amoxicillin-clavulanate) for 6 weeks

(D) Topical decongestant (oxymetazoline) long term

Q274. A 25-year-old man presents with spontaneous left-sided anterior epistaxis. The bleeding point is identified on the anteroinferior nasal septum (Little's area / Kiesselbach's plexus). He is haemodynamically stable. The **MOST appropriate initial management** is:

(A) Posterior nasal packing with Foley catheter balloon

(B) Compression of the soft part of the nose for 10–20 minutes and chemical cauterisation with silver nitrate

(C) Urgent embolisation of the internal maxillary artery

(D) IV tranexamic acid and blood transfusion

Q275. A 10-year-old child with acute frontal sinusitis develops progressive proptosis, oedema and erythema of the eyelids, restricted ocular motility, and fever. CT orbit shows a collection between the periorbita and orbital walls. The **MOST likely complication** is:

(A) Pre-septal (periorbital) cellulitis

(B) Cavernous sinus thrombosis

(C) Subperiosteal abscess of the orbit

(D) Mucocele of the frontal sinus

Q276. A 62-year-old male smoker (40 pack-years) presents with hoarseness of voice for 6 months, now with dysphagia and right neck lymph node enlargement. Indirect laryngoscopy shows an irregular growth on the right true vocal cord extending to the supraglottis. Biopsy confirms squamous cell carcinoma. This tumour (supraglottic) has which **lymphatic drainage** making early nodal spread possible?

(A) No lymphatic drainage (glottis)

(B) Sparse lymphatic drainage — late metastasis

(C) Drains to submental nodes

(D) Rich bilateral lymphatic drainage to deep cervical nodes

Q277. A 50-year-old man is admitted to the ICU following Guillain-Barré syndrome with progressive respiratory failure. He has been intubated for 14 days. The team is considering tracheostomy. The **MOST important indication** for tracheostomy in this scenario is:

(A) Prolonged mechanical ventilation requiring long-term airway access

(B) Acute upper airway obstruction only

(C) To facilitate oral feeding

(D) Laryngeal tumour resection

Q278. A 2-year-old child is brought to the emergency department with sudden onset choking, coughing, and stridor after playing with peanuts. He is conscious, able to cry, and has partial airway obstruction. The **MOST appropriate immediate manoeuvre** for a conscious child with partial airway obstruction is:

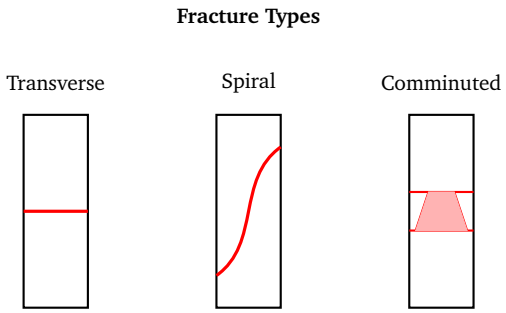
(A) Blind finger sweep in the mouth

(B) 5 back blows alternating with 5 chest thrusts (for child >1 year: abdominal thrusts after back blows)

(C) Emergency tracheostomy

(D) Immediate rigid bronchoscopy in the ED

Q279. A 25-year-old motorcyclist sustains a twisting injury to the right tibia. X-ray shows a fracture line that spirals around the bone shaft at an oblique angle over a long segment. This type of fracture is classified as:



(A) Transverse fracture

(B) Comminuted fracture

(C) Spiral fracture

(D) Greenstick fracture

Q280. A 28-year-old man with closed fracture of the right femur develops, 48 hours after conservative management, acute confusion, dyspnoea (SpO2 88

(A) Pulmonary thromboembolism

(B) ARDS from sepsis

(C) Haemothorax from rib fractures

(D) Fat embolism syndrome (Gurd's criteria)

Q281. A 55-year-old osteoporotic woman falls on an outstretched hand. She presents with a dinner-fork deformity of the right wrist, pain, and swelling. X-ray shows a fracture of the distal radius with dorsal displacement, dorsal angulation, radial deviation, and impaction. This is **MOST consistent** with:

(A) Colles' fracture

(B) Smith's fracture

(C) Barton's fracture

(D) Monteggia fracture-dislocation

Q282. A 16-year-old boy presents with progressive pain and swelling around the left knee for 3 months. X-ray shows a destructive lesion in the distal femur metaphysis with periosteal reaction (Codman's triangle and sunburst pattern) and soft tissue extension. Serum ALP is markedly elevated. Biopsy shows malignant osteoid production. The **MOST likely diagnosis** is:

(A) Giant cell tumour of bone

(B) Osteosarcoma

(C) Ewing's sarcoma

(D) Chondrosarcoma

Q283. A 19-year-old man sustains a closed fracture of the right tibial shaft managed in a plaster cast. Three hours later he develops severe unrelenting pain, worse on passive stretching of the toes, with a tense swollen leg, paraesthesia in the foot, and pallor. Compartment pressure is 45 mmHg (diastolic BP - compartment pressure < 30 mmHg). The **MOST appropriate treatment** is:

(A) Elevate the limb and administer IV morphine

(B) Apply ice packs and observe for 4 hours

(C) Emergency fasciotomy of all four compartments of the leg

(D) Urgent angiography to rule out vascular injury

Q284. A 45-year-old man on long-term high-dose prednisolone for SLE presents with progressive right hip pain and limited range of movement over 8 months. MRI right hip shows a subchondral crescent sign and collapse of the femoral head. The **MOST likely diagnosis** is:

(A) Osteoarthritis of the hip

(B) Septic arthritis

(C) Rheumatoid arthritis

(D) Avascular necrosis (AVN) of the femoral head

Q285. A 10-year-old boy presents with a 5-day history of fever, exquisite tenderness, and swelling over the proximal tibia. He refuses to weight-bear. WBC is 18,000/μL, ESR 80 mm/hr, CRP 95 mg/L. X-ray shows periosteal elevation after 10 days. The **MOST common causative organism** of acute haematogenous osteomyelitis in this age group is:

(A) Staphylococcus aureus

(B) Streptococcus pyogenes

(C) Salmonella typhi

(D) Pseudomonas aeruginosa

Q286. A 30-year-old man sustains a fracture of the mid-shaft humerus in a road traffic accident. Following plaster cast application, he develops wrist drop and inability to extend the fingers at the metacarpophalangeal joints. Sensation is impaired over the dorsum of the hand and thumb web space. The **MOST likely nerve injured** is:

Seddon Classification of Nerve Injuries		
Neuropraxia	Axonotmesis	Neurotmesis
Conduction block only	Axon disrupted, Endoneurium intact	Complete nerve division
Full recovery	Recovery possible	No spontaneous recovery
Weeks	Months	Surgery needed

(A) Median nerve

(B) Radial nerve

(C) Ulnar nerve

(D) Musculocutaneous nerve

Q287. A 22-year-old man falls on an outstretched abducted, externally rotated arm. He presents with the arm held in abduction and external rotation,

loss of shoulder contour, and a palpable fullness in the axilla. He cannot internally rotate or adduct the arm. The **MOST common type** of shoulder dislocation is:

(A) Posterior dislocation

(B) Inferior dislocation (luxatio erecta)

(C) Anterior glenohumeral dislocation

(D) Superior dislocation

Q288. A 14-year-old boy presents with pain, swelling, and fever over the mid-shaft of the right femur. X-ray shows a permeative destructive lesion in the diaphysis with multilayered periosteal reaction (onion-skin appearance) and a soft tissue mass. ESR and LDH are elevated. The **MOST likely diagnosis** is:

(A) Osteosarcoma

(B) Acute osteomyelitis

(C) Giant cell tumour

(D) Ewing's sarcoma

Q289. During a pre-operative anaesthetic assessment, a 45-year-old obese woman opens her mouth maximally and protrudes her tongue. The anaesthetist can see only the soft palate with no uvula or faucial pillars visible. According to Mallampati classification, this is:

(A) Class III (difficult intubation predicted)

(B) Class I (easy intubation)

(C) Class II (moderate difficulty)

(D) Class IV (impossible intubation without adjuncts)

Q290. A 35-year-old healthy woman is scheduled for elective laparoscopic cholecystectomy under general anaesthesia. The surgeon requests intraperitoneal CO2 insufflation to 14 mmHg. Considering the risk of aspiration, which airway device is **PREFERRED**?

(A) Laryngeal mask airway (LMA) — supraglottic device

(B) Endotracheal tube (ETT) with cuffed tube for airway protection

(C) Nasopharyngeal airway only

(D) Oropharyngeal airway (Guedel) only

Q291. A 28-year-old woman undergoes laparoscopic gynaecological surgery under general anaesthesia with opioids and nitrous oxide. In the recovery room she develops severe nausea and vomiting (PONV). Her Apfel PONV risk score is 4 (female sex, non-smoker, history of PONV, opioid use). The **FIRST-LINE antiemetic** recommended for PONV prophylaxis in high-risk patients is:

(A) Haloperidol IV

(B) Metoclopramide IV

(C) Ondansetron IV (5HT3 antagonist)

(D) Promethazine IM

Q292. A 60-year-old man undergoes elective open abdominal aortic aneurysm repair. Post-operatively he is expected to have severe pain. The anaesthetist plans multimodal analgesia. Which of the following regional anaesthesia techniques provides the **BEST** post-operative analgesia for open abdominal surgery?

(A) Femoral nerve block

(B) IV morphine PCA alone

(C) Intercostal nerve block at one level

(D) Epidural analgesia (thoracic epidural catheter)

Q293. A 40-year-old man presents with sudden-onset severe abdominal pain and guarding. An erect chest X-ray is performed. The radiological sign that **MOST specifically** indicates a perforated hollow viscus on erect CXR is:

(A) Air under the right hemidiaphragm (Pneumoperitoneum)

(B) Air-fluid levels in the colon only

(C) Double bubble sign

(D) Sail sign

Q294. A 2-week-old neonate presents with cyanosis, tachypnoea, and poor feeding from birth. CXR shows a figure-of-8 or snowman-shaped cardiac silhouette. The **MOST likely diagnosis** associated with this X-ray finding is:

(A) Tetralogy of Fallot

(B) Total anomalous pulmonary venous connection (TAPVC) — supracardiac type

(C) Transposition of the great arteries

(D) Coarctation of the aorta

Q295. A neonate born to a mother with polyhydramnios presents with bilious vomiting from the first feed. AXR shows two gas-filled bubbles (stomach + proximal duodenum) with no distal bowel gas. Down syndrome features are noted on examination. The **MOST likely diagnosis** is:

(A) Pyloric stenosis

(B) Jejunal atresia

(C) Duodenal atresia

(D) Malrotation with midgut volvulus

Q296. A 55-year-old man is undergoing CT pulmonary angiography with IV iodinated contrast. Within 5 minutes he develops urticaria, facial oedema, stridor, and hypotension (BP 80/50 mmHg). This is classified as a severe anaphylactoid contrast reaction. The **MOST appropriate IMMEDIATE treatment** is:

(A) IV hydrocortisone 200 mg and IV antihistamine

(B) IV normal saline bolus alone

(C) Oral prednisolone and observe

(D) IM adrenaline (epinephrine) 0.5 mg (1:1000) and IV fluid resuscitation

Q297. A 2-year-old child with acute respiratory illness has a CXR showing thymic enlargement with a triangular opacity in the right upper mediastinum with a wavy lateral border (also called thymic wave sign). This radiological sign in children is known as:

(A) Sail sign (thymic sail sign)

(B) Hampton's hump

(C) Westermark's sign

(D) Air bronchogram sign

Q298. A 50-year-old man presents with progressive jaundice, dark urine, pale stools, and weight loss of 5 kg over 2 months. He has no pain. On examination: deep jaundice, palpable non-tender gallbladder. Investigations: total bilirubin 22 mg/dL (predominantly conjugated), ALP 850 U/L, ALT 120 U/L, CA 19-9 markedly elevated. CT scan shows a mass in the head of the pancreas. The **MOST appropriate surgical procedure** for potentially curable disease is:

(A) Whipple's procedure (pancreaticoduodenectomy)

(B) Distal pancreatectomy with splenectomy

(C) Billroth II gastrectomy

(D) ERCP with stenting only (palliative)

Q299. A 65-year-old hypertensive smoker presents with sudden severe tearing chest pain radiating to the back between the shoulder blades, unequal blood pressures in both arms (right 170 mmHg, left 120 mmHg), and aortic regurgitation murmur on auscultation. ECG shows no ST changes. The **MOST appropriate investigation** to confirm the diagnosis urgently is:

(A) ECG and troponin levels

(B) CT angiography of the aorta (CT aortogram)

(C) Coronary angiography

(D) Transthoracic echocardiography alone

Q300. A 70-year-old man with known diabetes presents from the emergency department with fever (40°C), confusion, HR 130/min, BP 80/50 mmHg, respiratory rate 28/min, SpO2 90

(A) CT abdomen first to identify the source before antibiotics

(B) Administer norepinephrine and monitor for 2 hours

(C) Obtain central venous access and monitor CVP

(D) IV broad-spectrum antibiotics within 1 hour + IV fluid resuscitation + oxygen

Detailed Solutions

Q1.

Solution

Concept — Dermatomal map of the upper limb: The medial aspect of the forearm is supplied by the medial cutaneous nerve of the forearm (C8–T1), and the medial one-and-a-half fingers are supplied by the ulnar nerve (C8–T1). Both distributions map to the C8–T1 dermatomes, corresponding to the lower trunk of the brachial plexus.

Step 1 — Identifying the distribution: Medial forearm + medial 1.5 fingers = ulnar and medial cutaneous nerve of forearm territory = C8–T1.

Step 2 — Matching to nerve root: C8 and T1 form the lower trunk; sensory loss in this distribution localises to these roots.

Why other options are wrong:

- Option A: Correct.
- Option B: C6–C7 supplies the lateral forearm and middle/index fingers (median nerve territory).
- Option C: C5–C6 covers the lateral arm and forearm (musculocutaneous territory).
- Option D: T2–T3 is the axilla and medial upper arm, not the forearm or fingers.

Final Answer: Medial forearm and medial 1.5 fingers = C8–T1 dermatomes $\Rightarrow$ A

Answer: (A) Go Back to Q 1

Q2.

Solution

Concept — Contents of the posterior triangle of the neck: The accessory nerve (CN XI) runs through the posterior triangle, supplying trapezius and sternocleidomastoid. Damage to CN XI causes inability to shrug the shoulder (trapezius weakness) and winging of the scapula when pushing against a wall.

Step 1 — Identifying the deficit: Inability to abduct the shoulder beyond 90° (trapezius weakness) + scapular winging on forward pushing = CN XI palsy.

Step 2 — Anatomical correlation: CN XI is superficial in the posterior triangle

and vulnerable during lymph node surgery.

Why other options are wrong:

- Option A: Phrenic nerve arises from C3–C5 in the anterior triangle; injury causes hemi-diaphragm paralysis, not scapular winging.
- Option B: Correct.
- Option C: Greater auricular nerve (C2–C3) provides cutaneous supply to the ear; it does not innervate any muscle.
- Option D: Dorsal scapular nerve (C5) supplies rhomboids and levator scapulae; its injury causes medial scapular winging, but it does not course through the posterior triangle in a surgically accessible position.

Final Answer: Posterior triangle lymph node biopsy $\rightarrow$ accessory nerve (CN XI) injury $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 2

Q3.

Solution

Concept — Brachial plexus: lower trunk and its named branches: The lower trunk is formed by C8 and T1. Unlike the upper trunk (which gives the suprascapular nerve and nerve to subclavius), the lower trunk itself gives no major named branches. The medial pectoral nerve, medial cutaneous nerve of arm (MCNA), and medial cutaneous nerve of forearm (MCNFA) arise from the medial cord, which is derived from the lower trunk's anterior division.

Step 1 — Avulsion at the lower trunk (point X): Avulsion of C8 and T1 roots at the lower trunk level abolishes function of the medial cord and, combined with posterior cord contribution, affects all terminal branches.

Step 2 — Named branches from the lower trunk: The lower trunk per se does not give named pre-divisional branches. The medial cord (from the lower trunk anterior division) gives medial pectoral nerve, MCNA, MCNFA, ulnar nerve, and contributes to the median nerve.

Why other options are wrong:

- Option A: Correct — the lower trunk contributes to medial cord; medial pectoral and medial cutaneous nerves originate from this cord/root level.
- Option B: Upper trunk gives suprascapular nerve and nerve to subclavius — not the lower trunk.

- Option C: The middle trunk (C7) gives no pre-divisional named branches — partially correct statement but wrong trunk identification in this question.
- Option D: Long thoracic (C5–C7) and dorsal scapular (C5) arise from the roots of the upper and middle portions of the plexus, not the lower trunk.

Final Answer: Lower trunk (C8–T1); medial pectoral and medial cutaneous nerves arise from the medial cord derived from this trunk $\Rightarrow$ A

Answer: (A) Go Back to Q 3

Q4.

Solution

Concept — Lenticulostriate arteries and the middle cerebral artery: The lenticulostriate (lateral striate) arteries are perforating branches of the M1 segment of the middle cerebral artery (MCA). They supply the basal ganglia and internal capsule, and are the classic site of hypertensive intracerebral haemorrhage.

Step 1 — Identify the parent vessel: Lenticulostriate = lateral striate arteries = perforators of the MCA.

Step 2 — Clinical correlation: Hypertensive haemorrhage in the basal ganglia is caused by rupture of these lenticulostriate arteries.

Why other options are wrong:

- Option A: Posterior cerebral artery supplies the occipital lobe and thalamus; its perforators are the posterior choroidal and thalamoperforating arteries.
- Option B: Anterior cerebral artery supplies medial frontal/parietal cortex; Heubner's artery (recurrent artery of Heubner) is its main perforator.
- Option C: Correct.
- Option D: Basilar artery gives paramedian pontine perforators and the superior cerebellar, AICA, and PICA.

Final Answer: Lenticulostriate arteries arise from the middle cerebral artery (MCA) $\Rightarrow$ C

Answer: (C) Go Back to Q 4

Q5.

Solution

Concept — Posterior interventricular artery (PDA) and its territory: The posterior interventricular artery (posterior descending artery, PDA) runs in the posterior interventricular groove. In right-dominant circulation (70–80% of people), it is a branch of the right coronary artery (RCA). It supplies the posterior one-third of the interventricular septum and the posterior wall of the left ventricle.

Step 1 — Identify the artery: Posterior interventricular groove = posterior descending artery (PDA).

Step 2 — Territory of supply: Posterior 1/3 of the IVS and posterior LV wall; also the AV node (in most people).

Why other options are wrong:

- Option A: Anterior wall of the right ventricle is supplied by the conus artery (RCA) and marginal branches.
- Option B: Lateral LV wall is supplied by the obtuse marginal branches of the left circumflex artery.
- Option C: Anterior 2/3 of the IVS is supplied by the LAD (left anterior descending artery).
- Option D: Correct.

Final Answer: Posterior interventricular artery (PDA) $\rightarrow$ posterior 1/3 IVS and posterior LV wall $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 5

Q6.

Solution

Concept — Renal segmental arteries as end-arteries: The five renal segmental arteries (superior, anterosuperior, anteroinferior, inferior, and posterior) are true end-arteries with no significant anastomoses between them. Ligation of any segmental artery results in infarction of its renal segment.

Step 1 — Definition of end-artery: An end-artery has no anastomotic connections. Ischaemia beyond the point of occlusion causes infarction.

Step 2 — Clinical implication: Aberrant polar arteries (arising directly from the aorta) must be preserved during surgery; ligation causes polar infarction.

Why other options are wrong:

- Option A: Correct.
- Option B: They do *not* anastomose freely; infarction does follow ligation.
- Option C: They enter deeply into the renal parenchyma, not just the sinus.
- Option D: Segmental arteries arise from the renal artery (arterial system), not from the renal vein.

Final Answer: Renal segmental arteries are end-arteries; ligation causes segmental infarction $\Rightarrow$ A

Answer: (A) Go Back to Q 6

Q7.

Solution

Concept — Dermatomal levels of the trunk: Key trunk dermatomal landmarks are: T4 = nipple, T7 = xiphisternum, T10 = umbilicus, T12 = inguinal ligament. These are tested in clinical examinations of sensory levels after spinal cord injury.

Step 1 — Umbilical landmark: The umbilicus corresponds to the T10 dermatome.

Step 2 — Clinical relevance: A herpes zoster outbreak at T10 produces a band-like rash around the trunk at the level of the umbilicus.

Why other options are wrong:

- Option A: T6 corresponds to the level of the xiphoid process / upper abdomen.
- Option B: T8 supplies the epigastric area between T6 and T10.
- Option C: Correct — T10 = umbilicus.
- Option D: T12 corresponds to the inguinal region.

Final Answer: Dermatomal level of the umbilicus = T10 $\Rightarrow$ C

Answer: (C) Go Back to Q 7

Q8.

Solution

Concept — Compartments of the leg and their contents: The leg has four osseofascial compartments. The *anterior compartment* contains the tibialis anterior, extensor hallucis longus, extensor digitorum longus, the anterior tibial artery, and the deep peroneal nerve.

Step 1 — Identify affected structures: Deep peroneal nerve + anterior tibial artery = anterior compartment.

Step 2 — Clinical presentation: Anterior compartment syndrome causes pain on passive toe dorsiflexion, foot drop (deep peroneal nerve), and diminished dorsalis pedis pulse.

Why other options are wrong:

- Option A: Lateral compartment contains the peroneus longus and brevis, and the superficial peroneal nerve.
- Option B: Correct.
- Option C: Superficial posterior compartment contains gastrocnemius, soleus, plantaris, and the sural nerve.
- Option D: Deep posterior compartment contains the deep flexors (FHL, FDL, tibialis posterior), posterior tibial artery, and tibial nerve.

Final Answer: Deep peroneal nerve and anterior tibial artery are in the anterior compartment $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 8

Q9.

Solution

Concept — Lymphatic drainage of the breast: Approximately 75% of lymph from the breast drains to the axillary lymph nodes, primarily the anterior (pectoral) group. The internal mammary nodes receive about 20–25% of drainage, mainly from the medial quadrants.

Step 1 — Primary drainage: Lateral quadrant carcinomas drain predominantly to axillary nodes (anterior/pectoral group).

Step 2 — Sentinel node implication: The sentinel node is most commonly found in the axilla (anterior group) for lateral breast tumours.

Why other options are wrong:

- Option A: Internal mammary nodes receive mainly medial quadrant drainage (20–25
- Option B: Supraclavicular nodes are tertiary drainage and indicate advanced (N3) disease.
- Option C: Deltopectoral (infraclavicular) nodes receive some drainage but are not the primary group.
- Option D: Correct — anterior (pectoral) axillary nodes receive approximately 75% of breast lymph.

Final Answer: Primary lymphatic drainage of the breast $\rightarrow$ anterior (pectoral) axillary nodes $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 9

Q10.

Solution

Concept — Blood supply of the adult femoral head: In adults, the primary blood supply to the femoral head is via the retinacular vessels (superior and inferior retinacular arteries), which are branches of the medial circumflex femoral artery (MCFA). These ascend along the femoral neck within the capsule. An intracapsular fracture tears these vessels, causing avascular necrosis (AVN).

Step 1 — Primary adult supply: MCFA $\rightarrow$ retinacular branches $\rightarrow$ superior and inferior retinacular arteries $\rightarrow$ femoral head.

Step 2 — Why intracapsular fractures are dangerous: The retinacular vessels run inside the capsule; intracapsular fractures disrupt them, cutting off femoral head perfusion.

Why other options are wrong:

- Option A: Correct.
- Option B: The artery of the ligamentum teres (from obturator artery) contributes significantly only in children; in adults it is a minor or absent supply.
- Option C: Lateral circumflex femoral artery contributes to the femoral neck and trochanter, but not primarily the head.
- Option D: Inferior gluteal artery does not supply the femoral head.

Final Answer: Primary femoral head blood supply = retinacular branches of medial circumflex femoral artery $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 10

Q11.

Solution

Concept — Brown-Séquard syndrome: A hemisection of the spinal cord produces ipsilateral loss of fine touch, vibration, and proprioception (dorsal columns) and contralateral loss of pain and temperature (spinothalamic/anterolateral system), because spinothalamic fibres cross within 1–2 levels of entry while dorsal column fibres ascend ipsilaterally.

Step 1 — Analysing the deficit: Right-sided pain/temperature loss + left-sided proprioception/fine touch loss = right-side hemisection (injury on the right at T6).

Step 2 — Why this pattern: Dorsal columns are ipsilateral; spinothalamic fibres cross and ascend contralaterally.

Why other options are wrong:

- Option A: Central cord syndrome: bilateral upper extremity > lower extremity motor weakness; sacral sparing of sensation.
- Option B: Anterior cord syndrome: bilateral loss of motor function and pain/temperature with preserved dorsal column function.
- Option C: Correct.
- Option D: Posterior cord syndrome: loss of proprioception and vibration bilaterally; pain/temperature preserved.

Final Answer: Hemisection of the cord = Brown-Séquard syndrome $\Rightarrow$ C

Answer: (C) Go Back to Q 11

Q12.

Solution

Concept — Direct vs indirect inguinal hernia: Direct inguinal hernias protrude medial to the inferior epigastric vessels through Hesselbach's triangle (bounded by the inguinal ligament, inferior epigastric vessels, and lateral border of rectus abdominis). They pass through the superficial inguinal ring only (not the deep ring).

Step 1 — Key feature: Medial to inferior epigastric vessels + through the superficial ring only = direct inguinal hernia.

Step 2 — Hesselbach's triangle: Lateral boundary = inferior epigastric vessels; medial = rectus abdominis; inferior = inguinal ligament.

Why other options are wrong:

- Option A: Indirect hernia passes through the deep ring (lateral to inferior epigastric vessels) and follows the inguinal canal.
- Option B: Femoral hernia lies below the inguinal ligament in the femoral canal; no cough impulse through the superficial inguinal ring.
- Option C: Spigelian hernia is through the linea semilunaris at the level of the arcuate line.
- Option D: Correct.

Final Answer: Medial to inferior epigastric vessels, through superficial ring = direct inguinal hernia through Hesselbach's triangle $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q 12

Q13.

Solution

Concept — Jugular foramen contents: The jugular foramen transmits cranial nerves IX (glossopharyngeal), X (vagus), and XI (accessory), as well as the internal jugular vein and inferior petrosal sinus. A lesion here produces the jugular foramen syndrome (Vernet's syndrome).

Step 1 — Foramen identification: Jugular foramen = CN IX, X, XI.

Step 2 — Clinical signs: Dysphagia (CN IX, X), hoarseness (CN X), shoulder weakness (CN XI), loss of gag reflex.

Why other options are wrong:

- Option A: Correct — CN IX, X, XI traverse the jugular foramen.
- Option B: CN III, IV, VI pass through the superior orbital fissure (and cavernous sinus).
- Option C: V1 (ophthalmic) and V2 (maxillary) pass through the superior orbital fissure and foramen rotundum; CN VI through the superior orbital fissure/cavernous sinus.
- Option D: CN VII and CN VIII pass through the internal acoustic meatus.

Final Answer: Jugular foramen contains CN IX, X, XI $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q 13

Q14.

Solution

Concept — Midgut rotation during embryological development: During the 6th week, the midgut herniates into the umbilical cord due to inadequate abdominal space. As it returns (10th week), it undergoes a 270° counterclockwise rotation (as viewed from the anterior). This places the caecum in the right iliac fossa and the duodenojejunal flexure to the left of the midline.

Step 1 — The rotation: 270° counterclockwise rotation around the superior mesenteric artery (SMA) axis.

Step 2 — Clinical significance: Failure of full rotation (malrotation) predisposes to midgut volvulus around the SMA.

Why other options are wrong:

- Option A: 90° is the rotation that occurs during herniation (out), not the complete return rotation.
- Option B: Correct — 270° counterclockwise.
- Option C: 180° would result in malposition; the full rotation is 270°.
- Option D: 360° is not physiological for midgut rotation.

Final Answer: Midgut returns with 270° counterclockwise rotation $\Rightarrow$ B

Answer: (B) Go Back to Q 14

Q15.

Solution

Concept — Histology of the proximal convoluted tubule (PCT): The PCT has the most complex histology in the nephron: tall columnar cells, dense brush border (microvilli greatly increasing surface area), abundant mitochondria (for active Na^+/K^+ transport), and highly interdigitated basolateral membranes that reduce visible luminal space in cross-section.

Step 1 — Feature analysis: Brush border + abundant mitochondria + complex basolateral interdigitations = PCT.

Step 2 — Function correlate: PCT reabsorbs 65–70% of filtered Na^+, water, glucose, amino acids, and bicarbonate — consistent with this high metabolic activity.

Why other options are wrong:

- Option A: Collecting duct cells are cuboidal/columnar but lack a brush bor-

der; they have principal cells and intercalated cells.
- Option B: Descending thin limb has flat, simple squamous epithelium with few mitochondria.
- Option C: Correct.
- Option D: Glomerular parietal epithelium is simple squamous and lines Bowman's capsule.

Final Answer: Tall columnar cells with brush border and abundant mitochondria = proximal convoluted tubule $\Rightarrow$ C

Answer: (C) Go Back to Q 15

Q16.

Solution

Concept — Porto-systemic anastomoses and caput medusae: Caput medusae (dilated veins radiating from the umbilicus) results from reversal of flow through para-umbilical veins (in the falciform ligament) that connect the left portal vein (portal system) to the superficial and inferior epigastric veins (systemic). These become dilated and visible in portal hypertension.

Step 1 — Sites of porto-systemic anastomoses: (1) lower oesophagus, (2) lower rectum, (3) para-umbilical (caput medusae), (4) retroperitoneum (veins of Retzius), (5) bare area of liver.

Step 2 — Caput medusae specifically: Para-umbilical veins (portal) $\leftrightarrow$ superficial epigastric veins (systemic) at the umbilicus via the falciform ligament.

Why other options are wrong:

- Option A: Lower rectum anastomosis (superior vs inferior rectal veins) produces haemorrhoids, not caput medusae.
- Option B: Bare area anastomosis produces no classic visible surface sign.
- Option C: Retroperitoneal veins of Retzius do not produce a visible surface pattern.
- Option D: Correct — para-umbilical veins via the falciform ligament produce caput medusae.

Final Answer: Caput medusae = para-umbilical vein anastomosis at the falciform ligament $\Rightarrow$ D

Answer: (D) Go Back to Q 16

Q17.

Solution

Concept — Facial nerve branches and the stapedius reflex: The facial nerve (CN VII) innervates the stapedius muscle via a branch in the facial canal (nerve to stapedius). The stapedius dampens excessive movement of the ossicular chain in response to loud sounds. When the facial nerve is paralysed proximal to the nerve to stapedius, the stapedius is paralysed, and loud sounds cause disproportionately intense vibrations perceived as hyperacusis.

Step 1 — Localise the lesion: Complete facial nerve palsy with hyperacusis localises the lesion to the facial canal proximal to the stapedial branch (i.e., in or near the mastoid segment of the canal).

Step 2 — Stapedius function: Normally contracts in response to loud sounds via the stapedius reflex; loss causes hyperacusis.

Why other options are wrong:

- Option A: Greater petrosal nerve loss causes reduced lacrimation (dry eye), not hyperacusis.
- Option B: Correct.
- Option C: Chorda tympani loss causes loss of taste on anterior 2/3 of the tongue — this patient may also have this, but it does not cause hyperacusis.
- Option D: Auriculotemporal nerve is a branch of CN V3 (mandibular division), not CN VII.

Final Answer: Hyperacusis in facial nerve palsy = paralysis of stapedius muscle $\Rightarrow$ B

Answer: (B) Go Back to Q 17

Q18.

Solution

Concept — Action potential phases in neurons: Phase III (peak/overshoot) of the action potential is the point at which the membrane potential reaches its maximum positive value (+30 to +40 mV). This is driven by rapid influx of Na^+ through voltage-gated Na^+ channels that opened at threshold. At the peak, Na^+ channels are still open (though beginning to inactivate) and K^+ channels are beginning to open.

Step 1 — Phase identification: Phase III = the peak overshoot = maximum

depolarisation = maximum inward Na^+ current.

Step 2 — Ion channel state: Voltage-gated Na^+ channels are in the open (conducting) state; inactivation gates start to close at the peak.

Why other options are wrong:

- Option A: Correct.
- Option B: K^+ channel opening (repolarisation) begins after the peak = phase IV (falling phase).
- Option C: Na^+–K^+ ATPase is too slow to contribute to the acute phase; it restores gradients during the refractory period.
- Option D: Na^+ channel inactivation occurs at the peak and afterwards (absolute refractory period = during and after the peak), but the description of the peak itself is driven by open Na^+ channels.

Final Answer: Phase III (peak overshoot): voltage-gated Na^+ channels open, rapid Na^+ influx $\Rightarrow$ A

Answer: (A) Go Back to Q 18

Q19.

Solution

Concept — Resting membrane potential and K^+ conductance: The resting membrane potential (RMP) is maintained primarily by the high permeability of the membrane to K^+ through inward-rectifier K^+ channels. K^+ efflux drives the membrane potential toward the K^+ equilibrium potential (-90 mV). If this outward K^+ current is blocked, the membrane cannot maintain its negative potential and depolarises.

Step 1 — Normal RMP mechanism: K^+ leaks out through resting K^+ channels, making inside negative. Blocking these channels = loss of outward K^+ current = membrane becomes less negative (depolarises).

Step 2 — Drug effect: Selective block of resting K^+ channels $\rightarrow$ reduced outward K^+ current $\rightarrow$ depolarisation.

Why other options are wrong:

- Option A: Na^+ influx would cause depolarisation (correct direction), but blocking K^+ channels does not increase Na^+ influx as the primary mechanism.
- Option B: Correct.

- Option C: Na^+–K^+ ATPase cannot compensate immediately and does not set RMP acutely.
- Option D: Cl^- conductance is not the primary determinant of RMP in cardiac myocytes.

Final Answer: Blocking resting K^+ channels $\rightarrow$ RMP less negative (depolarisation) $\Rightarrow$ B

Answer: (B) Go Back to Q 19

Q20.

Solution

Concept — Isovolumetric contraction (IVC): IVC is the phase of the cardiac cycle that begins with mitral valve closure (first heart sound, S1) and ends with aortic valve opening. During IVC, both valves are closed, ventricular volume is constant, and left ventricular pressure rises rapidly from end-diastolic pressure ($\sim$8 mmHg) to aortic diastolic pressure ($\sim$80 mmHg).

Step 1 — Key features: Mitral valve closed + aortic valve closed + LV pressure rising = IVC.

Step 2 — Wiggers diagram correlation: IVC follows S1 and precedes the opening of the aortic valve (aortic ejection).

Why other options are wrong:

- Option A: Rapid ventricular filling: mitral valve open, aortic closed, pressure falling/low.
- Option B: Isovolumetric relaxation: both valves closed but pressure is falling (after aortic valve closes = S2).
- Option C: Correct.
- Option D: Rapid ejection: aortic valve open, mitral closed, pressure is rising and then falling.

Final Answer: Mitral closed + aortic closed + LV pressure rising = isovolumetric contraction $\Rightarrow$ C

Answer: (C) Go Back to Q 20

Q21.

Solution

Concept — Fick principle for cardiac output: Cardiac output (CO) = O_2 consumption / (arteriovenous O_2 content difference). CO = $\frac{250 \text{ mL/min}}{(200-150) \text{ mL/L}} = \frac{250}{50} = 5.0$ L/min.

Step 1 — Formula: CO = $\dot{V}O_2$ / (CaO_2 – CvO_2) where CaO_2 = pulmonary vein content (200 mL/L) and CvO_2 = pulmonary artery content (150 mL/L).

Step 2 — Calculation: $\frac{250}{200-150} = \frac{250}{50} = 5.0$ L/min.

Why other options are wrong:

- Option A: 3.5 L/min would require an A-V difference of ~71 mL/L.
- Option B: 4.0 L/min requires A-V difference of 62.5 mL/L.
- Option C: 4.5 L/min requires A-V difference of ~56 mL/L.
- Option D: Correct — CO = 250/50 = 5.0 L/min.

Final Answer: Fick CO = 250 / (200−150) = 5.0 L/min ⇒ D

Answer: (D) Go Back to Q 21

Q22.

Solution

Concept — Left ventricular pressure-volume loop: In the PV loop, the segment from point 2 (mitral valve closes = end-diastole) to point 3 (aortic valve opens) represents isovolumetric contraction. Volume is constant (isovolumetric), pressure rises steeply, and both valves are closed.

Step 1 — Identify points 2 and 3: Point 2 = end-diastole (mitral closes); point 3 = aortic valve opens. The segment between them is vertical (constant volume) on the PV loop.

Step 2 — Phase and valves: Isovolumetric contraction; both mitral and aortic valves are closed.

Why other options are wrong:

- Option A: Correct.
- Option B: Rapid ejection (point 3 to point 4): aortic valve open, volume decreasing.
- Option C: Isovolumetric relaxation (point 4 to point 1): both valves closed, pressure falling.

- Option D: Rapid ventricular filling (point 1 to point 2): mitral open, volume increasing.

Final Answer: Segment 2→3 = isovolumetric contraction; both valves closed ⇒ A

Answer: (A) Go Back to Q 22

Q23.

Solution

Concept — Spirometric pattern in obstructive lung disease: Obstructive disease (COPD, asthma) is characterised by airflow limitation: decreased FEV_1/FVC ratio. Air trapping leads to hyperinflation with increased RV and FRC. TLC is normal or increased (not decreased).

Step 1 — Obstructive pattern: FEV_1/FVC ratio <0.7; RV and FRC increased; TLC normal or increased.

Step 2 — Distinguishing from restrictive: Restrictive disease shows decreased TLC and FVC with normal or increased FEV_1/FVC ratio.

Why other options are wrong:

- Option A: Decreased TLC is the hallmark of restrictive disease.
- Option B: Decreased FEV_1/FVC with decreased TLC is a mixed picture; TLC should be normal or increased in pure obstructive disease.
- Option C: Correct — decreased FEV_1/FVC + increased RV and FRC (air trapping).
- Option D: Normal FEV_1/FVC with decreased FVC and TLC = restrictive pattern.

Final Answer: Obstructive pattern: decreased FEV_1/FVC ratio, increased RV and FRC (air trapping) ⇒ C

Answer: (C) Go Back to Q 23

Q24.

Solution

Concept — ACE inhibitors and GFR: Angiotensin II preferentially constricts the efferent arteriole more than the afferent. This efferent constriction maintains glomerular hydrostatic pressure and GFR even when renal perfusion pressure falls. ACE inhibitors block angiotensin II production, dilating the efferent arteriole, reducing glomerular hydrostatic pressure, and decreasing GFR.

Step 1 — ACE inhibitor mechanism: Block angiotensin II $\rightarrow$ efferent arteriole dilates $\rightarrow$ glomerular hydrostatic pressure falls $\rightarrow$ GFR falls $\rightarrow$ creatinine rises.

Step 2 — Clinical significance: A modest rise in creatinine (up to 30%) is expected and acceptable with ACE inhibitors; a rise $>$30% raises concern for renal artery stenosis.

Why other options are wrong:

- Option A: ACE inhibitors dilate (not constrict) afferent arterioles; afferent constriction would further reduce GFR.
- Option B: Correct.
- Option C: ACE inhibitors reduce aldosterone, causing natriuresis, not sodium retention.
- Option D: Angiotensin II does cause mesangial contraction, but the primary mechanism of GFR change with ACE inhibitors is via efferent arteriolar dilation.

Final Answer: ACE inhibitor $\rightarrow$ efferent dilation $\rightarrow$ reduced GFR $\rightarrow$ rising creatinine $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 24

Q25.

Solution

Concept — Transport maximum (T_m) and renal glucose handling: Glucose is freely filtered at the glomerulus and completely reabsorbed in the proximal tubule by SGLT2 (and SGLT1 in the late PCT) as long as the filtered load does not exceed the tubular transport maximum ($T_m \approx 375$ mg/min). The plasma glucose threshold at which glycosuria begins is approximately 180 mg/dL in most individuals.

Step 1 — T_m concept: Below 180 mg/dL plasma glucose, all filtered glucose is

within the capacity of SGLT2/SGLT1 to reabsorb → no urinary glucose.

Step 2 — SGLT2 mutation: With SGLT2 dysfunction (renal glycosuria), the effective T_m is drastically reduced; glucose appears in urine even at normal blood glucose levels.

Why other options are wrong:

- Option A: Glucose is not reabsorbed in the loop of Henle, and reabsorption is active (via SGLT), not passive.
- Option B: SGLT2 is a Na^+-linked co-transporter, not driven by simple concentration gradient alone.
- Option C: Correct.
- Option D: The GBM is freely permeable to glucose; filtration occurs at plasma glucose concentrations.

Final Answer: Glucose T_m not exceeded below ∼180 mg/dL → all filtered glucose reabsorbed ⇒ C

Answer: (C) Go Back to Q 25

Q26.

Solution

Concept — Conjugation of bilirubin and neonatal jaundice: Bilirubin is conjugated in hepatocytes by UDP-glucuronosyltransferase (UGT1A1), which adds glucuronic acid to unconjugated (indirect) bilirubin, making it water-soluble for biliary excretion. In neonates, UGT1A1 expression is transiently low, resulting in accumulation of unconjugated bilirubin and physiological jaundice in the first week of life.

Step 1 — Enzyme identification: UGT1A1 conjugates bilirubin using UDP-glucuronic acid.

Step 2 — Neonatal physiology: UGT1A1 matures to full activity by 2–4 weeks of age; neonatal physiological jaundice resolves as enzyme expression increases.

Why other options are wrong:

- Option A: Biliverdin reductase converts biliverdin → bilirubin (not conjugation); haem oxygenase converts haem → biliverdin.
- Option B: ALT is a transaminase involved in amino acid metabolism, not bilirubin conjugation.

- Option C: Alkaline phosphatase is a marker of cholestasis, not directly involved in bilirubin conjugation.
- Option D: Correct.

Final Answer: Bilirubin conjugation enzyme = UGT1A1; transiently low in neonates $\Rightarrow$ D

Answer: (D) Go Back to Q 26

Q27.

Solution

Concept — LH surge mechanism (positive feedback by oestrogen): During most of the follicular phase, oestradiol exerts negative feedback on GnRH/LH. However, when oestradiol from the dominant follicle rises above $\sim$200 pg/mL for $\geq$48 hours, the feedback switches to positive, amplifying GnRH pulse frequency and amplitude. This triggers the preovulatory LH surge, which induces ovulation within 36–40 hours.

Step 1 — Positive feedback switch: High sustained oestradiol $\rightarrow$ positive feedback on hypothalamus and pituitary $\rightarrow$ GnRH surge $\rightarrow$ LH surge.

Step 2 — Timing: LH surge on day 13; ovulation on day 14 (in a 28-day cycle).

Why other options are wrong:

- Option A: Correct.
- Option B: Progesterone fall triggers FSH/LH rise for the next cycle, not the midcycle LH surge.
- Option C: Inhibin B suppresses FSH (not LH) and does not directly trigger LH release.
- Option D: Prolactin suppresses GnRH and gonadotropin release; it does not stimulate ovulation.

Final Answer: LH surge triggered by positive feedback from rising oestradiol on the hypothalamo-pituitary axis $\Rightarrow$ A

Answer: (A) Go Back to Q 27

Q28.

Solution

Concept — Frank-Starling curve and positive inotropy: A shift of the Starling curve upward and to the left (curve X) means that for the same preload (EDV), the heart generates a greater stroke volume. This is the definition of increased myocardial contractility (positive inotropy). Dobutamine, a β_1-adrenergic agonist, is the classic positive inotrope.

Step 1 — Curve X characteristics: Higher stroke volume at any given EDV = increased contractility.

Step 2 — Dobutamine mechanism: Activates β_1 receptors $\rightarrow$ increased cAMP $\rightarrow$ increased Ca^{2+} release from SR $\rightarrow$ stronger contraction.

Why other options are wrong:

- Option A: Beta-blockers reduce contractility and shift the curve downward (toward curve Y).
- Option B: Correct.
- Option C: Haemorrhage reduces preload (moves along the same curve); it does not shift the curve.
- Option D: Cardiac tamponade limits filling (reduced preload/EDV); moves along the curve, does not shift it upward.

Final Answer: Upward shift of Starling curve = increased contractility; caused by dobutamine $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 28

Q29.

Solution

Concept — Hypothalamic responses to hyperthermia: When core temperature rises, the anterior hypothalamus (the body's thermostat for heat dissipation) activates heat-losing mechanisms: cutaneous vasodilation (redirects blood to the skin for radiation/convection), sweating (evaporative cooling), and reduced skeletal muscle tone (reduced heat production).

Step 1 — Heat dissipation mechanisms: Vasodilation + sweating + reduced muscle tone = all heat-losing effectors.

Step 2 — Distinguishing from heat conservation: Vasoconstriction, piloerection, and shivering are responses to *hypothermia*, not hyperthermia.

Why other options are wrong:

- Option A: Vasoconstriction + piloerection + shivering = hypothermia responses.
- Option B: Reduced metabolic rate alone and GI absorption are not primary thermoregulatory effectors.
- Option C: Correct.
- Option D: ADH and peripheral vasoconstriction are volume/cold responses, not heat dissipation.

Final Answer: Hyperthermia effectors: cutaneous vasodilation, sweating, reduced muscle tone $\Rightarrow$ C

Answer: (C) Go Back to Q 29

Q30.

Solution

Concept — ABO blood group system and universal donor: Group O red cells lack both A and B antigens on their surface. When transfused into a recipient with blood group A, B, or AB, the recipient's anti-A or anti-B antibodies cannot bind to the O red cells (no targets), preventing complement-mediated intravascular haemolysis. This is why group O is called the "universal donor" for red cells.

Step 1 — Group O red cell surface: No A or B antigens; H antigen only.

Step 2 — Why safe: Recipient antibodies (anti-A, anti-B) have no ABO antigens to bind on the transfused O cells $\rightarrow$ no haemolytic reaction from red cell antigens.

Why other options are wrong:

- Option A: H antigen is present on O cells; anti-H is a rare naturally occurring antibody (in Bombay phenotype), not in most recipients.
- Option B: Group O cells carry only H antigen, not A or B antigens.
- Option C: Group O plasma (which contains anti-A and anti-B) can cause reactions if given in large volumes; this refers to plasma, not the red cell safety.
- Option D: Correct.

Final Answer: Group O red cells lack A and B antigens $\rightarrow$ recipient antibodies cannot bind $\Rightarrow$ D

Answer: (D) Go Back to Q 30

Q31.

Solution

Concept — CSF production and circulation: Approximately 70–80% of CSF is produced by the choroid plexus (located in the lateral, third, and fourth ventricles). The total CSF volume is approximately 150 mL; it is produced at a rate of approximately 20 mL/hour, equalling approximately 500 mL/day. CSF is reabsorbed via arachnoid granulations into the dural venous sinuses.

Step 1 — Production site: Choroid plexus of the lateral (predominantly), third, and fourth ventricles.

Step 2 — Production rate: $\approx$20 mL/hour $\times$ 24 hours $\approx$ 500 mL/day; the total volume is only $\approx$150 mL, so it is fully turned over $\approx$3 times per day.

Why other options are wrong:

- Option A: Correct.
- Option B: Ependymal lining contributes some CSF, but the choroid plexus is the primary source; 100 mL/day underestimates.
- Option C: Arachnoid granulations are for reabsorption, not production.
- Option D: Dural venous sinuses drain CSF but do not produce it.

Final Answer: CSF produced by choroid plexus; $\approx$500 mL/day $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 31

Q32.

Solution

Concept — Right shift of the O_2-haemoglobin dissociation curve (Bohr effect): The curve shifts right (decreased O_2 affinity, facilitating O_2 unloading at tissues) with: increased pCO_2, acidosis (decreased pH), increased temperature, increased 2,3-DPG. This COPD patient has all of: elevated $PaCO_2$ (55 mmHg), acidosis (pH 7.32), and chronically elevated 2,3-DPG.

Step 1 — COPD blood gas analysis: $PaCO_2$ 55 mmHg (elevated), pH 7.32 (acidosis) = right-shifting factors.

Step 2 — 2,3-DPG: Chronic hypoxia stimulates increased red cell 2,3-DPG production, further right-shifting the curve.

Why other options are wrong:

- Option A: Decreased CO_2 + alkalosis + decreased temperature all cause a

left shift.

- Option B: Correct.
- Option C: All three factors (CO_2, pH, 2,3-DPG) are relevant to the O_2-Hb dissociation curve.
- Option D: Chronic hypoxia increases 2,3-DPG (causing right shift), not decreases it.

Final Answer: Elevated CO_2, acidosis, and increased 2,3-DPG cause right shift of Hb-O_2 dissociation curve $\Rightarrow$ B

Answer: (B) Go Back to Q 32

Q33.

Solution

Concept — Water deprivation test and desmopressin response: In central (cranial) diabetes insipidus (DI), the posterior pituitary fails to secrete adequate ADH. The kidney's collecting ducts are normal (intact V2 receptors). Therefore, exogenous ADH (desmopressin) restores urine concentration. In nephrogenic DI, the kidney does not respond to ADH, so desmopressin fails to concentrate urine. In primary polydipsia, dehydration alone can concentrate urine moderately.

Step 1 — Dehydration test result: Urine osmolality 150 mOsm/kg after 8 hours = failure to concentrate = DI (central or nephrogenic).

Step 2 — Desmopressin response: Urine rises to 650 mOsm/kg after desmopressin = the collecting duct responds normally = central DI (lack of endogenous ADH).

Why other options are wrong:

- Option A: Primary polydipsia: after dehydration, urine can concentrate (osmolality >750–800 mOsm/kg); desmopressin causes only a small further rise.
- Option B: Nephrogenic DI: urine remains dilute even after desmopressin (no response to ADH).
- Option C: Correct — central DI: urine concentrates after desmopressin.
- Option D: Type 2 diabetes causes osmotic diuresis; urine glucose would be present and osmolality elevated due to glucose, not free water loss.

Final Answer: Dilute urine after dehydration + concentration after desmopressin = central DI $\Rightarrow$ C

Answer: (C) Go Back to Q 33

Q34.

Solution

Concept — Sinoatrial node automaticity and the funny current (I_f): Unlike ventricular myocytes, SA node cells lack fast Na^+ channels and do not have a stable resting potential. Phase 4 (spontaneous depolarisation) is driven by: (1) the "funny current" I_f — a mixed inward Na^+/K^+ current activated on hyperpolarisation; (2) declining outward K^+ current (I_K decay); and (3) a small T-type Ca^{2+} inward current. Together these drive the membrane from -60 mV to the threshold (-40 mV).

Step 1 — I_f (funny current): Activated when the membrane hyperpolarises below -50 mV; non-selective Na^+/K^+ inward current; blocked by ivabradine.

Step 2 — Contributing currents: Declining I_K + rising I_f + T-type Ca^{2+} = pacemaker depolarisation.

Why other options are wrong:

- Option A: Fast Na^+ channels (I_{Na}) are absent in SA node cells; this is the ventricular mechanism.
- Option B: Outward K^+ current opposes depolarisation; its decay contributes but alone cannot cause spontaneous depolarisation.
- Option C: T-type Ca^{2+} channels contribute but I_f is the primary pacemaker current.
- Option D: Correct.

Final Answer: SA node automaticity driven by I_f (funny current) + declining I_K + T-type Ca^{2+} $\Rightarrow$ D

Answer: (D) Go Back to Q 34

Q35.

Solution

Concept — Competitive inhibition is reversible and overcome by excess substrate: A competitive inhibitor competes with substrate for the active site. At high substrate concentrations, the probability of substrate binding exceeds that of the inhibitor, effectively overcoming inhibition. Therefore, the V_{max} is the same as the uninhibited enzyme, but the apparent K_m is increased (more substrate needed to reach half-maximal velocity).

Step 1 — Competitive inhibition kinetics: Same V_{max}; increased apparent K_m

(K'_m).

Step 2 — Effect of excess substrate: High [S] $\gg K_i$ means substrate displaces inhibitor; velocity approaches V_{max}.

Why other options are wrong:

- Option A: Correct.
- Option B: Permanently reduced V_{max} regardless of [S] describes non-competitive inhibition.
- Option C: In competitive inhibition, V_{max} is unchanged and K_m is increased (not reduced).
- Option D: Irreversible inactivation is a feature of mechanism-based (suicide) inhibitors, not typical competitive inhibitors.

Final Answer: Competitive inhibition: reversible; excess substrate restores V_{max} $\Rightarrow$ A

Answer: (A) Go Back to Q 35

Q36.

Solution

Concept — ATP yield from aerobic glucose oxidation using modern P/O ratios: Glycolysis: 2 ATP (net) + 2 NADH (cytoplasmic). Pyruvate dehydrogenase ($\times 2$): 2 NADH. TCA cycle (per glucose): 6 NADH + 2 $FADH_2$ + 2 GTP. Total NADH = 10; Total $FADH_2$ = 2. Using P/O: NADH $\times$ 2.5 = 25 ATP; $FADH_2$ $\times$ 1.5 = 3 ATP. Substrate-level: 2 ATP (glycolysis) + 2 GTP (TCA) = 4. Total $\approx$ 25 + 3 + 4 = 32 ATP (range 30–32 depending on cytoplasmic NADH shuttle efficiency).

Step 1 — Count reducing equivalents: 10 NADH $\times$ 2.5 = 25; 2 $FADH_2$ $\times$ 1.5 = 3.

Step 2 — Add substrate-level phosphorylation: 2 (glycolysis) + 2 (GTP from TCA) = 4. Total = 32.

Why other options are wrong:

- Option A: 36–38 ATP uses outdated P/O ratios of 3 for NADH and 2 for $FADH_2$.
- Option B: Correct — 30–32 ATP with modern P/O ratios.
- Option C: 18 ATP (substrate-level phosphorylation only) is drastically underestimated.
- Option D: 40 ATP is not achievable under standard biochemical accounting.

Final Answer: Aerobic glucose oxidation yields ≈30–32 ATP using modern P/O ratios ⇒ B

Answer: (B) Go Back to Q 36

Q37.

Solution

Concept — Thiamine pyrophosphate (TPP)-dependent enzymes: TPP is an essential cofactor for: (1) pyruvate dehydrogenase (PDH), (2) α-ketoglutarate dehydrogenase (part of the TCA cycle), (3) transketolase (pentose phosphate pathway), and (4) branched-chain α-keto acid dehydrogenase. Pyruvate carboxylase uses biotin (vitamin B_7) as its cofactor, not TPP.

Step 1 — TPP-requiring enzymes: PDH, α-KG dehydrogenase, transketolase, BCKDH.

Step 2 — Pyruvate carboxylase cofactor: Biotin (not TPP); it converts pyruvate to oxaloacetate in gluconeogenesis.

Why other options are wrong:

- Option A: PDH requires TPP — this is a TPP-dependent enzyme.
- Option B: α-Ketoglutarate dehydrogenase requires TPP — TPP-dependent.
- Option C: Correct — pyruvate carboxylase requires biotin, NOT TPP.
- Option D: Transketolase requires TPP — classic assay for thiamine deficiency.

Final Answer: Pyruvate carboxylase uses biotin (not TPP) ⇒ C

Answer: (C) Go Back to Q 37

Q38.

Solution

Concept — Orotic aciduria in urea cycle defects: In urea cycle enzyme deficiencies (except arginase deficiency), carbamoyl phosphate accumulates in the mitochondria. It exits to the cytosol and enters the pyrimidine synthesis pathway, where it is converted to orotic acid. Since orotic acid is not further metabolised (UMP synthase may be overwhelmed), it accumulates and is excreted in the urine. This helps distinguish urea cycle defects (high orotic acid) from organic acidaemias.

Step 1 — Carbamoyl phosphate accumulation: When argininosuccinate syn-

thetase is deficient, citrulline cannot be converted to argininosuccinate; carbamoyl phosphate backs up.

Step 2 — Cytosolic diversion: Excess carbamoyl phosphate crosses to the cytosol → combines with aspartate via CAD complex → orotic acid.

Why other options are wrong:

- Option A: Reduced arginine/ornithine synthesis does decrease feedback on CPS-I (correct mechanism), but the primary driver of orotic aciduria is carbamoyl phosphate overflow, not altered OTC feedback alone.
- Option B: Arginine catabolism does not produce orotic acid.
- Option C: Orotic acid is an intermediate of pyrimidine synthesis, not the urea cycle.
- Option D: Correct — carbamoyl phosphate spillover into cytosol → pyrimidine pathway → orotic acid accumulation.

Final Answer: Carbamoyl phosphate overflows into cytosol → pyrimidine synthesis → orotic aciduria ⇒ D

Answer: (D) Go Back to Q 38

Q39.

Solution

Concept — Fatty acid synthesis vs beta-oxidation: compartment and cofactors: Fatty acid synthesis occurs in the cytoplasm (cytosol) and uses NADPH (from the pentose phosphate pathway and malic enzyme) as the reducing agent. Beta-oxidation occurs in the mitochondrial matrix and generates NADH and $FADH_2$, which feed into the electron transport chain.

Step 1 — Compartmentalisation: Synthesis = cytoplasm (via fatty acid synthase complex, FAS). Beta-oxidation = mitochondrial matrix.

Step 2 — Cofactors: Synthesis: NADPH consumed. Beta-oxidation: NADH and $FADH_2$ produced.

Why other options are wrong:

- Option A: Correct.
- Option B: They occur in different compartments and use different carriers (malonyl-CoA vs acyl-carnitine).
- Option C: This reverses the cofactors; synthesis uses NADPH; oxidation generates NADH and $FADH_2$.

- Option D: Beta-oxidation generates acetyl-CoA, some of which is used for ketogenesis/TCA, not directly for cholesterol biosynthesis rate-limiting step (which is HMG-CoA reductase).

Final Answer: Fatty acid synthesis: cytoplasm, NADPH. Beta-oxidation: mitochondrial matrix, NADH + $FADH_2 \Rightarrow$ A

Answer: (A) Go Back to Q 39

Q40.

Solution

Concept — Lineweaver-Burk plot and non-competitive inhibition: On the Lineweaver-Burk (double-reciprocal) plot, non-competitive inhibition shows: (1) the y-intercept ($1/V_{max}$) increases (reduced V_{max}), and (2) the x-intercept ($-1/K_m$) is unchanged (same K_m), because the inhibitor binds an allosteric site and does not compete with substrate for the active site.

Step 1 — Non-competitive inhibition features: V_{max} decreases (y-intercept increases); K_m unchanged (x-intercept unchanged). The lines cross on the x-axis.

Step 2 — Contrast with competitive: Competitive inhibition: y-intercept same (same V_{max}); x-intercept moves right (apparent K_m increased).

Why other options are wrong:

- Option A: Rightward shift of x-intercept (more positive, less negative) with unchanged y-intercept = competitive inhibition.
- Option B: Both intercepts shifting describes mixed inhibition.
- Option C: Correct — y-intercept increases (lower V_{max}), x-intercept unchanged (same K_m).
- Option D: Lines converging at y-axis would mean same V_{max} = competitive inhibition pattern.

Final Answer: Non-competitive inhibition: y-intercept increases, x-intercept unchanged (reduced V_{max}, same K_m) $\Rightarrow$ C

Answer: (C) Go Back to Q 40

Q41.

Solution

Concept — Purine salvage pathway and HGPRT: HGPRT (hypoxanthine-guanine phosphoribosyltransferase) catalyses the salvage of hypoxanthine and guanine: Hypoxanthine + PRPP $\rightarrow$ IMP + PP_i; Guanine + PRPP $\rightarrow$ GMP + PP_i. This recycles purine bases, conserving energy. HGPRT deficiency (Lesch-Nyhan syndrome) forces purines down the de novo degradation pathway $\rightarrow$ xanthine $\rightarrow$ uric acid, causing hyperuricaemia, gout, neurological problems, and self-mutilation.

Step 1 — HGPRT function: Salvages hypoxanthine and guanine using PRPP as the ribose-5-phosphate donor.

Step 2 — Clinical consequence: Without salvage, all degraded purines become uric acid; allopurinol reduces uric acid but does not restore HGPRT function.

Why other options are wrong:

- Option A: De novo purine synthesis starts with PRPP + glutamine (via amidophosphoribosyltransferase); HGPRT is not involved.
- Option B: Correct.
- Option C: De novo pyrimidine synthesis involves carbamoyl phosphate; HGPRT is specific to purines.
- Option D: Thymidylate synthesis uses methyleneTHF and thymidylate synthase; not related to HGPRT.

Final Answer: HGPRT is essential for the purine salvage pathway $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 41

Q42.

Solution

Concept — Niacin (Vitamin B_3) and its coenzyme derivatives: Niacin (nicotinic acid/nicotinamide) is the precursor of NAD^+ (nicotinamide adenine dinucleotide) and $NADP^+$ (nicotinamide adenine dinucleotide phosphate). These coenzymes serve as electron carriers in hundreds of oxidation-reduction reactions including glycolysis, TCA cycle, beta-oxidation, and the pentose phosphate pathway.

Step 1 — Biochemical role of niacin: Niacin $\rightarrow$ $NAD^+/NADP^+$; essential for H^- (hydride) transfer in catabolic and anabolic reactions.

Step 2 — Pellagra deficit: Without NAD^+, energy metabolism, DNA repair (PARP

uses NAD^+), and biosynthesis fail.

Why other options are wrong:

- Option A: Correct — niacin $\rightarrow$ $NAD^+/NADP^+$.
- Option B: Coenzyme A is derived from pantothenic acid (vitamin B_5), not niacin.
- Option C: FAD and FMN are derived from riboflavin (vitamin B_2).
- Option D: TPP is derived from thiamine (vitamin B_1).

Final Answer: Niacin is the precursor of NAD^+ and $NADP^+$ $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q 42

Q43.

Solution

Concept — DNA mismatch repair (MMR) and Lynch syndrome: The MMR system recognises base-base mismatches and small insertion-deletion loops (IDLs) that escape proofreading by DNA polymerase during replication. In humans, MMR proteins (MSH2, MSH6 recognise mismatches; MLH1, PMS2 coordinate repair) use PCNA and strand discrimination signals to identify the newly synthesised (error-containing) strand. Germline mutations in MLH1, MSH2, MSH6, PMS2 cause Lynch syndrome (hereditary non-polyposis colorectal cancer, HNPCC).

Step 1 — MMR function: Recognises post-replicative mismatches and IDLs; removes the erroneously synthesised strand; resynthesises correctly.

Step 2 — Strand discrimination in humans: PCNA marks the newly synthesised strand; MMR excises mismatches from this strand.

Why other options are wrong:

- Option A: UV-induced thymine dimers are corrected by nucleotide excision repair (NER), not MMR.
- Option B: Double-strand breaks are repaired by homologous recombination (BRCA1/2) or NHEJ, not MMR.
- Option C: Correct.
- Option D: MMR operates in nuclear DNA; mitochondrial DNA uses a limited base excision repair; the statement is false.

Final Answer: MMR recognises post-replicative mismatches and IDLs using PCNA-based strand discrimination $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q 43

Q44.

Solution

Concept — Oncogenes vs tumour suppressor genes: Oncogenes are mutated/amplified versions of proto-oncogenes that promote cell growth; they are gain-of-function and act dominantly (one mutant allele sufficient). Tumour suppressor genes (e.g., BRCA1, TP53, RB1) inhibit cell growth; they are loss-of-function and follow the two-hit Knudson hypothesis — both alleles must be inactivated.

Step 1 — Oncogene characteristics: Gain-of-function; dominant; one allele sufficient (e.g., RAS, MYC, HER2).

Step 2 — TSG characteristics: Loss-of-function; recessive; both alleles needed (e.g., BRCA1, TP53, RB1).

Why other options are wrong:

- Option A: Oncogenes are gain-of-function and dominant; they do not require both alleles to be mutated.
- Option B: Correct.
- Option C: Tumour suppressor genes inhibit cell growth; their overexpression would suppress tumours, not cause cancer.
- Option D: Many oncogenic mutations are somatic (not inherited); germline inheritance occurs but is not universal.

Final Answer: Oncogenes: gain-of-function, dominant (one allele); TSGs: loss-of-function, both alleles needed $\Rightarrow$ B

Answer: (B) Go Back to Q 44

Q45.

Solution

Concept — Functions of albumin: Albumin (produced by the liver) serves as: (1) the main determinant of plasma colloid osmotic pressure (COP); (2) transporter of unconjugated bilirubin, free fatty acids, drugs (warfarin, aspirin), hormones (thyroid hormones), and ions (Ca^{2+}, Mg^{2+}); (3) buffer for acid-base balance. Acute-phase proteins (CRP, fibrinogen, haptoglobin, ferritin, complement) are produced by the liver in response to inflammatory cytokines (IL-1, IL-6, TNF-α), but albumin

is a *negative* acute-phase reactant (its levels fall during inflammation). CRP and fibrinogen are entirely separate proteins.

Step 1 — Albumin's actual functions: Transport (bilirubin, FFAs, drugs); oncotic pressure; buffer.

Step 2 — What albumin does NOT do: It does not synthesise other acute-phase proteins; CRP is an independent protein.

Why other options are wrong:

- Option A: Albumin does bind and transport unconjugated bilirubin in the bloodstream.
- Option B: Albumin transports free fatty acids in the bloodstream.
- Option C: Albumin is the primary determinant of plasma COP.
- Option D: Correct — albumin does NOT synthesise CRP or fibrinogen; these are separate hepatic proteins.

Final Answer: Albumin does not synthesise acute-phase proteins; CRP and fibrinogen are separate liver products $\Rightarrow$ D

Answer: (D) Go Back to Q 45

Q46.

Solution

Concept — PFK-1 as the key regulatory enzyme of glycolysis: In the glycolytic pathway, E1 (phosphoglucose isomerase) converts G6P to F6P. E2 (phosphofructokinase-1, PFK-1) phosphorylates F6P to F1,6-BP consuming 1 ATP — this is the committed, rate-limiting step. E3 (aldolase) cleaves F1,6-BP into DHAP and G3P. PFK-1 is allosterically inhibited by ATP and citrate (high energy signal) and activated by AMP and fructose-2,6-bisphosphate (low energy / fed state signal).

Step 1 — Identify E2: F6P $\rightarrow$ F1,6-BP + 1 ATP consumed = PFK-1.

Step 2 — Regulatory profile: PFK-1 inhibited by ATP and citrate; activated by AMP and F-2,6-BP. This is the key energy-sensing regulatory step of glycolysis.

Why other options are wrong:

- Option A: Correct — E2 is PFK-1.
- Option B: Hexokinase phosphorylates glucose to G6P (step 1, E0); it is inhibited by G6P (product inhibition), not by ATP/citrate.

- Option C: Phosphoglucose isomerase (E1 in this diagram) converts G6P to F6P; it is not a regulatory enzyme.
- Option D: Pyruvate kinase acts at the last step of glycolysis (PEP → pyruvate); it does not consume ATP or convert F6P.

Final Answer: E2 = PFK-1; the committed rate-limiting step of glycolysis ⇒ A

Answer: (A) Go Back to Q 46

Q47.

Solution

Concept — Ketogenesis: rate-limiting enzyme is HMG-CoA synthase (mitochondrial): In the liver, when glucose availability is low and acetyl-CoA accumulates (from beta-oxidation), ketogenesis proceeds: 2 acetyl-CoA → acetoacetyl-CoA → HMG-CoA (by mitochondrial HMG-CoA synthase, the rate-limiting step) → acetoacetate + acetyl-CoA. The rate-limiting enzyme of ketone body synthesis is the mitochondrial HMG-CoA synthase (distinct from the cytosolic HMG-CoA synthase of cholesterol synthesis).

Step 1 — Ketogenesis pathway: acetyl-CoA → acetoacetyl-CoA $\xrightarrow{\text{mito HMG-CoA synthase}}$ HMG-CoA $\xrightarrow{\text{HMG-CoA lyase}}$ acetoacetate.

Step 2 — Rate-limiting step: Mitochondrial HMG-CoA synthase is the committed, regulated step.

Why other options are wrong:

- Option A: Acetyl-CoA carboxylase is the rate-limiting enzyme of fatty acid synthesis (cytoplasm), not ketogenesis.
- Option B: Acyl-CoA dehydrogenase is the first step of beta-oxidation, not ketogenesis.
- Option C: Correct — mitochondrial HMG-CoA synthase is the rate-limiting enzyme of ketogenesis.
- Option D: HMG-CoA lyase cleaves HMG-CoA to acetoacetate + acetyl-CoA; it is not the rate-limiting step.

Final Answer: Rate-limiting enzyme of ketogenesis = mitochondrial HMG-CoA synthase ⇒ C

Answer: (C) Go Back to Q 47

Q48.

Solution

Concept — Vitamin D hydroxylation sequence: Vitamin D (cholecalciferol from skin/diet) undergoes: (1) 25-hydroxylation in the liver by 25-hydroxylase (CYP2R1/CYP27A1) to form 25-hydroxyvitamin D (calcidiol, the storage form measured in serum); (2) 1α-hydroxylation in the kidney (proximal tubule) by 1α-hydroxylase (CYP27B1) to form 1,25-dihydroxyvitamin D (calcitriol, the active form). PTH stimulates renal 1α-hydroxylase; FGF-23 and calcitriol itself inhibit it.

Step 1 — First hydroxylation: 25-OH in the liver (measured as 25-OH vitamin D in clinical assays).

Step 2 — Second hydroxylation: 1α-OH in the kidney; stimulated by PTH; produces active calcitriol.

Why other options are wrong:

- Option A: The order is reversed; 25-OH occurs first in the liver, not 1α-OH in the kidney.
- Option B: Correct.
- Option C: Both hydroxylations do not occur in the kidney; calcitonin does not regulate 1α-hydroxylase.
- Option D: 25-hydroxylation occurs in the liver, not the skin; the skin only produces cholecalciferol from 7-dehydrocholesterol upon UV-B exposure.

Final Answer: 25-OH in liver $\rightarrow$ 1α-OH in kidney (by PTH-stimulated 1α-hydroxylase) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 48

Q49.

Solution

Concept — Complex I of the electron transport chain (NADH dehydrogenase): Complex I accepts electrons from NADH, oxidising it to NAD^+, and transfers the electrons to ubiquinone (coenzyme Q, CoQ), reducing it to ubiquinol. This reaction is coupled to the pumping of 4 H^+ from the mitochondrial matrix to the intermembrane space per NADH, contributing to the proton gradient used by ATP synthase.

Step 1 — Complex I reaction: $NADH + H^+ + CoQ \rightarrow NAD^+ + CoQH_2$; simultaneously pumps 4 H^+ outward.

Step 2 — LHON relevance: Mutations in mtDNA-encoded Complex I subunits impair the proton pump, reducing ATP synthesis particularly in high-energy cells (retinal ganglion cells).

Why other options are wrong:

- Option A: Electrons from $FADH_2$ to CoQ + proton pumping = Complex II (succinate dehydrogenase) does NOT pump H^+ (pumps 0 H^+), or this describes Complex I incorrectly. Complex II transfers $FADH_2$ electrons to CoQ without pumping protons.
- Option B: Electrons from ubiquinol to cytochrome c + 4 H^+ pumped = Complex III (cytochrome bc1).
- Option C: Reduction of O_2 to H_2O using electrons from cytochrome c + proton pumping = Complex IV (cytochrome c oxidase).
- Option D: Correct.

Final Answer: Complex I: NADH $\rightarrow$ CoQ electron transfer + 4 H^+ pumped per NADH $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q 49

Q50.

Solution

Concept — C-reactive protein (CRP) in innate immunity: CRP is a pentraxin acute-phase protein produced by the liver in response to IL-6 (released during inflammation/infection). It binds phosphocholine moieties on the surface of apoptotic cells, necrotic cells, and microorganisms. CRP-bound targets then activate the classical complement pathway (via C1q binding) and serve as opsonins, enhancing phagocytosis by macrophages and neutrophils.

Step 1 — CRP molecular function: Binds phosphocholine $\rightarrow$ activates classical complement (C1q) $\rightarrow$ C3b deposition $\rightarrow$ opsonisation.

Step 2 — Acute-phase response role: CRP rises within 6–12 hours of an inflammatory stimulus (faster than ESR); used clinically to monitor inflammation/infection.

Why other options are wrong:

- Option A: CRP does not competitively bind TLR-4 to neutralise endotoxin; LPS-binding protein (LBP) facilitates TLR-4 activation, not CRP.
- Option B: CRP is part of innate immunity; it does not present antigens to T

cells (which is MHC-mediated adaptive immunity).
- Option C: Correct.
- Option D: CRP does not suppress IL-6 secretion; it is a downstream product of IL-6 signalling.

Final Answer: CRP binds phosphocholine on microorganisms/apoptotic cells → activates classical complement and opsonisation ⇒ C

Answer: (C) Go Back to Q 50

Q51.

Solution

Concept — Types of necrosis: Coagulative necrosis is the hallmark of ischemic infarction in solid organs (except brain). The cell architecture (outlines) is preserved but organelles and nuclei undergo denaturation. This occurs because ischemia denatures structural and enzymatic proteins simultaneously.

Step 1 — Identifying the pattern: Preserved cellular outlines with loss of nuclei (karyolysis/karyorrhexis) and eosinophilic cytoplasm in a 18-hour-old myocardial infarct = coagulative necrosis.

Step 2 — Linking to pathophysiology: The zone X (necrotic core) in the diagram shows the area of ischemic cell death — coagulative necrosis is universal here.

Why other options are wrong:

- Option A: Correct — coagulative necrosis is the right answer.
- Option B: Liquefactive necrosis occurs in brain infarcts and bacterial abscesses (enzymatic digestion dissolves cellular architecture).
- Option C: Caseous necrosis is the hallmark of TB — amorphous cheese-like material, no preserved outlines.
- Option D: Fat necrosis occurs in the pancreas/breast — saponification of fat by lipases, calcium soap deposits.

Final Answer: Preserved cellular outlines with loss of nuclei after myocardial ischemia = coagulative necrosis ⇒ A

Answer: (A) Go Back to Q 51

Q52.

Solution

Concept — Apoptosis vs. Necrosis: Apoptosis is programmed, energy (ATP)-dependent cell death mediated by caspases. It is characterized by cell shrinkage, chromatin condensation, membrane blebbing, and apoptotic body formation — WITHOUT plasma membrane disruption or inflammation.

Step 1 — Identifying apoptosis: Shrunken eosinophilic hepatocytes (Councilman bodies) with pyknotic nuclei, clear halo, no inflammation = apoptosis (not necrosis).

Step 2 — Key distinguishing feature: Apoptosis is ATP-dependent and requires caspase activation (intrinsic or extrinsic pathway). Necrosis is passive, ATP-independent, with membrane rupture and inflammation.

Why other options are wrong:

- Option A: Loss of plasma membrane integrity is a feature of NECROSIS, not apoptosis.
- Option B: Release of lysosomal enzymes causing autolysis is a feature of necrosis.
- Option C: Correct — ATP-dependent caspase activation is the hallmark of apoptosis.
- Option D: Neutrophilic infiltration is seen in necrosis; apoptosis does NOT trigger inflammation.

Final Answer: Apoptosis is distinguished by ATP-dependent caspase activation without inflammation $\Rightarrow$ C

Answer: (C) Go Back to Q 52

Q53.

Solution

Concept — Granuloma formation in TB: A granuloma is an organized collection of activated macrophages (epithelioid histiocytes) surrounded by lymphocytes, with or without Langhans giant cells and central caseous necrosis. In TB, it is driven by Th1-mediated delayed hypersensitivity.

Step 1 — Cell origin: All macrophage-derived cells in granulomas (epithelioid macrophages AND Langhans giant cells) originate from circulating monocytes that differentiate under IFN-γ stimulation.

Step 2 — Hallmark cell: The HALLMARK of granulomatous inflammation is the epithelioid macrophage — activated macrophage with abundant pale cytoplasm and vesicular nucleus, named for its resemblance to epithelial cells.

Why other options are wrong:

- Option A: Langhans giant cells are also monocyte-derived but are formed by fusion of epithelioid macrophages — they are a component, not the hallmark.
- Option B: Correct — epithelioid macrophage is the hallmark, derived from circulating monocytes.
- Option C: CD8+ cytotoxic T cells are present but are NOT the hallmark or the derived-from-monocyte cell.
- Option D: Plasma cells are B-cell derivatives (antibody-secreting), not the hallmark of granulomatous inflammation.

Final Answer: Hallmark of granulomatous inflammation derived from monocytes = epithelioid macrophage $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 53

Q54.

Solution

Concept — Wound healing phases: After the proliferative phase (granulation tissue, type III collagen), the remodeling phase begins (day 21 onward) and lasts up to 1 year. Key events: MMP-mediated degradation of type III collagen and replacement with type I collagen (stronger, thicker fibers), increasing tensile strength to approximately 70–80% of normal skin.

Step 1 — Collagen transition: Granulation tissue = type III collagen (provisional). Remodeling replaces type III with type I collagen via MMP (collagenase) activity — scar becomes less cellular and more fibrous.

Step 2 — Tensile strength: Maximum tensile strength (70–80% of original) is achieved by 3 months. It never fully returns to 100%.

Why other options are wrong:

- Option A: WRONG direction — type III (early) is replaced by type I (late), not the other way around.
- Option B: Tensile strength reaches 70–80% (not capped at 50%).
- Option C: MMPs are ACTIVE during remodeling; their activity is regulated

by TIMPs (tissue inhibitors).
- Option D: Correct — type III replaced by type I, progressive increase in tensile strength.

Final Answer: Remodeling = type III → type I collagen, increasing tensile strength ⇒ D

Answer: (D) Go Back to Q 54

Q55.

Solution

Concept — Mediators of vascular permeability: Vascular permeability in acute inflammation occurs in two phases: (1) Immediate phase (0–30 min): histamine and serotonin from mast cells/platelets causing endothelial cell contraction. (2) Delayed phase: bradykinin, leukotrienes, and cytokines.

Step 1 — Immediate mediator: Within the FIRST 30 minutes, histamine (released from mast cell degranulation triggered by IgE, complement C3a/C5a, or direct injury) is the PRIMARY mediator of vascular permeability (venule endothelial contraction).

Step 2 — Ruling out later mediators: LTB4 is a late-phase chemotactic mediator (hours). IL-1 is a cytokine acting over hours. C3a alone without histamine is insufficient for immediate response.

Why other options are wrong:

- Option A: Correct — histamine from mast cells is the earliest and most important mediator within 30 minutes.
- Option B: LTB4 is primarily a neutrophil chemotactic agent; it acts later (hours) and causes permeability as a delayed mediator.
- Option C: IL-1 is a systemic cytokine mediating fever and late vascular changes; onset is hours to days.
- Option D: C3a (anaphylatoxin) does trigger mast cell degranulation but acts via histamine release — alone it does not directly cause immediate permeability without histamine.

Final Answer: Histamine from mast cells is the primary mediator of increased permeability in first 30 min ⇒ A

Answer: (A) Go Back to Q 55

Q56.

Solution

Concept — TNM staging of breast cancer: Stage III is locally advanced disease. Stage IIIB includes T4 (chest wall/skin invasion) with any N status up to N2, M0. Stage IV requires M1 (distant metastasis). Stage I/II are early stages with small tumors and limited nodal involvement.

Step 1 — Applying TNM: T4 = tumor invades chest wall or skin (locally advanced). N2 = 4–9 ipsilateral axillary nodes (or internal mammary nodes). M0 = no distant metastasis.

Step 2 — Stage determination: T4, N2, M0 = Stage IIIB (locally advanced breast cancer, not metastatic).

Why other options are wrong:

- Option A: Stage I = T1, N0, M0 — very early, small tumor, no nodes.
- Option B: Stage II = T1/T2 with limited nodal involvement; chest wall invasion (T4) is NOT Stage II.
- Option C: Correct — T4, N2, M0 = Stage IIIB.
- Option D: Stage IV requires M1 (distant metastases); M0 here rules out Stage IV.

Final Answer: T4, N2, M0 = Stage IIIB $\Rightarrow$ C

Answer: (C) Go Back to Q 56

Q57.

Solution

Concept — WT1 tumor suppressor in Wilms tumor: Wilms tumor (nephroblastoma) is the most common pediatric renal tumor. It arises from loss of the WT1 (Wilms Tumor 1) gene on chromosome 11p13. WT1 is a transcription factor that suppresses growth-promoting genes during normal genitourinary development.

Step 1 — WT1 function: WT1 encodes a zinc-finger transcription factor that normally SUPPRESSES transcription of growth-promoting genes (e.g., IGF-2, PDGFA). Loss of both alleles (two-hit hypothesis) leads to uncontrolled proliferation of metanephric blastema.

Step 2 — Histology match: Triphasic Wilms tumor (blastemal + epithelial + stromal components) + 11p13 deletion = WT1 loss-of-function.

Why other options are wrong:

- Option A: Activating RAS mutation is a proto-oncogene gain-of-function — WT1 is a tumor SUPPRESSOR (loss-of-function).
- Option B: Phosphorylating CDK4 is a function of cyclin D; WT1 does NOT activate CDK4.
- Option C: Encoding a growth factor receptor with TK activity describes RET, EGFR, etc., not WT1.
- Option D: Correct — WT1 suppresses transcription of growth-promoting genes.

Final Answer: WT1 suppresses transcription of proliferative genes; its loss causes Wilms tumor $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 57

Q58.

Solution

Concept — Iron deficiency anemia (IDA) lab profile: IDA shows: low serum iron, LOW ferritin, HIGH TIBC (compensatory increase). The RDW is elevated because of anisocytosis (mixed cell sizes during recovery or depletion). MCHC is low (hypochromic).

Step 1 — Understanding TIBC in IDA: TIBC (transferrin capacity) rises in IDA because the body upregulates transferrin synthesis to maximize iron capture from the depleted pool.

Step 2 — Confirming pattern: Low ferritin (depleted stores) + high TIBC + low serum iron = classic IDA. Ferritin is an acute-phase reactant and can be falsely normal in IDA with coexisting infection.

Why other options are wrong:

- Option A: Correct — TIBC is INCREASED in IDA.
- Option B: Decreased TIBC is seen in anemia of chronic disease (ACD), NOT IDA.
- Option C: Normal serum iron with increased ferritin suggests anemia of chronic disease or iron overload.
- Option D: Increased serum iron with decreased TIBC is seen in iron overload (hemochromatosis) or sideroblastic anemia.

Final Answer: Iron deficiency anemia = increased TIBC $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 58

Q59.

Solution

Concept — Hereditary spherocytosis (HS): HS is an autosomal dominant hemolytic anemia caused by a defect in RBC membrane cytoskeletal proteins (spectrin, ankyrin, band 3, or protein 4.2). The resulting spherocytes are osmotically fragile and destroyed in the spleen.

Step 1 — Key molecular defect: Spectrin or ankyrin deficiency disrupts the vertical linkage between lipid bilayer and cytoskeleton, causing membrane loss (microvesiculation) and sphere formation. Elevated MCHC reflects water efflux from spherical cells.

Step 2 — Diagnostic tests: Positive osmotic fragility test + elevated MCHC + family history = HS. Eosin-5-maleimide (EMA) binding test is the modern diagnostic tool.

Why other options are wrong:

- Option A: G6PD deficiency causes episodic hemolysis triggered by oxidants (drugs, infection) — not family history of chronic hemolysis with spherocytes.
- Option B: Correct — defect in spectrin/ankyrin of the RBC cytoskeleton.
- Option C: Abnormal hemoglobin (HbS) causes sickle cell disease — target cells and sickle cells, not spherocytes.
- Option D: Complement-mediated destruction describes PNH (paroxysmal nocturnal hemoglobinuria) — acquired, not familial.

Final Answer: Hereditary spherocytosis = spectrin/ankyrin cytoskeletal defect $\Rightarrow$ B

Answer: (B) Go Back to Q 59

Q60.

Solution

Concept — CML and BCR-ABL: Chronic myeloid leukemia (CML) is characterized by the Philadelphia chromosome t(9;22)(q34;q11), which juxtaposes the BCR gene on chromosome 22 with the ABL1 proto-oncogene on chromosome 9. The resulting BCR-ABL fusion protein has constitutively active tyrosine kinase activity driving uncontrolled myeloid proliferation.

Step 1 — Translocation consequence: BCR-ABL = constitutively active tyrosine kinase (no need for growth factor activation). It activates RAS, STAT5, and PI3K pathways, suppressing apoptosis and driving proliferation.

Step 2 — Clinical correlation: Massive splenomegaly + marked leukocytosis with left shift + basophilia + low LAP score + thrombocytosis = classic CML. LAP is low in CML (low in leukemic cells) vs. high in leukemoid reaction.

Why other options are wrong:

- Option A: Mutant RAS with constitutive GTPase activity is incorrect — RAS mutations IMPAIR GTPase, keeping RAS constitutively active (gain-of-function mutation loses GTP hydrolysis).
- Option B: BCL-2/IgH fusion describes follicular lymphoma t(14;18).
- Option C: Correct — BCR-ABL fusion with constitutive tyrosine kinase activity.
- Option D: WT1 loss is associated with Wilms tumor, not CML.

Final Answer: t(9;22) creates BCR-ABL fusion with constitutive tyrosine kinase activity $\Rightarrow$ C

Answer: (C) Go Back to Q 60

Q61.

Solution

Concept — Hodgkin lymphoma and Reed-Sternberg cells: Classical Hodgkin lymphoma (HL) is defined by the presence of Reed-Sternberg (RS) cells — large binucleated or multinucleated cells with prominent "owl-eye" eosinophilic nucleoli — in a reactive inflammatory background (lymphocytes, plasma cells, eosinophils, neutrophils).

Step 1 — RS cell identification: RS cells are derived from germinal center B-cells that have lost Ig expression. They stain CD15+, CD30+, CD45-. Their "owl-eye"

nucleoli are pathognomonic.

Step 2 — Clinical features: Young male, painless cervical + mediastinal adenopathy, B symptoms (fever, night sweats, weight loss) = classic cHL presentation.

Why other options are wrong:

- Option A: Correct — Reed-Sternberg cells of Hodgkin lymphoma.
- Option B: Burkitt lymphoma has a ”starry-sky” pattern from macrophages engulfing apoptotic cells — tumor cells are NOT binucleated RS cells.
- Option C: Follicular lymphoma with BCL-2 overexpression shows follicular pattern; no RS cells.
- Option D: RS cells are PATHOGNOMONIC of Hodgkin lymphoma; ”RS variant cells in non-Hodgkin lymphoma” is not a recognized entity in this context.

Final Answer: Owl-eye binucleated large cells in inflammatory background = Reed-Sternberg cells of Hodgkin lymphoma $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 61

Q62.

Solution

Concept — Foam cells in atherosclerosis: Foam cells are lipid-laden macrophages that are the hallmark of the fatty streak (earliest atherosclerotic lesion). Circulating monocytes adhere to activated endothelium, migrate into the intima, differentiate into macrophages, and scavenge oxidized LDL via scavenger receptors (SR-A, CD36) — becoming lipid-laden foam cells.

Step 1 — Foam cell origin: PRIMARILY macrophages (monocyte-derived) ingesting oxidized LDL. A smaller proportion ($<$10%) may derive from smooth muscle cells that have migrated into the intima.

Step 2 — Significance: Foam cells release inflammatory cytokines (IL-1, TNF-α), MMPs, and tissue factor, promoting plaque progression and instability.

Why other options are wrong:

- Option A: Smooth muscle cells contribute a minority of foam cells and are NOT the PRIMARY source.
- Option B: Endothelial cells do NOT become foam cells; they contribute to endothelial dysfunction but do not ingest LDL to form foam cells.

- Option C: Platelets aggregate on ruptured plaques but do NOT form foam cells.
- Option D: Correct — macrophages ingesting oxidized LDL via scavenger receptors = foam cells.

Final Answer: Foam cells = macrophages ingesting oxidized LDL $\Rightarrow$ D

Answer: (D) Go Back to Q 62

Q63.

Solution

Concept — Post-MI mechanical complications: Mechanical complications of MI occur between days 3–7 as ischemic myocardium undergoes coagulative necrosis and softening. Rupture of the interventricular septum (VSD) causes acute left-to-right shunt $\rightarrow$ step-up in O2 saturation from RA to RV on catheterization.

Step 1 — New VSD post-MI: Harsh pansystolic murmur at the left sternal border with thrill + left-to-right shunt on echo = post-infarction VSD (interventricular septal rupture). Occurs most commonly after anterior (LAD) MI, days 3–7.

Step 2 — Differentiating from acute MR: Acute mitral regurgitation (papillary muscle rupture) causes a soft murmur at the apex radiating to the axilla, with NO left-to-right shunt — instead a large V wave on PCWP.

Why other options are wrong:

- Option A: Free wall rupture causes hemopericardium and tamponade (Beck's triad: hypotension, JVD, muffled heart sounds) — NOT a new murmur.
- Option B: Correct — interventricular septal rupture causing VSD with left-to-right shunt.
- Option C: Papillary muscle rupture (acute MR) causes murmur at apex, NOT left sternal border, and NO shunt.
- Option D: LV aneurysm presents with persistent ST elevation + dyskinesis — NOT an acute new shunt murmur.

Final Answer: New left sternal pansystolic murmur + left-to-right shunt after MI = interventricular septal rupture $\Rightarrow$ B

Answer: (B) Go Back to Q 63

Q64.

Solution

Concept — Post-streptococcal glomerulonephritis (PSGN): PSGN occurs 2–4 weeks after pharyngeal strep infection (Group A Streptococcus). It is a nephritic syndrome with hematuria, RBC casts, mild-moderate proteinuria, low C3. EM shows subepithelial "humps" (immune complex deposits on the epithelial/urinary side of GBM).

Step 1 — Subepithelial humps: Subepithelial humps on EM are pathognomonic of PSGN (immune complex Type III hypersensitivity). The complexes contain streptococcal antigens, IgG, and C3 deposited on the outer (urinary) aspect of the GBM.

Step 2 — Confirming nephritic pattern: Child + post-pharyngitis + low C3 + hematuria + RBC casts + moderate proteinuria (nephritic $>$ nephrotic) = PSGN.

Why other options are wrong:

- Option A: Minimal change disease (MCD) causes nephrotic syndrome (massive proteinuria, no hematuria, no RBC casts); EM shows foot process effacement, NOT subepithelial humps.
- Option B: IgA nephropathy occurs within 24–48 hrs of URI (synpharyngitic), C3 is normal; EM shows mesangial IgA deposits (mesangial, not subepithelial).
- Option C: Correct — PSGN with subepithelial humps ("gargoyle" deposits), low C3, nephritic syndrome after strep pharyngitis.
- Option D: Membranous nephropathy shows subepithelial deposits in adults with nephrotic syndrome (not nephritic), C3 is normal.

Final Answer: Post-streptococcal GN = subepithelial humps on EM, low C3, nephritic syndrome $\Rightarrow$ C

Answer: (C) Go Back to Q 64

Q65.

Solution

Concept — Minimal change disease (MCD) treatment: MCD is the most common cause of nephrotic syndrome in children (peak 2–8 years). It is characterized by normal light microscopy, diffuse foot process effacement on EM, and NO immune deposits on IF. It is highly steroid-responsive.

Step 1 — Treatment of choice: First-line treatment for MCD in children is prednisolone (corticosteroids) for 4–8 weeks; 90% of children achieve complete remission. Corticosteroids restore the negative charge on the GBM and normalize podocyte function.

Step 2 — Subsequent management: Frequent relapsers or steroid-dependent cases are treated with cyclophosphamide or calcineurin inhibitors (tacrolimus/cyclosporine).

Why other options are wrong:

- Option A: Correct — prednisolone is the first-line treatment for childhood MCD.
- Option B: Cyclophosphamide is used for steroid-dependent or frequently relapsing MCD — NOT first-line initial therapy.
- Option C: ACE inhibitors reduce proteinuria but do NOT treat the underlying disease; they are adjuncts.
- Option D: Furosemide alone addresses edema symptomatically but does NOT treat the nephrotic syndrome.

Final Answer: MCD treatment of choice = prednisolone $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 65

Q66.

Solution

Concept — Mallory-Denk bodies in alcoholic liver disease: Mallory-Denk bodies (MDB) are intracytoplasmic eosinophilic inclusions seen in hepatocytes in alcoholic hepatitis (also Wilson disease, NASH, and primary biliary cholangitis). They are composed of aggregated intermediate filaments — specifically cytokeratin 8 and cytokeratin 18 — along with ubiquitin and p62.

Step 1 — Alcohol-specific liver pathology: Alcoholic hepatitis features: (1) macrovesicular steatosis, (2) hepatocyte ballooning, (3) Mallory-Denk bodies, (4)

neutrophilic infiltration, (5) pericellular "chicken wire" fibrosis.

Step 2 — MDB composition: MDBs are NOT glycogen, NOT lipid, NOT alpha-1-antitrypsin — they are cytokeratin 8/18 intermediate filament aggregates (the hepatocyte's cytoskeletal proteins).

Why other options are wrong:

- Option A: Glycogen granules are PAS-positive, seen normally in hepatocytes or in glycogen storage diseases; they do NOT form eosinophilic inclusions.
- Option B: Fat vacuoles cause macrovesicular steatosis (clear optically empty vacuoles), which is separate from MDBs.
- Option C: PAS-positive α-1-antitrypsin globules are seen in α-1-AT deficiency, NOT alcoholic hepatitis.
- Option D: Correct — Mallory-Denk bodies = aggregated cytokeratin 8/18 intermediate filaments.

Final Answer: Mallory-Denk bodies = aggregated cytokeratin 8/18 intermediate filaments $\Rightarrow$ D

Answer: (D) Go Back to Q 66

Q67.

Solution

Concept — Hepatitis B serology and infectivity: HBeAg (hepatitis B e antigen) is a marker of active viral replication and HIGH infectivity. It is a soluble secreted form of the core protein. HBeAg positivity correlates with high HBV DNA levels and greater risk of transmission.

Step 1 — Markers of infectivity: HBsAg+ (surface antigen) indicates infection. HBeAg+ indicates HIGH viral replication $\rightarrow$ HIGH infectivity. Anti-HBe+ (e antibody) indicates low replication phase $\rightarrow$ LOW infectivity (except in pre-core mutants).

Step 2 — Other markers: Anti-HBs (surface antibody) = immunity (post-infection or post-vaccination). Anti-HBc IgM = acute infection marker. Anti-HBc IgG = past/current infection.

Why other options are wrong:

- Option A: Anti-HBs positivity indicates IMMUNITY to HBV (cleared infection or vaccination), NOT active high infectivity.

- Option B: Correct — HBeAg positivity indicates high viral replication and high infectivity.
- Option C: Anti-HBe positivity indicates LOW replication / low infectivity phase (unless pre-core mutant).
- Option D: Anti-HBc IgG alone with negative HBsAg represents past resolved infection — NOT active high infectivity.

Final Answer: HBeAg positivity = high viral replication = HIGH infectivity $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 67

Q68.

Solution

Concept — Portal hypertension and varices: In liver cirrhosis, hepatic fibrosis and regenerating nodules distort the sinusoidal architecture, increasing intrahepatic vascular resistance $\rightarrow$ portal hypertension. Elevated portal pressure causes portosystemic collaterals to open (esophageal varices, hemorrhoids, caput medusae).

Step 1 — Mechanism: Increased intrahepatic vascular resistance (due to fibrosis, activated hepatic stellate cells, and sinusoidal compression) $\rightarrow$ elevated portal venous pressure $\rightarrow$ portosystemic shunting $\rightarrow$ esophageal varices.

Step 2 — Role of endothelin and NO: In cirrhosis, there is INCREASED endothelin (vasoconstriction) and REDUCED intrahepatic NO, worsening intrahepatic resistance. Splanchnic vasodilation (from systemic NO excess) also worsens portal hypertension.

Why other options are wrong:

- Option A: Decreased hepatic arterial pressure does NOT cause portal hypertension; the portal vein system is separate.
- Option B: Hypoalbuminemia causes ascites and peripheral edema from reduced oncotic pressure, NOT variceal formation (which requires portal hypertension).
- Option C: Correct — portal hypertension from increased intrahepatic vascular resistance.
- Option D: Portal vein thrombosis can cause portal hypertension but is a separate mechanism; the PRIMARY mechanism in cirrhosis is intrahepatic resistance, not thrombosis.

Final Answer: Esophageal varices in cirrhosis = portal hypertension from in-

creased intrahepatic vascular resistance $\Rightarrow$ C

Answer: (C) Go Back to Q 68

Q69.

Solution

Concept — Community-acquired pneumonia (CAP) etiology: *Streptococcus pneumoniae* (pneumococcus) is the MOST COMMON cause of community-acquired lobar pneumonia in adults worldwide. It produces α-hemolysis (viridans-type on blood agar) but is classified by its polysaccharide capsule and pneumolysin toxin.

Step 1 — Gram stain identification: Gram-positive diplococci in pairs (lanceolate) = *S. pneumoniae*. This is confirmed by optochin sensitivity and bile solubility.

Step 2 — CAP epidemiology: *S. pneumoniae*: most common overall. Atypicals (Mycoplasma, Chlamydophila, Legionella): common in younger adults with atypical features. Klebsiella: alcoholics/aspiration. Staph: post-influenza.

Why other options are wrong:

- Option A: Correct — *S. pneumoniae* is the most common cause of CAP in adults.
- Option B: *Klebsiella pneumoniae* causes lobar pneumonia in alcoholics with "currant jelly" sputum — it is Gram-negative, NOT Gram-positive diplococci.
- Option C: *Staphylococcus aureus* causes post-influenza or hospital-acquired pneumonia with cavitation — Gram-positive cocci in clusters.
- Option D: *H. influenzae* causes bronchopneumonia in COPD patients — it is Gram-negative coccobacilli, NOT diplococci.

Final Answer: Most common CAP in adults = *Streptococcus pneumoniae* $\Rightarrow$ A

Answer: (A) Go Back to Q 69

Q70.

Solution

Concept — TB pathology in severe immunocompromise: Classic granuloma formation requires intact Th1 immunity (CD4+ T cells secreting IFN-γ activating macrophages). In severe HIV (CD4 $<$100 cells/μL), impaired Th1 response leads to failure to form well-organized granulomas.

Step 1 — Effect of severe immunosuppression on TB pathology: With very low CD4 counts, TB in HIV presents atypically: (1) NUMEROUS organisms (poor containment), (2) diffuse infiltrates / miliary dissemination, (3) extensive caseation possible, but (4) POORLY FORMED or ABSENT granulomas (inadequate macrophage activation).

Step 2 — Least likely finding: Well-formed epithelioid granulomas with Langhans giant cells require competent Th1 immunity. In CD4 80 cells/μL, these are the LEAST likely to be seen — instead there are sheets of poorly organized macrophages with numerous AFB.

Why other options are wrong:

- Option A: Numerous AFB are EXPECTED due to poor containment of mycobacteria.
- Option B: Widespread caseation CAN occur even in immunocompromised hosts.
- Option C: Miliary dissemination is characteristic of severe immunosuppression with TB.
- Option D: Correct — well-formed granulomas with Langhans giant cells are LEAST likely in severe immunosuppression.

Final Answer: Least likely in severe HIV-associated TB = well-formed granulomas with Langhans giant cells $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 70

Q71.

Solution

Concept — Lung cancer histology: Squamous cell carcinoma (SCC) is strongly associated with heavy smoking and central/hilar location. Histologically: large polygonal cells with intercellular bridges (desmosomes) and keratin pearl formation. It can cause hypercalcemia via PTHrP secretion (paraneoplastic).

Step 1 — Histological identification: Intercellular bridges (desmosomes) + keratin pearls = squamous differentiation = squamous cell carcinoma. Central/hilar origin is also characteristic.

Step 2 — Hypercalcemia link: SCC produces PTHrP (parathyroid hormone-related protein), which activates PTH receptors → hypercalcemia without elevated true PTH.

Why other options are wrong:

- Option A: Small cell carcinoma (oat cell) shows small neuroendocrine cells with scant cytoplasm, nuclear molding, and "salt and pepper" chromatin — NO keratin pearls; causes SIADH, Cushing syndrome.
- Option B: Correct — squamous cell carcinoma with intercellular bridges and keratin pearls.
- Option C: Adenocarcinoma occurs peripherally, shows glandular formation and mucin production — NOT keratin pearls; it is the most common lung cancer in non-smokers.
- Option D: Large cell carcinoma is a diagnosis of exclusion (anaplastic, no squamous or glandular differentiation).

Final Answer: Keratin pearls + intercellular bridges in hilar lung mass = squamous cell carcinoma ⇒ B

Answer: (B) Go Back to Q 71

Q72.

Solution

Concept — Tumor markers in clinical practice: Tumor markers are substances (proteins, glycoproteins, hormones) produced by tumors detectable in blood/urine. They are primarily used for MONITORING treatment response, detecting recurrence, and prognosis — NOT for population screening (lack specificity/sensitivity for this purpose).

Step 1 — Key principle: No tumor marker is specific enough for population-based screening (low prevalence = many false positives). Markers are best used in already-diagnosed patients.

Step 2 — Specific markers: PSA = prostate; AFP = hepatocellular carcinoma + germ cell tumors; CEA = colorectal, gastric, breast (non-specific); CA-125 = ovarian cancer (monitoring, not screening).

Why other options are wrong:

- Option A: AFP is the marker for hepatocellular carcinoma and non-seminomatous germ cell tumors, NOT prostate cancer. PSA is the prostate marker.
- Option B: CEA is NOT specific for colorectal cancer and is NOT recommended for population-based screening (elevated in many benign conditions).
- Option C: Correct — tumor markers are used for monitoring treatment response and detecting recurrence.
- Option D: CA-125 lacks sufficient specificity for ovarian cancer screening in the general population (elevated in endometriosis, PID, ascites); it is used for monitoring, NOT screening.

Final Answer: Tumor markers are useful for monitoring treatment response and detecting recurrence $\Rightarrow$ C

Answer: (C) Go Back to Q 72

Q73.

Solution

Concept — Epinephrine in anaphylaxis: Epinephrine (adrenaline) is the drug of choice for anaphylactic shock because it acts on all adrenergic receptor subtypes: α_1 (vasoconstriction, raising BP), β_1 (positive chronotropy/inotropy, increasing cardiac output), and β_2 (bronchodilation, reversing bronchospasm). IM route (lateral thigh) is preferred for rapid, reliable absorption.

Step 1 — Mechanism in anaphylaxis: α_1: reverses vasodilation and angioedema. β_1: treats hypotension and compensatory tachycardia with positive inotropy. β_2: reverses bronchospasm. Also suppresses mast cell mediator release (β_2).

Step 2 — Route and dose: 0.3–0.5 mg of 1:1000 solution IM (vastus lateralis). IV route reserved for cardiac arrest or refractory anaphylaxis in ICU.

Why other options are wrong:

- Option A: Correct — epinephrine 0.5 mg IM is the drug of choice for anaphylaxis.
- Option B: Norepinephrine has minimal β_2 activity $\rightarrow$ does NOT reverse bronchospasm; used for septic shock, not anaphylaxis.
- Option C: Phenylephrine is a pure α_1 agonist $\rightarrow$ raises BP but causes reflex bradycardia and has NO bronchodilatory effect.
- Option D: Dopamine at 5 μg/kg/min (dopaminergic dose) mainly acts on D1 receptors to increase renal blood flow — insufficient for anaphylaxis.

Final Answer: Drug of choice for anaphylactic shock = epinephrine IM (α_1, β_1, β_2 actions) $\Rightarrow$ A

Answer: (A) Go Back to Q 73

Q74.

Solution

Concept — Pralidoxime (PAM) in organophosphate poisoning: Pralidoxime is an acetylcholinesterase (AChE) reactivator. It dephosphorylates the OP-AChE complex, restoring AChE activity. However, the OP-AChE complex undergoes irreversible "aging" (loss of an alkyl group) over 24–48 hours, after which PAM is ineffective. Therefore, early administration is CRITICAL.

Step 1 — Window of efficacy: PAM must be given within 24–48 hours before aging occurs. After aging, PAM cannot remove the phosphate group — the bond becomes permanent.

Step 2 — PAM limitations: PAM does NOT cross the BBB well $\rightarrow$ primarily addresses peripheral (nicotinic) features (muscle fasciculations, weakness, paralysis). Atropine is given FIRST (and concomitantly) for muscarinic features.

Why other options are wrong:

- Option A: After 72 hours, aging is complete $\rightarrow$ PAM is INEFFECTIVE; delaying is harmful.
- Option B: PAM should be started IMMEDIATELY with atropine (simultaneously), not after atropine requirements are met.
- Option C: PAM as sole agent is inadequate; atropine must always be co-administered for muscarinic features.
- Option D: Correct — PAM must be given within 24–48 hours before aging makes AChE irreversibly inhibited.

Final Answer: Pralidoxime is effective only within 24–48 hours before "aging" of AChE-OP complex $\Rightarrow$ D

Answer: (D) Go Back to Q 74

Q75.

Solution

Concept — Valproate teratogenicity: Sodium valproate is the antiepileptic with the HIGHEST teratogenic risk. Its most significant congenital anomaly is neural tube defects (spina bifida, 1–2% risk vs. 0.06% background) due to folate antagonism. Other risks: cardiac defects, craniofacial abnormalities, neonatal hepatotoxicity.

Step 1 — Neural tube defect risk: Valproate inhibits folic acid metabolism $\rightarrow$ folate deficiency during neurulation (weeks 3–4) $\rightarrow$ failure of neural tube closure $\rightarrow$ spina bifida (most common) or anencephaly.

Step 2 — Clinical guidance: High-dose folic acid (5 mg/day) should be given preconceptionally and throughout the first trimester to reduce (but not eliminate) the risk.

Why other options are wrong:

- Option A: Neonatal hypoglycemia is associated with sulphonylureas and beta-blockers used near term — not valproate's primary teratogenic risk.
- Option B: Correct — neural tube defects (spina bifida) are the most significant teratogenic risk of valproate in the first trimester.
- Option C: Cleft palate is associated with phenytoin and benzodiazepines (fetal hydantoin syndrome) — not the PRIMARY risk of valproate.
- Option D: Neonatal hemorrhage from vitamin K deficiency is associated with enzyme-inducing antiepileptics (phenytoin, carbamazepine, phenobarbitone) that increase vitamin K catabolism — not valproate's primary risk.

Final Answer: Valproate's most significant teratogenic risk = neural tube defects (spina bifida) $\Rightarrow$ B

Answer: (B) Go Back to Q 75

Q76.

Solution

Concept — Tardive dyskinesia (TD): Tardive dyskinesia is a late-onset (tardive = delayed) movement disorder caused by prolonged dopamine receptor blockade (D2 receptors in the striatum). Chronic blockade leads to receptor upregulation (supersensitivity), causing involuntary, repetitive orofacial and limb movements that PERSIST even after drug reduction or cessation.

Step 1 — Clinical features of TD: Slow, writhing (choreoathetoid) or repetitive movements of tongue, lips, jaw (lip-smacking, tongue protrusion), trunk, and limbs; onset after months-years of antipsychotic use; PERSISTS despite drug reduction.

Step 2 — Distinguishing from other EPSs:

- Akathisia: restlessness, urge to move legs — occurs early (weeks).
- Acute dystonia: painful sustained muscle contraction (torticollis) — occurs within days.
- TD: slow writhing involuntary movements — late onset (months-years), persists.
- NMS: rigidity, fever, autonomic instability, elevated CK — medical emergency.

Why other options are wrong:

- Option A: Akathisia = inner restlessness and motor restlessness (pacing); not slow writhing movements.
- Option B: Acute dystonia = occurs within the FIRST 24–72 hours of starting an antipsychotic; not after 6 months.
- Option C: Correct — tardive dyskinesia after 6 months of haloperidol with persistent orofacial movements.
- Option D: NMS presents with hyperthermia, "lead-pipe" rigidity, autonomic instability, and elevated CK — not slow writhing movements.

Final Answer: Late-onset persistent orofacial involuntary movements on antipsychotics = tardive dyskinesia $\Rightarrow$ C

Answer: (C) Go Back to Q 76

Q77.

Solution

Concept — Serotonin syndrome: Serotonin syndrome (SS) is a potentially life-threatening drug-drug interaction caused by excess serotonergic activity at 5-HT_{1A} and 5-HT_{2A} receptors. It is characterized by the TRIAD: (1) Altered mental status (agitation, confusion), (2) Autonomic instability (hyperthermia, diaphoresis, tachycardia), (3) Neuromuscular abnormalities (myoclonus, hyperreflexia, clonus).

Step 1 — Drug interaction: Fluoxetine (SSRI) inhibits serotonin reuptake via SERT. Tramadol inhibits serotonin reuptake AND weakly activates μ-opioid receptors. Combined $\rightarrow$ marked increase in synaptic serotonin $\rightarrow$ serotonin syndrome.

Step 2 — Key differentiator from NMS: SS = acute onset (hours), myoclonus/hyperreflexia, responds to cyproheptadine. NMS = subacute onset (days), "lead-pipe" rigidity, responds to bromocriptine + dantrolene.

Why other options are wrong:

- Option A: Correct — serotonin syndrome from excess serotonergic activity (fluoxetine + tramadol).
- Option B: NMS = dopamine blockade by antipsychotics — NOT the mechanism here; tramadol and fluoxetine do not block dopamine.
- Option C: Anticholinergic toxidrome = dry skin, urinary retention, absent bowel sounds, delirium — none of these are described here.
- Option D: Sympathomimetic crisis involves cocaine, amphetamines — NOT fluoxetine + tramadol.

Final Answer: Fluoxetine + tramadol $\rightarrow$ excess serotonin $\rightarrow$ serotonin syndrome $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 77

Q78.

Solution

Concept — Opioid overdose reversal: Naloxone is a pure competitive μ-opioid receptor ANTAGONIST with high affinity and rapid onset. It reverses opioid-induced respiratory depression, CNS depression, and miosis. It has NO agonist activity and does not cause respiratory depression.

Step 1 — Drug of choice: Naloxone IV (or IM/intranasal) is the antidote for

opioid overdose. Dose: 0.4–2 mg IV, repeat every 2–3 min if needed. Its duration (1–4 hours) may be shorter than the opioid → repeat dosing or infusion may be needed.

Step 2 — Other opioid-related drugs: Nalbuphine, pentazocine = mixed agonist-antagonists (partial agonists). Buprenorphine = partial μ-agonist + κ-antagonist. None are suitable for reversal of full agonist opioid overdose.

Why other options are wrong:

- Option A: Nalbuphine is a mixed agonist-antagonist — it would PARTIALLY reverse opioid effects but itself has agonist activity and does NOT fully reverse overdose.
- Option B: Buprenorphine is a partial μ-agonist used for opioid dependence — it COMPETES with morphine but is NOT the antidote for acute overdose.
- Option C: Pentazocine is a mixed agonist-antagonist — NOT used for reversal; has its own opioid agonist effects.
- Option D: Correct — naloxone IV is the drug of choice for opioid overdose reversal.

Final Answer: Opioid overdose reversal = naloxone IV ⇒ D

Answer: (D) Go Back to Q 78

Q79.

Solution

Concept — Propofol mechanism of action: Propofol (2,6-diisopropylphenol) is a short-acting IV general anaesthetic. Its primary mechanism is POSITIVE ALLOSTERIC MODULATION of $GABA_A$ receptors, enhancing chloride ion influx → hyperpolarization of the neuronal membrane → CNS depression and loss of consciousness.

Step 1 — $GABA_A$ mechanism: Propofol binds to a site on the $GABA_A$ receptor (distinct from benzodiazepine and barbiturate sites) and increases the frequency and duration of Cl^- channel opening in response to GABA, producing profound CNS depression.

Step 2 — Clinical properties: Propofol: rapid onset (30 sec), short duration (5–10 min), anti-emetic, causes apnea and hypotension; used for TIVA and induction.

Why other options are wrong:

- Option A: NMDA glutamate receptor blockade is the mechanism of KE-

TAMINE — not propofol.

- Option B: Correct — propofol potentiates $GABA_A$ receptor-mediated chloride influx.
- Option C: Voltage-gated Na^+ channel inhibition is the mechanism of LOCAL anaesthetics (lidocaine) and some antiepileptics — not propofol.
- Option D: μ-opioid receptor agonism is the mechanism of morphine/fentanyl — not propofol; propofol has no opioid activity.

Final Answer: Propofol mechanism = potentiation of $GABA_A$ receptor Cl^- influx $\Rightarrow$ B

Answer: (B) Go Back to Q 79

Q80.

Solution

Concept — Potency vs. efficacy on dose-response curves: Potency = the dose (ED50) required to produce 50% of the maximum effect — a LEFT-shifted curve = more potent (lower dose needed). Efficacy = maximum effect (Emax) achievable — the PLATEAU of the sigmoid curve.

Step 1 — Reading the graph: Drug A has a lower ED50 (left-shifted) than Drug B $\rightarrow$ Drug A is MORE POTENT. Both curves reach the same maximum effect (100%) plateau $\rightarrow$ SAME efficacy.

Step 2 — Conclusion: Drug A: greater potency (lower ED50), same maximum efficacy as Drug B.

Why other options are wrong:

- Option A: Greater efficacy would require Drug A to reach a HIGHER plateau — both reach the same 100% plateau, so efficacy is equal.
- Option B: Same efficacy but lesser potency describes Drug B (higher ED50), not Drug A.
- Option C: Correct — Drug A has greater potency (lower ED50) with the same maximum efficacy as Drug B.
- Option D: Lesser potency and lesser efficacy describes a drug with a higher ED50 AND lower plateau — Drug A has neither.

Final Answer: Drug A vs Drug B: greater potency, same maximum efficacy $\Rightarrow$ C

Answer: (C) Go Back to Q 80

Q81.

Solution

Concept — Alpha-1 blockers for hypertension + BPH: α_1-adrenergic receptor blockers (prazosin, terazosin, doxazosin, tamsulosin) relax smooth muscle in blood vessels (reducing peripheral vascular resistance $\rightarrow$ antihypertensive) AND relax the internal urethral sphincter and bladder neck smooth muscle (reducing outflow obstruction $\rightarrow$ improved urine flow in BPH).

Step 1 — Dual mechanism: α_1 receptors are present in: (1) vascular smooth muscle $\rightarrow$ blockade = vasodilation = $\downarrow$BP; (2) prostate stroma and bladder neck $\rightarrow$ blockade = relaxation = improved urinary flow in BPH.

Step 2 — Drug selection: Non-selective α_1 blockers (doxazosin, terazosin) treat both. Tamsulosin (α_{1A} selective) is prostate-selective with less systemic hypotension.

Why other options are wrong:

- Option A: β_1 blockers treat hypertension by reducing cardiac output but do NOT relax the bladder neck or improve BPH symptoms.
- Option B: ACE inhibitors treat hypertension effectively but have NO effect on urethral smooth muscle in BPH.
- Option C: Calcium channel blockers are excellent antihypertensives but do NOT relax the internal urethral sphincter.
- Option D: Correct — α_1 blockade causes vasodilation (antihypertensive) AND bladder neck relaxation (BPH treatment).

Final Answer: Drug for hypertension + BPH = α_1 blocker (vasodilation + bladder neck relaxation) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 81

Q82.

Solution

Concept — Amiodarone for stable VT: Amiodarone is a class III antiarrhythmic (primarily potassium channel blockade) but also has class I (Na^+ channel), class II (β-blockade), and class IV (Ca^{2+} channel) properties. It is the preferred drug for hemodynamically STABLE ventricular tachycardia in patients with structural heart disease (post-MI), per ACLS guidelines.

Step 1 — Drug choice for stable VT: Hemodynamically stable VT: amiodarone IV

(150 mg over 10 min, then infusion). If unstable → synchronized DC cardioversion.

Step 2 — Amiodarone classification: Despite having Class IA/IB-like sodium channel effects, amiodarone's primary classification is Class III (K^+ channel blocker), prolonging the action potential duration and refractory period.

Why other options are wrong:

- Option A: Correct — amiodarone IV is the drug of choice for hemodynamically stable VT.
- Option B: Verapamil (Class IV) is used for SVT (AV nodal re-entrant) — it is CONTRAINDICATED in VT as it can worsen hemodynamics.
- Option C: Digoxin increases vagal tone and is used for rate control in AF — NOT for VT; it can be proarrhythmic.
- Option D: Adenosine terminates AV-node-dependent SVT (AVNRT, AVRT) — it is ineffective for VT.

Final Answer: Hemodynamically stable VT drug of choice = amiodarone IV ⇒ A

Answer: (A) Go Back to Q 82

Q83.

Solution

Concept — Mortality-reducing drugs in HFrEF: Evidence-based mortality-reducing drugs in HFrEF: ACE inhibitors/ARBs, beta-blockers, aldosterone antagonists (spironolactone/eplerenone), SGLT2 inhibitors, and sacubitril/valsartan. Aldosterone antagonists (RALES trial: spironolactone) reduce mortality by 30% in severe HFrEF when added to standard therapy.

Step 1 — Spironolactone mechanism: Spironolactone competitively blocks aldosterone receptors, reducing sodium retention, potassium wasting, cardiac fibrosis, and collagen deposition — all of which contribute to HF progression.

Step 2 — Evidence: RALES trial (spironolactone) and EMPHASIS-HF (eplerenone) both showed significant mortality reduction in HFrEF on top of standard therapy (ACE inhibitor + beta-blocker).

Why other options are wrong:

- Option A: Digoxin improves symptoms and reduces hospitalization in HFrEF but does NOT reduce mortality (DIG trial).

- Option B: Correct — spironolactone (aldosterone antagonist) reduces mortality in HFrEF (RALES trial).
- Option C: Hydralazine alone has no mortality benefit; hydralazine + isosorbide dinitrate (ISDN) has benefit in African Americans who are ACE inhibitor-intolerant (A-HeFT trial).
- Option D: Amlodipine (calcium channel blocker) is not harmful in HFrEF but does NOT reduce mortality; most CCBs are generally avoided in HFrEF.

Final Answer: Mortality-reducing addition in HFrEF on ACEI + beta-blocker = spironolactone $\Rightarrow$ B

Answer: (B) Go Back to Q 83

Q84.

Solution

Concept — Rivaroxaban mechanism: Rivaroxaban is a direct oral anticoagulant (DOAC) that DIRECTLY and SELECTIVELY inhibits Factor Xa (both free Factor Xa and Factor Xa within the prothrombinase complex), preventing thrombin generation without requiring antithrombin as a cofactor.

Step 1 — Factor Xa inhibition: Factor Xa converts prothrombin (Factor II) to thrombin (Factor IIa). Rivaroxaban blocks this step $\rightarrow$ prevents thrombin generation $\rightarrow$ prevents fibrin clot formation.

Step 2 — DOACs classification: Factor Xa inhibitors: rivaroxaban, apixaban, edoxaban. Direct thrombin (IIa) inhibitors: dabigatran.

Why other options are wrong:

- Option A: Direct thrombin (IIa) inhibition is the mechanism of DABIGATRAN, not rivaroxaban.
- Option B: Inhibition of vitamin K-dependent clotting factor synthesis is the mechanism of WARFARIN (blocks vitamin K epoxide reductase).
- Option C: Correct — rivaroxaban directly inhibits Factor Xa.
- Option D: Activation of antithrombin III is the mechanism of HEPARIN (and LMWH) — rivaroxaban does NOT require antithrombin.

Final Answer: Rivaroxaban = direct Factor Xa inhibitor $\Rightarrow$ C

Answer: (C) Go Back to Q 84

Q85.

Solution

Concept — Penicillin allergy and alternatives: Urticaria to amoxicillin (aminopenicillin) is a true IgE-mediated penicillin allergy. Cephalosporins share the β-lactam ring but have different side chains; cross-reactivity with true penicillin allergy is $<2\%$. However, when the allergy was to a specific aminopenicillin (ampicillin/amoxicillin), the R1 side chain is shared with first-generation cephalosporins like cephalexin — but cross-reactivity is still low. For **true penicillin allergy**, macrolides (azithromycin) are the safest alternative with no structural similarity.

Step 1 — Allergy classification: Urticaria = likely IgE-mediated. Contraindicate ALL penicillins (ampicillin, amoxicillin, piperacillin). Cephalosporins carry a small but non-zero risk. Macrolides are completely unrelated structurally.

Step 2 — Safe alternative: Azithromycin (macrolide) — no cross-reactivity with beta-lactams — is the safest alternative for penicillin-allergic children with otitis media.

Why other options are wrong:

- Option A: Ampicillin is also an aminopenicillin — same allergen, absolutely contraindicated.
- Option B: Cephalexin shares the aminopenicillin R1 side chain $\rightarrow$ slightly higher cross-reactivity risk in amoxicillin allergy; avoid when a safer alternative exists.
- Option C: Piperacillin-tazobactam is a penicillin — absolutely contraindicated in penicillin allergy.
- Option D: Correct — azithromycin (macrolide) with no cross-reactivity to penicillins.

Final Answer: Penicillin allergy (urticaria) $\rightarrow$ safest alternative = azithromycin (macrolide) $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 85

Q86.

Solution

Concept — Aminoglycoside toxicity monitoring: Aminoglycosides (gentamicin, amikacin, tobramycin) are concentration-dependent antibiotics with two major adverse effects: (1) nephrotoxicity (dose-dependent, reversible) and (2) ototoxicity (cochlear and vestibular, often irreversible). Monitoring serum trough levels (and peaks in conventional dosing) helps prevent toxicity.

Step 1 — Trough levels and nephrotoxicity: Trough levels (just before next dose) reflect drug accumulation in the kidney. Elevated troughs correlate with nephrotoxicity. The goal is to keep trough <2 μg/mL (gentamicin) for conventional dosing.

Step 2 — Audiometry: Ototoxicity (cochlear: high-frequency hearing loss $\rightarrow$ tinnitus $\rightarrow$ deafness; vestibular: dizziness) monitoring requires regular audiometry, especially in prolonged courses, elderly patients, or pre-existing hearing loss.

Why other options are wrong:

- Option A: Correct — trough serum levels (nephrotoxicity monitoring) and audiometry (ototoxicity monitoring) are the most important parameters.
- Option B: LFTs for hepatotoxicity are NOT required for aminoglycosides; hepatotoxicity is not a clinically significant adverse effect.
- Option C: Thrombocytopenia is NOT a recognized adverse effect of aminoglycosides.
- Option D: INR for anticoagulant interaction is not relevant; aminoglycosides do NOT significantly affect INR.

Final Answer: Aminoglycoside monitoring = trough serum levels (nephrotoxicity) + audiometry (ototoxicity) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 86

Q87.

Solution

Concept — Fluoroquinolone-divalent cation chelation: Fluoroquinolones (ciprofloxacin, levofloxacin, moxifloxacin) form stable chelate complexes with divalent (Mg^{2+}, Ca^{2+}, Al^{3+}, Fe^{2+}, Zn^{2+}) and trivalent cations in the GI tract. This chelation MARKEDLY reduces oral absorption (bioavailability drops by 50–90%) — making the drug ineffective.

Step 1 — Clinical consequence: Antacids containing Mg^{2+} or Al^{3+}, iron supplements, dairy products, and multivitamins all impair fluoroquinolone absorption. Ciprofloxacin should be taken 2 hours BEFORE or 6 hours AFTER antacids.

Step 2 — Other fluoroquinolone interactions: Ciprofloxacin inhibits CYP1A2 $\rightarrow$ increases theophylline levels. It also prolongs QTc (caution with other QTc-prolonging drugs). These are separate from the antacid interaction.

Why other options are wrong:

- Option A: Ciprofloxacin does NOT increase renal excretion of magnesium; this is not a pharmacokinetic concern.
- Option B: Magnesium does NOT induce CYP450 enzymes — this mechanism is incorrect.
- Option C: Correct — divalent cations (Mg^{2+}) chelate ciprofloxacin in the gut, markedly reducing absorption.
- Option D: QTc prolongation with Mg is a valid concern in hypomagnesemia, but the PRIMARY pharmacokinetic concern with antacids is reduced absorption via chelation, not QTc.

Final Answer: Mg^{2+} in antacids chelates ciprofloxacin $\rightarrow$ markedly reduced oral absorption $\Rightarrow$ C

Answer: (C) Go Back to Q 87

Q88.

Solution

Concept — Amphotericin B mechanism: Amphotericin B is a polyene antifungal that binds to ERGOSTEROL (the primary sterol in fungal cell membranes — analogous to cholesterol in mammalian cells). This binding inserts pores into the fungal membrane $\rightarrow$ loss of membrane integrity $\rightarrow$ leakage of ions (K^+, Na^+, H^+) $\rightarrow$ fungal cell death. The higher affinity for ergosterol vs cholesterol accounts for selectivity (though cholesterol binding causes nephrotoxicity and other side effects).

Step 1 — Ergosterol binding: Amphotericin B $\rightarrow$ binds ergosterol $\rightarrow$ forms pores $\rightarrow$ disrupts membrane permeability $\rightarrow$ fungicidal.

Step 2 — Clinical context: Cryptococcal meningitis in HIV = treated with Amphotericin B (induction phase) + flucytosine, followed by fluconazole consolidation and maintenance.

Why other options are wrong:

- Option A: Inhibition of beta-glucan synthase (1,3-β-d-glucan) is the mechanism of ECHINOCANDINS (caspofungin, micafungin, anidulafungin) — not amphotericin B.
- Option B: Correct — amphotericin B binds ergosterol causing pore formation.
- Option C: Inhibition of lanosterol 14α-demethylase (CYP51) is the mechanism of AZOLES (fluconazole, itraconazole, voriconazole).
- Option D: Inhibition of fungal DNA synthesis (via conversion to 5-fluorouracil) is the mechanism of FLUCYTOSINE — not amphotericin B.

Final Answer: Amphotericin B mechanism = binding ergosterol $\rightarrow$ forming membrane pores $\Rightarrow$ B

Answer: (B) Go Back to Q 88

Q89.

Solution

Concept — HIV integrase strand transfer inhibitors (INSTIs): Dolutegravir belongs to the INSTI class of antiretroviral drugs. After HIV reverse transcriptase converts viral RNA to double-stranded DNA, the integrase enzyme catalyzes the integration of viral DNA into the host cell genome. INSTIs block the strand transfer step of this integration process.

Step 1 — INSTI mechanism: Dolutegravir (and raltegravir, elvitegravir, bictegravir) bind to the catalytic core of HIV integrase and chelate the Mg^{2+} ions in the active site, blocking the strand transfer step $\rightarrow$ viral DNA cannot integrate into host genome $\rightarrow$ no productive infection.

Step 2 — First-line regimen: TDF + FTC + DTG (tenofovir + emtricitabine + dolutegravir) is the preferred first-line ART globally (WHO 2024) due to high efficacy, high barrier to resistance, and favorable tolerability.

Why other options are wrong:

- Option A: NRTI (tenofovir, emtricitabine, abacavir, zidovudine) inhibit reverse transcriptase — dolutegravir is NOT an NRTI.
- Option B: Protease inhibitors (lopinavir, atazanavir, darunavir) prevent cleavage of the viral gag-pol polyprotein — dolutegravir does NOT inhibit protease.
- Option C: NNRTIs (efavirenz, nevirapine, rilpivirine) bind the allosteric site

of reverse transcriptase — dolutegravir does NOT affect reverse transcriptase.

- Option D: Correct — dolutegravir is an INSTI that blocks integration of viral DNA into the host genome.

Final Answer: Dolutegravir = INSTI — prevents HIV DNA integration into host genome ⇒ D

Answer: (D) Go Back to Q 89

Q90.

Solution

Concept — Isoniazid-induced peripheral neuropathy: Isoniazid (INH) is the most important first-line antitubercular drug. Its principal adverse effect is peripheral neuropathy, caused by INH-mediated depletion of pyridoxine (vitamin B6). INH is a structural analog of pyridoxine and inhibits pyridoxal phosphate synthesis, which is essential for myelin formation and neural function.

Step 1 — Mechanism: INH inhibits pyridoxal kinase → reduced active pyridoxal-5-phosphate (PLP) → impaired GABA synthesis and neural metabolism → peripheral neuropathy (sensory > motor; glove-and-stocking pattern).

Step 2 — Prevention and treatment: Pyridoxine (B6) 25–50 mg/day is given prophylactically to patients at high risk (malnourished, alcoholics, diabetics, elderly, pregnant). Treatment of established neuropathy = high-dose B6.

Why other options are wrong:

- Option A: Rifampicin inhibits mycobacterial DNA-dependent RNA polymerase. Its neurological toxicity includes flu-like syndrome but NOT peripheral neuropathy.
- Option B: Ethambutol causes OPTIC NEURITIS (central scotoma, red-green color blindness) — NOT peripheral neuropathy. This is the distinguishing exam point.
- Option C: Correct — isoniazid causes peripheral neuropathy via pyridoxine (B6) depletion.
- Option D: Pyrazinamide inhibits fatty acid synthesis and causes HYPERURICEMIA (arthropathy), hepatotoxicity — NOT peripheral neuropathy.

Final Answer: Isoniazid → pyridoxine depletion → peripheral neuropathy ⇒ C

Answer: (C) Go Back to Q 90

Q91.

Solution

Concept — NSAID-induced gastric mucosal injury: The most important mechanism of NSAID-induced GI injury is inhibition of COX-1 (constitutive), which is responsible for synthesizing cytoprotective prostaglandins (PGE2 and PGI2) in the gastric mucosa. These prostaglandins: (1) stimulate mucus and bicarbonate secretion, (2) maintain mucosal blood flow, (3) inhibit acid secretion.

Step 1 — COX-1 vs COX-2: COX-1 (constitutive) = GI protection, platelet thromboxane, renal blood flow. COX-2 (inducible) = inflammation, fever, pain, GI injury. NSAIDs (non-selective) inhibit BOTH — COX-1 inhibition is responsible for GI toxicity.

Step 2 — Clinical importance: Selective COX-2 inhibitors (celecoxib) spare COX-1 $\rightarrow$ reduced GI toxicity BUT increased cardiovascular risk (thrombosis) due to preserved thromboxane but reduced prostacyclin.

Why other options are wrong:

- Option A: Correct — COX-1 inhibition reduces PGE2, impairing mucosal defense (mucus, $HCO3^{-}$, blood flow) $\rightarrow$ ulceration.
- Option B: Direct topical acid injury is a MINOR and less important mechanism; enteric-coated NSAIDs still cause ulcers, confirming the primary systemic (COX-1) mechanism.
- Option C: Selective COX-2 inhibition INCREASES cardiovascular thrombotic risk (not the mechanism of GI injury); it actually REDUCES GI injury.
- Option D: NSAIDs do NOT inhibit H2 receptors; H2 receptor antagonism (ranitidine) is used to TREAT NSAID-induced ulcers, not cause them.

Final Answer: NSAID GI injury = COX-1 inhibition $\rightarrow$ decreased prostaglandins $\rightarrow$ impaired mucosal protection $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 91

Q92.

Solution

Concept — Tamoxifen as a SERM: Tamoxifen is a selective estrogen receptor modulator (SERM). It acts as a competitive antagonist at estrogen receptors in breast tissue (blocking estrogen's growth-promoting effects) while acting as a partial agonist in bone and endometrium. It is used in ER+/PR+ breast cancers.

Step 1 — Mechanism: Tamoxifen competes with estradiol for binding to estrogen receptors (ER) in breast cancer cells $\rightarrow$ blocks estrogen-driven transcription of growth genes (e.g., cyclin D1) $\rightarrow$ reduces tumor proliferation.

Step 2 — Adverse effects: Partial agonism in uterus $\rightarrow$ increased risk of endometrial cancer. Partial agonism in bone $\rightarrow$ prevents osteoporosis. Hot flashes are common. Thromboembolism risk is increased.

Why other options are wrong:

- Option A: Aromatase inhibition (preventing peripheral conversion of androgens to estrogens) is the mechanism of AROMATASE INHIBITORS (letrozole, anastrozole, exemestane) — NOT tamoxifen. AIs are used in postmenopausal women.
- Option B: Correct — tamoxifen is a competitive ER antagonist in breast tissue (SERM).
- Option C: Direct cytotoxic alkylation of DNA describes ALKYLATING AGENTS (cyclophosphamide, chlorambucil) — tamoxifen is NOT cytotoxic.
- Option D: Topoisomerase II inhibition causing DNA double-strand breaks describes ANTHRACYCLINES (doxorubicin) and EPIPODOPHYLLOTOXINS (etoposide).

Final Answer: Tamoxifen = competitive ER antagonist in breast tissue (SERM) $\Rightarrow \boxed{\text{B}}$

Answer: (B) Go Back to Q 92

Q93.

Solution

Concept — Short-acting benzodiazepines without active metabolites: In the elderly, accumulation of long-acting benzodiazepines and their active metabolites causes prolonged sedation, falls, cognitive impairment, and respiratory depression. Lorazepam, oxazepam, and temazepam (LOT) undergo Phase II glucuronidation only (no active metabolites) → safe in elderly, hepatic impairment.

Step 1 — Lorazepam properties: Lorazepam is a short-to-intermediate-acting benzodiazepine metabolized by direct conjugation (glucuronidation) → no active metabolites → no accumulation in elderly or those with hepatic disease.

Step 2 — Mnemonic LOT: Lorazepam, Oxazepam, Temazepam = safe in elderly, hepatic disease. "No active metabolites."

Why other options are wrong:

- Option A: Diazepam is LONG-ACTING with multiple active metabolites (desmethyldiazepam, oxazepam) with half-lives of 20–100 hours → accumulates in elderly → falls, sedation.
- Option B: Chlordiazepoxide is long-acting with several active metabolites (demoxepam, desmethylchlordiazepoxide, oxazepam) → NOT appropriate in elderly.
- Option C: Flurazepam is one of the LONGEST-ACTING benzodiazepines (active metabolite $t_{\frac{1}{2}}$ 47–100 hours) → most problematic in elderly.
- Option D: Correct — lorazepam is short-to-intermediate-acting with NO active metabolites → appropriate in elderly.

Final Answer: Short-acting benzodiazepine with no active metabolites for elderly = lorazepam ⇒ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 93

Q94.

Solution

Concept — Competitive inhibition on Lineweaver-Burk plot: In competitive inhibition, the inhibitor competes with substrate for the active site. More substrate can overcome the inhibitor → Vmax is UNCHANGED. However, the apparent affinity for substrate decreases (needs more substrate to achieve half-Vmax) → apparent Km INCREASES. On the Lineweaver-Burk plot: same y-intercept (1/Vmax), different x-intercept (-1/apparent Km).

Step 1 — Reading the graph: Both lines cross the y-axis at the same point (same 1/Vmax → same Vmax). The inhibitor line has a steeper slope and different x-intercept → higher apparent Km.

Step 2 — Comparison with non-competitive inhibition: Non-competitive inhibitor: decreased Vmax (lower y-intercept), unchanged Km (same x-intercept). Uncompetitive: both Vmax and Km decrease.

Why other options are wrong:

- Option A: Decreased Vmax, unchanged Km = non-competitive inhibition (uncompetitive inhibition decreases both).
- Option B: Decreased Vmax AND increased Km does not match a standard inhibition pattern precisely; competitive inhibition has UNCHANGED Vmax.
- Option C: Correct — competitive inhibition: unchanged Vmax (same y-intercept), increased apparent Km (different x-intercept).
- Option D: Unchanged Vmax, decreased Km describes uncompetitive inhibition (both Vmax and Km decrease proportionally — apparent Km decreases in some formulations, but the graph shows INCREASED Km here).

Final Answer: Competitive inhibition = unchanged Vmax, increased apparent Km ⇒ C

Answer: (C) Go Back to Q 94

Q95.

Solution

Concept — Staphylococcus aureus virulence factors: *S. aureus* produces numerous toxins. Panton-Valentine leukocidin (PVL) is a bicomponent pore-forming toxin that specifically targets and destroys polymorphonuclear leukocytes (neutrophils) and macrophages by forming pores in their membranes. PVL is associated with community-acquired MRSA (CA-MRSA) and severe skin/soft tissue infections, including necrotizing fasciitis and necrotizing pneumonia.

Step 1 — PVL mechanism: PVL consists of two protein components (LukS-PV + LukF-PV) that bind to receptors on leukocytes → assemble into a heptameric pore → leukocyte lysis → impaired host defense → severe infection.

Step 2 — Clinical association: Young, healthy individual + coagulase-positive staph + skin abscess after minor trauma = classic CA-MRSA with PVL.

Why other options are wrong:

- Option A: Correct — PVL destroys leukocytes; associated with CA-MRSA skin/soft tissue infections.
- Option B: TSST-1 causes toxic shock syndrome (fever, hypotension, diffuse erythematous rash, multi-organ failure) — NOT specifically leukocyte-destroying.
- Option C: Exfoliatin (epidermolytic toxin) causes staphylococcal scalded skin syndrome (SSSS) by cleaving desmoglein-1 in the epidermis — NOT a leukocytolytic toxin.
- Option D: Streptolysin O is produced by *Streptococcus pyogenes* (Group A Strep), NOT *S. aureus*; it is the basis of the ASO titer.

Final Answer: S. aureus toxin destroying leukocytes = Panton-Valentine leukocidin (PVL) $\Rightarrow$ A

Answer: (A) Go Back to Q 95

Q96.

Solution

Concept — Neonatal tetanus (Clostridium tetani): *Clostridium tetani* is a Gram-positive, spore-forming anaerobic bacillus. Its toxin, TETANOSPASMIN (TeNT), is a zinc-dependent metalloprotease that cleaves VAMP/synaptobrevin (a SNARE protein) at inhibitory interneurons (Renshaw cells) in the spinal cord and brainstem $\rightarrow$ prevents GABA and glycine release $\rightarrow$ disinhibition of motor neurons $\rightarrow$ tonic muscle rigidity and reflex spasms.

Step 1 — Neonatal tetanus: Contaminated umbilical cord cutting instrument introduces *C. tetani* spores. Toxin travels retroaxonally to the CNS. Presents on days 3–14 of life with trismus (lock-jaw), opisthotonus, and stimulus-sensitive spasms. Carries high mortality (>60%).

Step 2 — Organism identification: Gram-positive, spore-forming anaerobe with drum-stick (terminal) spores + neonatal presentation with spasms after unclean cord cutting = *C. tetani*.

Why other options are wrong:

- Option A: *C. perfringens* causes gas gangrene and food poisoning — NOT neonatal spasms; no connection to umbilical cord infection.
- Option B: *C. difficile* causes antibiotic-associated pseudomembranous colitis — NOT neonatal spasms.
- Option C: *Bacillus anthracis* causes anthrax (cutaneous, pulmonary, GI) — NOT neonatal tetanus; it is NOT a Clostridium species.

- Option D: Correct — *Clostridium tetani* via contaminated umbilical cord → neonatal tetanus with spasms.

Final Answer: Neonatal spasms after unclean cord cutting = *Clostridium tetani* ⇒ D

Answer: (D) Go Back to Q 96

Q97.

Solution

Concept — Vibrio cholerae cholera toxin: *Vibrio cholerae* grows on TCBS (Thiosulfate-Citrate-Bile salts-Sucrose) agar as yellow colonies (sucrose-fermenting). Its cholera toxin (CT) consists of a B subunit (pentamer binding GM1 ganglioside on enterocyte surface) and an A subunit. The A1 fragment of CT permanently activates Gs protein by ADP-ribosylation → constitutive adenylate cyclase activation → massive cAMP production → phosphorylation of CFTR → Cl^- secretion + Na^+ (water) loss → profuse rice-water diarrhea.

Step 1 — CT mechanism: B subunit binds GM1 → A1 subunit enters cell → ADP-ribosylates $Gs\alpha$ → locks Gs in active state → adenylate cyclase always ON → cAMP ↑↑ → PKA activation → CFTR phosphorylation → Cl^- efflux, Na^+ efflux, water loss → secretory diarrhea without mucosal invasion.

Step 2 — TCBS identification: Yellow colonies on TCBS = sucrose-fermenting = *V. cholerae* (O1 or O139).

Why other options are wrong:

- Option A: Inhibiting protein synthesis at the 60S subunit describes SHIGA TOXIN (produced by *Shigella dysenteriae* and STEC) — causes bloody diarrhea (dysentery), NOT rice-water diarrhea.
- Option B: Correct — permanent ADP-ribosylation of Gs → constitutive adenylate cyclase activation → massive cAMP → secretory diarrhea.
- Option C: Disrupting tight junctions describes certain enteropathogenic *E. coli* (EPEC) — different mechanism.
- Option D: Cytotoxin causing bloody diarrhea describes Shiga-toxin-producing *E. coli* (O157:H7) or *C. difficile* toxin B.

Final Answer: Cholera toxin = ADP-ribosylation of Gs → constitutive cAMP → secretory diarrhea ⇒ B

Answer: (B) Go Back to Q 97

Q98.

Solution

Concept — Enteric fever (typhoid): diagnosis timeline: *Salmonella typhi* causes typhoid fever. The classic diagnosis uses Widal test (agglutinins) and blood culture. BLOOD CULTURE is most sensitive in the FIRST WEEK (bacteremia phase) — up to 80% positive. Stool culture becomes positive in weeks 2–3 (intestinal phase). Urine culture positive in week 3. Widal test (serology) is positive from week 2 onward.

Step 1 — Stepwise fever presentation: Stepwise rising fever + relative bradycardia (pulse-temperature dissociation) + rose spots + splenomegaly = enteric fever due to *S. typhi*.

Step 2 — Blood culture timing: Week 1 = BLOOD CULTURE positive (>80%). Week 2–3 = stool + urine culture, Widal test. Blood culture remains the gold standard.

Why other options are wrong:

- Option A: *Shigella dysenteriae* causes bacillary dysentery (bloody mucoid stool, tenesmus) — NOT stepwise rising fever, rose spots, or splenomegaly.
- Option B: *Vibrio cholerae* causes profuse watery diarrhea without systemic fever — NOT enteric fever with splenomegaly.
- Option C: Correct — *Salmonella typhi* blood culture is most likely positive in the 1st week of enteric fever.
- Option D: *E. coli* O157:H7 causes hemorrhagic colitis and HUS — NOT typhoid with rose spots and splenomegaly.

Final Answer: Enteric fever (stepwise fever, rose spots, splenomegaly) week 1 = blood culture grows *Salmonella typhi* ⇒ C

Answer: (C) Go Back to Q 98

Q99.

Solution

Concept — HIV window period and early markers: After HIV infection, detectable markers appear in this order: (1) HIV RNA (viral load by PCR) — detectable within 7–10 days; (2) HIV p24 antigen — detectable within 14–18 days (end of 2nd week); (3) Anti-HIV IgM — weeks 3–4; (4) Anti-HIV IgG — weeks 4–12. The 4th-generation ELISA detects BOTH p24 antigen AND antibodies.

Step 1 — First serological marker (within 2 weeks): Among SEROLOGICAL markers (not PCR-based), HIV p24 antigen is the first to become detectable (weeks 2–3), before antibodies. It is shed directly by replicating virus.

Step 2 — Question framing: The question specifies "first identifiable serological marker within 2 weeks, during the window period of ANTIBODY testing" → HIV p24 antigen (detectable by 4th gen ELISA in the window period before antibodies).

Why other options are wrong:

- Option A: Correct — HIV p24 antigen is the first serological marker detectable (within 14–18 days of infection).
- Option B: Anti-HIV IgG antibodies appear LATE (weeks 4–12) — this is the basis of the "window period" (antibody-negative despite active infection).
- Option C: Anti-HIV IgM appears before IgG but AFTER p24 antigen (weeks 3–4).
- Option D: HIV RNA (viral load by PCR/NAAT) is the first MOLECULAR marker (days 7–10) but is NOT a serological marker detected by ELISA; the question specifies serological markers detected during the window period of antibody testing.

Final Answer: First identifiable serological marker within 2 weeks of HIV infection = HIV p24 antigen ⇒ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 99

Q100.

Solution

Concept — Hepatitis B carrier states: The inactive HBsAg carrier state ("inactive chronic HBV infection") is defined by: HBsAg positive >6 months, HBeAg negative, anti-HBe positive, very low or undetectable HBV DNA (<2,000 IU/mL), normal ALT/AST, minimal liver histology changes. It represents immune control of HBV with low viral replication.

Step 1 — Profile interpretation: HBsAg+ (infected), HBeAg- (low replication), anti-HBe+ (immune control of e antigen), HBV DNA 180 IU/mL (low), normal LFTs, asymptomatic = INACTIVE HBsAg carrier state.

Step 2 — Clinical significance: Inactive carriers have low risk of progression but should be monitored annually (LFTs, HBV DNA, AFP for HCC screening).

Why other options are wrong:

- Option A: Active hepatitis B with high infectivity = HBsAg+, HBeAg+, HIGH HBV DNA, elevated ALT — this patient has HBeAg- and very low HBV DNA.
- Option B: Acute hepatitis B = HBsAg+, anti-HBc IgM+, elevated ALT, symptomatic — this is not an acute presentation.
- Option C: Occult hepatitis B = HBsAg NEGATIVE but detectable HBV DNA in liver — this patient is HBsAg POSITIVE.
- Option D: Correct — HBsAg+, HBeAg-, anti-HBe+, low HBV DNA, normal LFTs = inactive HBsAg carrier state.

Final Answer: HBsAg+, HBeAg-, anti-HBe+, low HBV DNA, asymptomatic = inactive HBsAg carrier state $\Rightarrow$ D

Answer: (D) Go Back to Q 100

Q101.

Solution

Concept — Cryptococcal meningitis diagnosis: Cryptococcal meningitis (caused by *Cryptococcus neoformans*) is diagnosed by detecting the cryptococcal polysaccharide capsular antigen (CrAg) in CSF or serum using latex agglutination or lateral flow assay. Sensitivity and specificity exceed 95%. It is the BEST confirmatory test, faster and more sensitive than culture or India ink alone.

Step 1 — CrAg lateral flow assay: CrAg detects the polysaccharide glucuronoxylomannan (GXM) antigen shed by the fungal capsule. A titer >1:8 is strongly supportive. It is performed in minutes and does not require fresh CSF.

Step 2 — India ink vs CrAg: India ink stain (negative-staining capsule) is a rapid bedside test but has lower sensitivity (50–80%). CrAg is more sensitive and specific $\rightarrow$ BEST confirmatory test.

Why other options are wrong:

- Option A: Sabouraud dextrose agar culture at 37°C: definitive but slow (days–weeks); requires viable organisms; useful for speciation and antifungal sensitivity, NOT the BEST confirmatory test for rapid diagnosis.
- Option B: Correct — CSF cryptococcal antigen (CrAg) detection is the BEST confirmatory test (rapid, >95% sensitivity/specificity).
- Option C: Galactomannan antigen assay is specific for ASPERGILLUS (not Cryptococcus); used for invasive aspergillosis.

- Option D: KOH preparation is used for superficial fungal infections (dermatophytes, Candida in skin/nails) — NOT appropriate for meningitis diagnosis.

Final Answer: Best confirmatory test for cryptococcal meningitis = CSF cryptococcal antigen (CrAg) $\Rightarrow$ B

Answer: (B) Go Back to Q 101

Q102.

Solution

Concept — Scarlet fever and streptococcal pyrogenic exotoxin: Scarlet fever is caused by *Streptococcus pyogenes* (Group A Streptococcus, GAS) strains that produce streptococcal pyrogenic exotoxins (SPE, formerly called erythrogenic toxin). SPE A, B, C, and F act as superantigens, also causing the characteristic erythematous "sandpaper" rash by inducing widespread cytokine release and direct skin capillary effects.

Step 1 — SPE mechanism: SPE are superantigens that bypass normal antigen presentation $\rightarrow$ bind simultaneously to MHC class II (outside the antigen-binding groove) and T-cell receptor Vβ chain $\rightarrow$ massive non-specific T-cell activation $\rightarrow$ cytokine storm $\rightarrow$ systemic vasodilation $\rightarrow$ diffuse erythematous rash.

Step 2 — Clinical features of scarlet fever: "Strawberry tongue," pharyngeal exudates, sandpaper rash (blanches on pressure), Pastia's lines (flexural darkening), circumoral pallor.

Why other options are wrong:

- Option A: Leukocidin is produced by *Staphylococcus aureus* (PVL) — destroys leukocytes; it does NOT cause the scarlet fever rash.
- Option B: Streptolysin S is an oxygen-stable hemolysin of GAS causing beta-hemolysis on blood agar — NOT the toxin causing the erythematous rash.
- Option C: Correct — erythrogenic (pyrogenic) exotoxin (SPE) causes the characteristic rash of scarlet fever via superantigen mechanism.
- Option D: Streptokinase (fibrinolysin) dissolves fibrin clots, aiding bacterial spread — it does NOT cause skin rash.

Final Answer: Scarlet fever rash = streptococcal pyrogenic exotoxin (erythrogenic toxin) superantigen $\Rightarrow$ C

Answer: (C) Go Back to Q 102

Q103.

Solution

Concept — Plasmodium falciparum malaria: *P. falciparum* is the most virulent malaria species, causing fever every 48 hours (tertian) and banana-shaped (crescentic) gametocytes — pathognomonic on peripheral smear. It causes cerebral malaria through cytoadherence of parasitized RBCs to cerebral microvascular endothelium via PfEMP1 (encoded by var genes), causing vascular obstruction, inflammation, and cerebral hypoxia.

Step 1 — Identifying falciparum: Banana-shaped gametocytes (ONLY *P. falciparum* has this shape) + 48-hour fever + ring forms only in peripheral smear (mature forms sequestered in microvasculature) = *P. falciparum*.

Step 2 — Cerebral malaria mechanism: PfEMP1 on infected RBC surface → binds ICAM-1, CD36, PECAM-1 on cerebral endothelium → cytoadherence + rosetting → microvascular obstruction → cerebral hypoxia + inflammation → impaired consciousness, seizures, coma.

Why other options are wrong:

- Option A: Correct — *P. falciparum* causes cerebral malaria via cytoadherence of parasitized RBCs.
- Option B: *P. vivax* causes 48-hour (tertian) fever with relapse due to dormant liver hypnozoites — oval RBCs, Schüffner's dots; NO banana gametocytes.
- Option C: *P. malariae* causes quartan fever (72-hour cycle), quartan nephrotic syndrome — round gametocytes, band-form trophozoites; NO banana gametocytes.
- Option D: *P. ovale* causes mild tertian disease with relapses (hypnozoites) — oval, fimbriated RBCs; NO banana gametocytes.

Final Answer: Banana gametocytes + tertian fever = *P. falciparum* causing cerebral malaria ⇒ A

Answer: (A) Go Back to Q 103

Q104.

Solution

Concept — Amoebic liver abscess treatment: Amoebic liver abscess (ALA) is caused by *Entamoeba histolytica*. Treatment requires a TISSUE amebicide to kill trophozoites in the abscess (metronidazole — penetrates into abscess cavity) FOLLOWED by a LUMINAL amebicide (paromomycin or diloxanide furoate) to eradicate intestinal cysts and prevent relapse/transmission.

Step 1 — Metronidazole: Metronidazole 750 mg TDS for 5–10 days kills tissue trophozoites in the liver abscess (and intestinal wall). It does NOT adequately clear intestinal cyst carriage.

Step 2 — Luminal agent: Paromomycin (non-absorbable aminoglycoside) or diloxanide furoate (luminal amoebicide) eradicates intraluminal cysts $\rightarrow$ prevents reinfection and spread. Must be given AFTER completing metronidazole.

Why other options are wrong:

- Option A: Chloroquine alone is a second-line tissue amebicide; it does NOT clear intestinal cysts $\rightarrow$ incomplete treatment; not the standard DOC.
- Option B: Paromomycin alone (luminal agent) does NOT penetrate into the abscess cavity $\rightarrow$ will NOT treat established liver abscess.
- Option C: Albendazole is used for helminths (hookworm, tapeworm, hydatid, filaria) — NOT for amoebic liver abscess.
- Option D: Correct — metronidazole (tissue amebicide) followed by paromomycin or diloxanide furoate (luminal amebicide).

Final Answer: Amoebic liver abscess = metronidazole (tissue) followed by luminal amebicide $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 104

Q105.

Solution

Concept — Gell and Coombs hypersensitivity classification: Type IV (delayed-type hypersensitivity, DTH) is cell-mediated, antibody-independent. It is mediated by sensitized CD4+ Th1 cells (and CD8+ CTLs in contact dermatitis). Onset is 48–72 hours after antigen re-exposure. Examples: contact dermatitis (latex, nickel, poison ivy), tuberculin test, granulomatous diseases.

Step 1 — Contact dermatitis to latex: Latex allergy can be Type I (immediate

IgE-mediated urticaria within minutes) OR Type IV (delayed eczematous/vesicular rash 48–72 hours after contact). The question specifically describes a 48-HOUR delay and VESICULAR rash = Type IV (contact dermatitis).

Step 2 — Mechanism of Type IV: Previous sensitization → memory T cells formed. On re-exposure → Th1 cells recognize antigen-hapten-MHC II on APCs → release IFN-γ, TNF-α → macrophage activation → tissue damage → eczematous vesicular rash.

Why other options are wrong:

- Option A: Type I (IgE-mediated) = IMMEDIATE (within 30 min), urticaria, angioedema, anaphylaxis — onset is minutes, NOT 48 hours.
- Option B: Type II (cytotoxic) = IgG/IgM + complement against cell-surface antigens → hemolytic anemia, myasthenia gravis — NOT skin vesicular rash.
- Option C: Correct — Type IV (delayed-type hypersensitivity) 48 hours after latex contact = contact dermatitis.
- Option D: Type III (immune complex) = serum sickness, SLE, post-streptococcal GN — 1–3 weeks after exposure, NOT 48 hours after topical contact.

Final Answer: 48-hour vesicular rash after latex exposure = Type IV (delayed-type) hypersensitivity ⇒ C

Answer: (C) Go Back to Q 105

Q106.

Solution

Concept — Thrombocytopenia in dengue fever: Dengue fever causes thrombocytopenia through multiple mechanisms: (1) Direct bone marrow suppression by dengue virus → reduced platelet production; (2) Immune-mediated peripheral platelet destruction by anti-NS1 antibodies cross-reacting with platelets (molecular mimicry); (3) Accelerated platelet destruction by virus-infected monocytes/macrophages in the spleen. Both bone marrow suppression and immune-mediated destruction contribute.

Step 1 — Pathophysiology: Dengue virus infects hematopoietic progenitor cells → suppresses megakaryocyte maturation → reduced platelet production. Simultaneously, antibodies (anti-NS1, anti-prM) cross-react with platelet surface antigens → platelet phagocytosis by macrophages.

Step 2 — Clinical severity: Platelet count <20,000 increases hemorrhagic risk;

severe dengue (dengue hemorrhagic fever) has plasma leakage (capillary permeability) as the hallmark, NOT primarily thrombocytopenia alone.

Why other options are wrong:

- Option A: DIC does occur in severe dengue but is NOT the PRIMARY mechanism of thrombocytopenia in typical dengue; most dengue thrombocytopenia occurs WITHOUT overt DIC.
- Option B: Correct — combined bone marrow suppression + immune-mediated platelet destruction by dengue virus/immune mechanisms.
- Option C: Hypersplenism from dengue hepatitis contributes marginally but is NOT the MOST important mechanism.
- Option D: Anti-platelet IgG autoantibodies contribute but this is only ONE component; "only" makes this incomplete as the primary mechanism.

Final Answer: Dengue thrombocytopenia = bone marrow suppression + immune-mediated platelet destruction $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 106

Q107.

Solution

Concept — Gram-negative outer membrane and LPS: Gram-negative bacteria have a UNIQUE outer membrane (absent in Gram-positive bacteria) containing lipopolysaccharide (LPS), also called endotoxin. LPS consists of three parts: (1) Lipid A (toxic, causes septic shock by activating TLR4/MD-2 complex on macrophages $\rightarrow$ TNF-α, IL-1, IL-6 release), (2) Core polysaccharide, (3) O-antigen (species-specific, basis of serotyping).

Step 1 — LPS uniqueness: The outer membrane with LPS is unique to Gram-negative bacteria. Gram-positive bacteria have only a thick peptidoglycan layer and teichoic acids — NO outer membrane, NO LPS.

Step 2 — Clinical significance: LPS (endotoxin) is responsible for fever, hypotension, and septic shock in Gram-negative bacteremia. The Limulus amebocyte lysate (LAL) test detects endotoxin in pharmaceutical products and CSF.

Why other options are wrong:

- Option A: Teichoic acid is present in Gram-POSITIVE bacteria (wall teichoic acid) and is ABSENT in Gram-negatives (lipoteichoic acid is NOT in the outer membrane).

- Option B: Peptidoglycan is present in BOTH Gram-positive (thick) and Gram-negative (thin) bacteria — NOT unique to Gram-negative.
- Option C: Correct — LPS (endotoxin) is a unique component of the Gram-negative outer membrane, absent in Gram-positive bacteria.
- Option D: N-acetylmuramic acid is a component of PEPTIDOGLYCAN found in BOTH Gram-positive and Gram-negative bacteria.

Final Answer: Unique to Gram-negative outer membrane = lipopolysaccharide (LPS/endotoxin) $\Rightarrow$ C

Answer: (C) Go Back to Q 107

Q108.

Solution

Concept — Hookworm-induced anemia mechanism: Hookworm (*Ancylostoma duodenale* and *Necator americanus*) causes iron deficiency anemia primarily through CHRONIC INTESTINAL BLOOD LOSS. Adult hookworms attach to intestinal villi using buccal teeth/cutting plates and ingest blood directly. Each *A. duodenale* worm consumes approximately 0.2 mL/day of blood; heavy infections cause massive iron deficiency.

Step 1 — Blood loss mechanism: Adult worms: (1) attach firmly to intestinal mucosa, (2) suck blood actively, (3) also secrete anticoagulants (hookworm anticoagulant peptide) preventing clotting at attachment site $\rightarrow$ continuous bleeding even after worm detaches.

Step 2 — Additional iron loss: Migrating larvae cause tissue eosinophilia (loeffler's syndrome in lungs) and protein-losing enteropathy (hypoalbuminemia). Iron deficiency + blood loss = microcytic hypochromic anemia.

Why other options are wrong:

- Option A: Intravascular hemolysis describes malaria and hemolytic anemias — hookworm does NOT infect RBCs.
- Option B: B12 deficiency with ileal colonization describes FISH TAPEWORM (*Diphyllobothrium latum*), which competes for B12 in the ileum.
- Option C: Bone marrow suppression by helminthic toxins is NOT a recognized mechanism for hookworm anemia.
- Option D: Correct — chronic intestinal blood loss from worm feeding on intestinal villi and direct blood sucking, compounded by secreted anticoagulants.

Final Answer: Hookworm anemia = chronic intestinal blood loss from direct blood sucking + anticoagulant secretion $\Rightarrow$ D

Answer: (D) Go Back to Q 108

Q109.

Solution

Concept — Post-mortem changes and time of death estimation: Key post-mortem changes timeline: Rigor mortis begins at 2–3 hours, complete by 12 hours, starts resolving by 24–36 hours, complete by 48–72 hours. Early putrefaction (greenish discoloration of right iliac fossa) begins at 24–36 hours. Presence of WELL-ESTABLISHED rigor + early putrefaction (greenish discoloration) indicates death 12–24 hours before discovery.

Step 1 — Rigor mortis timing: "Well-established rigor mortis in ALL muscle groups" = maximum rigor = approximately 12 hours post-mortem.

Step 2 — Putrefaction timing: "Greenish discoloration of right iliac fossa" = beginning of putrefaction (due to H_2S from gut bacteria in the cecum/ascending colon) = typically starts at 24–36 hours post-mortem in temperate conditions. Combined with established rigor $\rightarrow$ death approximately 12–24 hours before discovery.

Why other options are wrong:

- Option A: Correct — 12–24 hours: established rigor + early putrefaction (greenish RIF) fits this window.
- Option B: 2–4 hours: only early rigor beginning (jaw/neck only), NO putrefaction $\rightarrow$ inconsistent with complete rigor and greenish discoloration.
- Option C: 36–48 hours: rigor would be RESOLVING (secondary relaxation) by this time, and putrefaction would be more extensive (gas formation, marbling).
- Option D: Over 5 days: advanced putrefaction with bloating, skin slippage, adipocere/mummification — NOT early greenish discoloration.

Final Answer: Established rigor + early greenish RIF discoloration = death 12–24 hours before discovery $\Rightarrow$ A

Answer: (A) Go Back to Q 109

Q110.

Solution

Concept — Suicidal hanging vs. homicidal strangulation: In suicidal hanging, the ligature mark is typically: oblique, incomplete (interrupted at the suspension point), situated ABOVE the thyroid cartilage, with evidence of constriction from all sides except the suspension point. Death in suicidal hanging (when not instantaneous) is due to venous obstruction (jugular veins occluded first) $\rightarrow$ cerebral hypoxia, combined with airway compression and carotid sinus pressure $\rightarrow$ asphyxia.

Step 1 — Features of suicidal hanging: Oblique mark + incomplete (not encircling fully) + above thyroid cartilage + Tardieu petechiae (conjunctival hemorrhages from venous congestion) + NO vertebral fracture (partial hanging) = suicidal hanging with asphyxial death.

Step 2 — Cause of death: Venous obstruction $\rightarrow$ raised intracranial pressure + cerebral hypoxia + airway compression $\rightarrow$ asphyxia.

Why other options are wrong:

- Option A: Judicial hanging has a fracture-dislocation of C2 (”hangman’s fracture”) with immediate death from cervical cord transection — judicial hanging is homicidal/judicial, with a long drop and a specific knot under the chin.
- Option B: Correct — suicidal hanging with asphyxial death (venous obstruction + airway compression), oblique incomplete ligature mark.
- Option C: Manual strangulation shows BILATERAL fingernail/fingertip marks on neck (petechiae), no ligature mark — NOT this case.
- Option D: Garroting (ligature strangulation) has a HORIZONTAL, COMPLETE (encircling) mark around the neck — NOT oblique or incomplete.

Final Answer: Oblique incomplete ligature mark above thyroid cartilage = suicidal hanging (asphyxia) $\Rightarrow$ B

Answer: (B) Go Back to Q 110

Q111.

Solution

Concept — Atropine in organophosphate poisoning: Organophosphates irreversibly inhibit acetylcholinesterase (AChE) → ACh accumulates at all cholinergic synapses. Atropine is a competitive antagonist at muscarinic acetylcholine receptors (M1, M2, M3) → blocks MUSCARINIC features (SLUDGE/DUMBELS: salivation, lacrimation, urination, defecation, GI cramps, emesis/bradycardia, bronchospasm, miosis). Atropine must be given in LARGE, REPEATED DOSES titrated to drying of secretions and heart rate normalization.

Step 1 — Atropine for muscarinic features: 2–4 mg IV every 5–10 minutes (repeated) until secretions dry, bronchospasm resolves, and heart rate normalizes. Total doses of 20–100 mg may be needed in severe poisoning. Crosses BBB → addresses central muscarinic features (confusion, coma).

Step 2 — Pralidoxime for nicotinic features: Pralidoxime (PAM) reactivates AChE at the neuromuscular junction → addresses NICOTINIC features (muscle fasciculations, weakness, paralysis). Must be given within 24–48 hours.

Why other options are wrong:

- Option A: Pralidoxime alone does NOT address muscarinic features; it reactivates AChE at the NMJ. It must be used WITH atropine, not alone.
- Option B: Diazepam controls seizures but does NOT address muscarinic features (salivation, bronchospasm, bradycardia).
- Option C: Correct — atropine in large repeated doses titrated to drying of secretions addresses central and peripheral muscarinic features.
- Option D: N-acetylcysteine (NAC) is the antidote for PARACETAMOL (acetaminophen) overdose — has no role in OP poisoning.

Final Answer: Organophosphate muscarinic features antidote = atropine (large repeated doses) ⇒ C

Answer: (C) Go Back to Q 111

Q112.

Solution

Concept — Classification of burns and vital reaction: Burns are classified by depth: (1) First degree (superficial/erythema): epidermis only, redness, no blistering. (2) Second degree (partial thickness): epidermis + part of dermis, blistering, moist red painful skin beneath blister. (3) Third degree (full thickness): entire dermis and epidermis destroyed, white/charred, insensate, no blistering. Vital reaction (inflammatory response) confirms antemortem occurrence.

Step 1 — Identifying burn degree: Destroyed epidermis AND PART of dermis + blistering + red moist skin beneath blister + vital reaction = SECOND DEGREE (partial thickness) burn.

Step 2 — Vital reaction significance: Presence of vital reaction (erythema, edema, neutrophil infiltration, leukocyte margination) confirms the burn occurred ANTEMORTEM. Postmortem burns lack vital reaction; artefactual blisters in postmortem burns contain gas or blood, not serous fluid.

Why other options are wrong:

- Option A: Correct — second-degree (partial thickness) antemortem burn: blistering + red moist base + vital reaction.
- Option B: First-degree burn = epidermis ONLY affected (redness/erythema, no blistering); this case has blistering and dermal involvement.
- Option C: Third-degree burn = FULL thickness (entire dermis destroyed); skin is white, leathery, or charred; NO blistering (all layers destroyed); painless (nerve endings destroyed).
- Option D: Charring (postmortem artefact) has no vital reaction and occurs after death — this case HAS vital reaction confirming antemortem occurrence.

Final Answer: Blistering + moist red base + vital reaction = second-degree (partial thickness) antemortem burn $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 112

Q113.

Solution

Concept — Carbon monoxide (CO) toxicity mechanisms: CO kills through a DUAL mechanism: (1) CO binds to hemoglobin with 240 times greater affinity than O_2 $\rightarrow$ forms carboxyhemoglobin (COHb) $\rightarrow$ reduced O_2 carrying capacity + left shift of HbO_2 dissociation curve (CO makes remaining Hb grip O_2 more tightly, further reducing O_2 delivery to tissues); (2) CO binds to mitochondrial cytochrome c oxidase (Complex IV) $\rightarrow$ inhibits cellular respiration $\rightarrow$ histotoxic hypoxia. Together: histemic + histotoxic hypoxia.

Step 1 — Cherry-red color: COHb is cherry-red (unlike dark deoxygenated Hb), causing the classic cherry-red discoloration of skin, blood, and organs at autopsy.

Step 2 — Dual mechanism importance: Even after COHb clears (with 100% O_2 treatment), cytochrome c oxidase inhibition persists, causing ongoing cellular hypoxia — hence delayed neurological sequelae in CO poisoning.

Why other options are wrong:

- Option A: MetHb formation is caused by OXIDIZING AGENTS (nitrites, dapsone, primaquine) — NOT CO. CO forms COHb, not MetHb.
- Option B: Direct cardiac toxicity by binding to cardiac myosin does occur to a minor extent (CO binds cardiac myoglobin causing contractile dysfunction), but this is NOT the primary killing mechanism.
- Option C: Cytochrome c oxidase inhibition AND carboxymyoglobin formation AND left-shift of HbO2 curve are all correct components, but "only" makes this option incomplete — it omits the high-affinity Hb binding component.
- Option D: Correct — CO binds Hb with 240$\times$ affinity (COHb, reduces O_2 carrying capacity + left-shifts O_2 dissociation curve) AND inhibits mitochondrial cytochrome c oxidase $\rightarrow$ histemic + histotoxic hypoxia.

Final Answer: CO toxicity = 240$\times$ affinity binding to Hb (COHb) + inhibition of cytochrome c oxidase $\rightarrow$ dual hypoxia $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 113

Q114.

Solution

Concept — Emergency consent and implied consent doctrine: In medical ethics and law, INFORMED CONSENT is required before any medical procedure. However, in a TRUE EMERGENCY where the patient is incapacitated and delay in treatment would cause death or grievous harm, the DOCTRINE OF IMPLIED CONSENT applies — the law presumes the patient would consent to life-saving treatment if able. This is upheld in Indian medical law and global medical ethics (MCI/NMC guidelines).

Step 1 — Doctrine of implied consent: When a patient is unconscious/unable to consent AND the situation is a life-threatening emergency AND no legal guardian is immediately available $\rightarrow$ the physician may proceed with emergency treatment under implied consent. The physician should document the emergency nature and notify next-of-kin as soon as possible.

Step 2 — Indian legal context: Under Indian law and NMC (National Medical Commission) guidelines, emergency treatment without consent is permissible to save life. Waiting for relatives or court orders would violate the duty to save life.

Why other options are wrong:

- Option A: Waiting for written next-of-kin consent in a life-threatening emergency would constitute negligence — patient may die during the delay.
- Option B: Correct — emergency surgery under the doctrine of implied consent is legally and ethically appropriate to save life.
- Option C: Police clearance is required for medico-legal cases (documentation/reporting) but does NOT delay emergency life-saving treatment.
- Option D: Magistrate consent is NOT required for any medical procedure; this is incorrect and would cause fatal delays.

Final Answer: Emergency surgery in unconscious patient without relatives = proceed under implied consent doctrine $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 114

Q115.

Solution

Concept — Sensitivity and specificity calculations: From the 2×2 table: TP=90, FP=20, FN=10, TN=180. Total disease positive = 100, Total disease negative = 200.

Step 1 — Sensitivity calculation: Sensitivity = TP/(TP+FN) = 90/(90+10) = 90/100 = **90%**. Sensitivity measures how well the test detects true disease cases ("detects the sick").

Step 2 — Specificity calculation: Specificity = TN/(TN+FP) = 180/(180+20) = 180/200 = **90%**. Specificity measures how well the test correctly identifies non-disease cases ("protects the healthy").

Why other options are wrong:

- Option A: Sensitivity 81.8% = PPV (TP/TP+FP = 90/110 = 81.8%); Specificity 94.7% = NPV (TN/TN+FN = 180/190 = 94.7%) — these are predictive values, not sensitivity/specificity.
- Option B: The values 94.7% and 81.8% are reversed PPV and NPV — incorrect assignment as sensitivity/specificity.
- Option C: Correct — Sensitivity = 90%, Specificity = 90%.
- Option D: Sensitivity 80%, Specificity 75% — incorrect calculations; these do not match the given data.

Final Answer: Sensitivity = 90/(90+10) = 90%; Specificity = 180/(180+20) = 90% ⇒ C

Answer: (C) Go Back to Q 115

Q116.

Solution

Concept — Relative Risk (RR) calculation in cohort studies: Relative Risk (RR) = (Risk of disease in exposed group) / (Risk of disease in unexposed group) = (Incidence in exposed) / (Incidence in unexposed).

Step 1 — Calculate incidence rates:

- Incidence in smokers (exposed) = 400/10,000 = 0.04 (4%)
- Incidence in non-smokers (unexposed) = 80/10,000 = 0.008 (0.8%)

Step 2 — Calculate RR: RR = 0.04/0.008 = **5.0**. This means smokers have a

5-fold higher risk of developing lung cancer compared to non-smokers. An RR >1 indicates increased risk; RR = 1 means no association; RR <1 means protective effect.

Why other options are wrong:

- Option A: RR = 2.0 would imply only 2-fold risk; this would occur if smoker risk were 1.6% (160 cases out of 10,000) → not consistent with given data.
- Option B: Correct — RR = 400/10,000 ÷ 80/10,000 = 4% / 0.8% = 5.0.
- Option C: RR = 8.0 would require incidence in smokers = 6.4% (640/10,000) — not consistent with 400/10,000.
- Option D: RR = 10.0 would require incidence in non-smokers = 0.4% (40/10,000) — not consistent with 80/10,000.

Final Answer: RR = 400/10,000 ÷ 80/10,000 = 5.0 ⇒ B

Answer: (B) Go Back to Q 116

Q117.

Solution

Concept — National Immunization Schedule (NIS) India at 6 weeks: At 6 weeks of age under India's NIS, the following vaccines are given: OPV (dose 1), Pentavalent 1 (DPT + Hep B + Hib), Rotavirus vaccine (dose 1), and fIPV (fractional inactivated polio vaccine dose 1 — at 6 weeks in some states). MMR is given at 9–12 months (first dose) and 16–24 months (second dose) — NOT at 6 weeks.

Step 1 — 6-week schedule: At birth: BCG, OPV-0, Hep B-0. At 6 weeks: OPV-1, Pentavalent-1 (DPT+HepB+Hib), Rotavirus-1, fIPV-1. MMR is NOT given at 6 weeks.

Step 2 — MMR timing: First dose of MMR (or MR/measles) is given at 9 months of age. This is because maternal antibodies interfere with the measles vaccine before 9 months.

Why other options are wrong:

- Option A: OPV IS given at 6 weeks (dose 1) — this IS administered at 6 weeks.
- Option B: Pentavalent vaccine IS given at 6 weeks (dose 1) — this IS administered at 6 weeks.
- Option C: Rotavirus vaccine IS given at 6 weeks (dose 1) — this IS adminis-

tered at 6 weeks.

- Option D: Correct — MMR is NOT given at 6 weeks; it is given at 9–12 months (first dose).

Final Answer: Vaccine NOT given at 6 weeks = MMR (given at 9–12 months) $\Rightarrow$ D

Answer: (D) Go Back to Q 117

Q118.

Solution

Concept — Hepatitis A as a water-borne self-limiting disease: Hepatitis A virus (HAV) is the classic water-borne/food-borne hepatitis. Key features: incubation period 15–45 days (average 28 days), feco-oral transmission, self-limiting (NEVER causes chronic infection), jaundice resolves in 4–6 weeks, NO carrier state, NO chronic hepatitis. Outbreaks typically follow contaminated water supply.

Step 1 — Distinguishing features: Self-limiting + NO chronic infection + 15–45 day incubation + post-flood outbreak = Hepatitis A. Hepatitis E also fits the epidemiology but has a longer incubation and is especially dangerous in pregnancy.

Step 2 — Hepatitis E vs A: Both are enteric (water-borne), self-limiting, and no chronic carrier state. However, HAV has a shorter incubation (15–45 days) vs HEV (15–60 days). HAV is MORE COMMON in mass outbreaks in all populations. The question says "most likely" $\rightarrow$ HAV.

Why other options are wrong:

- Option A: Hepatitis B is parenterally transmitted (blood, sexual, perinatal) — NOT water-borne; causes chronic infection in 5–10% of adults.
- Option B: Correct — Hepatitis A: water-borne, incubation 15–45 days, self-limiting, NO chronic infection.
- Option C: Hepatitis C is parenterally transmitted (blood, IV drug use) — NOT water-borne; causes chronic infection in 70–80%.
- Option D: Hepatitis E is also water-borne and self-limiting but is best known for its high mortality in pregnant women (fulminant hepatic failure) — HAV is the most common cause of epidemic water-borne jaundice in all populations.

Final Answer: Post-flood self-limiting jaundice (no chronic infection, 15–45 day incubation) = Hepatitis A virus $\Rightarrow$ B

Answer: (B) Go Back to Q 118

Q119.

Solution

Concept — Malaria vector control: source reduction: "Source reduction" in malaria control refers specifically to ELIMINATION OF MOSQUITO BREEDING SITES — removing, draining, or modifying standing water collections (pools, swamps, ponds, containers) where female *Anopheles* mosquitoes lay eggs. This is an environmental management strategy targeting the larval/pupal stages, interrupting the mosquito life cycle before adult mosquitoes emerge.

Step 1 — Definition of source reduction: Eliminating breeding habitats: draining stagnant water, filling ditches, clearing vegetation, removing water-holding containers. This prevents *Anopheles* larval development $\rightarrow$ reduces vector density.

Step 2 — Distinguishing from other strategies: IRS (indoor residual spraying) = insecticidal application to indoor walls targeting adult mosquitoes. LLINs = personal protection against adult mosquitoes. Bti = biological control (larviciding). All differ from source reduction.

Why other options are wrong:

- Option A: IRS with DDT/pyrethroids = ADULT mosquito control (insecticidal killing) — NOT source reduction.
- Option B: LLINs = personal barrier protection against adult mosquitoes biting at night — NOT source reduction.
- Option C: Correct — source reduction = elimination of mosquito breeding sites (draining stagnant water, filling swamps).
- Option D: Biological control using Bti = larviciding (killing larvae with bacterial toxin) — this targets larvae but by introducing a biological agent, NOT by eliminating the breeding site itself.

Final Answer: Source reduction = elimination of mosquito breeding sites (draining stagnant water) $\Rightarrow$ C

Answer: (C) Go Back to Q 119

Q120.

Solution

Concept — Infant Mortality Rate (IMR) as a sensitive health indicator: The Infant Mortality Rate (IMR) is defined as the number of deaths of children under 1 year of age per 1,000 live births in a given year. It is universally recognized as the MOST SENSITIVE and COMPREHENSIVE indicator of the health status of a community because it reflects: (1) maternal health and nutrition during pregnancy, (2) quality of antenatal care, (3) delivery care and neonatal care, (4) environmental hygiene and sanitation, (5) nutritional status, and (6) socioeconomic development.

Step 1 — Why IMR is most sensitive: Infant survival depends on numerous interacting health system and social determinants — making it the most sensitive barometer of community health and health system performance.

Step 2 — IMR vs MMR: IMR covers the broader population (all infants). MMR reflects only maternal risk during pregnancy/delivery. IMR is considered more sensitive for overall community health status.

Why other options are wrong:

- Option A: Correct — IMR is the most sensitive indicator of community health status and quality of maternal and pediatric care.
- Option B: Crude birth rate (CBR) reflects fertility, not health status; it is a demographic measure, not a health outcome indicator.
- Option C: Total fertility rate (TFR) reflects the average number of children per woman — a fertility/demographic measure, NOT a health status indicator.
- Option D: Maternal mortality ratio (MMR) reflects maternal health specifically but is less comprehensive than IMR as a community health indicator; however, both are important. IMR remains the most sensitive overall.

Final Answer: Most sensitive indicator of community health status = Infant Mortality Rate (IMR) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 120

Q121.

Solution

Concept — Secondary Attack Rate (SAR): SAR measures the proportion of susceptible contacts who develop the disease after exposure to a primary case. It is calculated as: Number of secondary cases among contacts / Total susceptible contacts $\times$ 100.

Step 1 — Calculation: Secondary cases among family contacts = 5. Total family contacts = 50. SAR = (5/50) $\times$ 100 = **10%**.

Why other options are wrong:

- Option A: 10% — **CORRECT**. (5/50 $\times$ 100 = 10%)
- Option B: 25% would require 12.5 secondary cases out of 50 contacts — incorrect calculation.
- Option C: 4.2% would be 5/120 — this incorrectly uses the original exposed population, not the contact group.
- Option D: 16.7% would be 5/30 — incorrectly uses primary cases as denominator instead of family contacts.

Final Answer: SAR among family contacts = 5/50 $\times$ 100 = 10% $\Rightarrow$ A

Answer: (A) Go Back to Q 121

Q122.

Solution

Concept — Specificity: Specificity = TN / (TN + FP) $\times$ 100. It measures the ability of a test to correctly identify those WITHOUT the disease.

Step 1 — Extract values from the table: TN = 230, FP = 20. Total culture-negative = 230 + 20 = 250. Specificity = 230/250 $\times$ 100 = **92%**.

Why other options are wrong:

- Option A: 93.3% would be sensitivity (TP/(TP+FN) = 140/150 = 93.3%), not specificity.
- Option B: 92.0% — **CORRECT.** TN/(TN+FP) = 230/250 = 92%.
- Option C: 87.5% does not correspond to any correct formula applied to this data.
- Option D: 96.0% = 240/250 — incorrectly counts both TN and a partial FP.

Final Answer: Specificity = 230/250 = 92% $\Rightarrow$ B

Answer: (B) Go Back to Q 122

Q123.

Solution

Concept — Point Prevalence: Prevalence = All existing cases at a point in time / Total population at that time × 100. It differs from incidence, which counts only NEW cases over a time period.

Step 1 — Apply formula: Existing hypertension cases at the time of survey = 2,500. Total population = 50,000. Point prevalence = 2500/50000 × 100 = **5%**.

Why other options are wrong:

- Option A: 1% uses 500 (new cases = incidence numerator), not existing cases, so this is closer to incidence rate.
- Option B: 0.5% = 250/50,000 — no clinical basis for this figure.
- Option C: 5% — **CORRECT.** 2,500/50,000 = 5% (all existing cases/population).
- Option D: 2% = 1,000/50,000 — no corresponding clinical figure in the question.

Final Answer: Point prevalence = 2500/50000 = 5% ⇒ C

Answer: (C) Go Back to Q 123

Q124.

Solution

Concept — Relative Risk (RR) in a Cohort Study: RR = Incidence in exposed / Incidence in unexposed = (a/(a+b)) / (c/(c+d)).

Step 1 — Calculate: Smokers: 100/1000 = 0.10 (10%). Non-smokers: 20/1000 = 0.02 (2%). RR = 0.10/0.02 = **5.0**.

Why other options are wrong:

- Option A: 2.5 — would require smoker risk of 5%, not 10%.
- Option B: 3.0 — requires smoker risk 6%; incorrect.
- Option C: 4.0 — requires smoker risk 8%; incorrect.
- Option D: 5.0 — **CORRECT.** 0.10/0.02 = 5.0; smokers are 5 times more likely to develop lung cancer.

Final Answer: RR = 0.10/0.02 = 5.0 ⇒ D

Answer: (D) Go Back to Q 124

Q125.

Solution

Concept — Number Needed to Treat (NNT): NNT = 1 / Absolute Risk Reduction (ARR). ARR = Event rate in control – Event rate in treatment.

Step 1 — Calculate ARR and NNT: ARR = 12% – 8% = 4% = 0.04. NNT = 1/0.04 = **25**.

Why other options are wrong:

- Option A: 25 — **CORRECT.** NNT = 1/ARR = 1/0.04 = 25.
- Option B: 20 — would require ARR of 5% (i.e., 12%–7%), not the case here.
- Option C: 50 — would require ARR of 2%, not 4%.
- Option D: 100 — would require ARR of only 1%; grossly overestimates the NNT.

Final Answer: NNT = 1/(0.12–0.08) = 1/0.04 = 25 ⇒ A

Answer: (A) Go Back to Q 125

Q126.

Solution

Concept — Cold Chain for EPI Vaccines: Different vaccines have different cold chain requirements. Most vaccines (BCG, OPV, DPT, Measles, Hepatitis B) must be stored at +2°C to +8°C in ice-lined refrigerators (ILR) at the district level. OPV must be stored at −15°C to −25°C at central/state level.

Step 1 — Apply knowledge: The ILR at district/PHC level stores vaccines at **+2°C to +8°C**. This is the standard temperature for most childhood vaccines.

Why other options are wrong:

- Option A: +2°C to +4°C — too narrow; standard is up to +8°C.
- Option B: +2°C to +8°C — **CORRECT.** Standard ILR temperature for most EPI vaccines at district/PHC level.
- Option C: −15°C to −25°C — this is for OPV storage at central/state level (deep freezer), not for the ILR.

- Option D: 0°C to +2°C — this risks freezing vaccines like DPT, Hep B, which are damaged by freezing.

Final Answer: ILR storage temperature = +2°C to +8°C ⇒ B

Answer: (B) Go Back to Q 126

Q127.

Solution

Concept — Fluoride in Drinking Water: WHO permissible limit for fluoride is 1.5 mg/L. Indian BIS standard is also 1.0–1.5 mg/L. Above 1.5 mg/L causes dental fluorosis; above 3 mg/L causes skeletal fluorosis.

Step 1 — Apply standard: WHO upper permissible limit = **1.5 mg/L**. This child has 3.5 mg/L, which is well above the safe limit, explaining both dental and skeletal fluorosis.

Why other options are wrong:

- Option A: 0.5 mg/L — the optimal fluoride level is 0.5–1.0 mg/L; 0.5 is the lower optimal, not permissible limit.
- Option B: 0.8 mg/L — some guidelines use 0.5–1.2 mg/L as optimal; 0.8 is within this range but not the permissible limit.
- Option C: 1.0 mg/L — this is the optimal level for dental caries prevention, not the maximum permissible limit.
- Option D: 1.5 mg/L — **CORRECT.** WHO and AERB permissible limit = 1.5 mg/L.

Final Answer: Permissible fluoride limit = 1.5 mg/L (WHO) ⇒ D

Answer: (D) Go Back to Q 127

Q128.

Solution

Concept — Malaria Control / Sporozoite Rate / IRS: Sporozoite rate = infected mosquitoes/total × 100 = 10/200 = 5%. *Anopheles culicifacies* is an endophilic (indoor-resting) species; the **most effective control** is Indoor Residual Spraying (IRS).

Step 1 — Calculate sporozoite rate: 10/200 × 100 = 5%.

Step 2 — Vector control: *An. culicifacies* rests indoors after feeding ⇒ IRS with DDT or synthetic pyrethroids is most appropriate.

Why other options are wrong:

- Option A: Sporozoite rate 5% correct but larviciding with temephos targets larvae in water, not indoor-resting adults; less effective for this species.
- Option B: Biological control with Gambusia is for larval habitats (ponds, slow-moving water) — useful but not primary for indoor-resting species.
- Option C: 10% is the wrong sporozoite rate (10/200 = 5%, not 10%). Aerial spraying is expensive and non-targeted.
- Option D: 5%; IRS — **CORRECT.** Sporozoite rate is 5%, and IRS is the recommended control for endophilic *An. culicifacies*.

Final Answer: 5%; IRS with DDT/pyrethroid ⇒ D

Answer: (D) Go Back to Q 128

Q129.

Solution

Concept — NTEP Category I DOTS Regimen: Under the National TB Elimination Programme (NTEP, formerly RNTCP), the first-line regimen for new smear-positive PTB consists of an Intensive Phase of **2 months of HRZE** (Isoniazid + Rifampicin + Pyrazinamide + Ethambutol) followed by a Continuation Phase of 4 months of HR.

Step 1 — Identify intensive phase: 2 months HRZE (H=Isoniazid, R=Rifampicin, Z=Pyrazinamide, E=Ethambutol) = **Option A.**

Why other options are wrong:

- Option A: 2 months HRZE — **CORRECT.** Standard intensive phase for Category I DOTS under NTEP.
- Option B: 3 months HRZ — this is not the current NTEP regimen; older regimens used 3HRZE for retreatment.
- Option C: 1 month HRZES — HRZES (with Streptomycin) was used in Category II retreatment; no longer in routine use under new NTEP guidelines.
- Option D: 2 months HR only — this is the continuation phase, not the intensive phase.

Final Answer: Intensive phase = 2 months HRZE ⇒ A

Answer: (A) Go Back to Q 129

Q130.

Solution

Concept — Infant Mortality Rate (IMR): IMR = Deaths of children under 1 year of age (in a given year) / Total live births in the same year $\times$ 1000. It is expressed per 1000 live births.

Step 1 — Correct formula: Numerator = deaths under 1 year. Denominator = total live births (NOT total population). Multiplier = 1000.

Why other options are wrong:

- Option A: Deaths <1 yr / Total population — this confuses IMR with crude mortality rate; denominator is wrong.
- Option B: Deaths <1 yr / Total live births $\times$ 1000 — **CORRECT.** This is the standard IMR formula.
- Option C: Deaths <5 yr / Total live births — this is the Under-5 Mortality Rate (U5MR), not IMR.
- Option D: Deaths <28 days / Total live births — this is the Neonatal Mortality Rate (NMR), not IMR.

Final Answer: IMR = Deaths under 1 year / Live births $\times$ 1000 $\Rightarrow$ B

Answer: (B) Go Back to Q 130

Q131.

Solution

Concept — Inferior STEMI Management: ST elevation in II, III, aVF = inferior STEMI (RCA territory). Reciprocal changes in I and aVL confirm. Primary PCI is the gold standard if achievable within 90–120 minutes of door-to-balloon time. If PCI is not available within 120 minutes, thrombolysis should be administered within 30 minutes of hospital arrival.

Step 1 — Best management: The most comprehensive, correct option is dual antiplatelet (Aspirin + Clopidogrel/Ticagrelor) + anticoagulation (IV heparin/LMWH) + primary PCI within 90 minutes. If door-to-balloon >90–120 min, thrombolysis is the fallback.

Why other options are wrong:

- Option A: Aspirin + Clopidogrel + IV heparin + primary PCI within 90 min — **CORRECT**; captures the class I recommendation.
- Option B: IV morphine + furosemide + thrombolysis with streptokinase

— furosemide is not indicated in STEMI without flash pulmonary oedema; streptokinase is a last resort.
- Option C: Partially correct (acknowledges thrombolysis if PCI unavailable after 120 min), but primary PCI target is 90 min for non-transfer and 120 min for transfer; Option C states "120 min" as the primary target incorrectly.
- Option D: Conservative management only — incorrect; STEMI requires reperfusion; beta-blocker should be deferred until haemodynamically stable.

Final Answer: Aspirin + Clopidogrel + IV heparin + primary PCI within 90 min $\Rightarrow$ C

Answer: (C) Go Back to Q 131

Q132.

Solution

Concept — Heart Failure with Reduced EF (HFrEF) / Sacubitril-Valsartan: The PARADIGM-HF trial (2014) demonstrated that sacubitril/valsartan (ARNi) reduces mortality and hospitalisation by 20% compared to enalapril in patients with HFrEF (EF $\leq$40%). It is now the preferred RAAS agent in HFrEF over ACE inhibitors if tolerated.

Step 1 — Identify correct drug: EF = 30%, symptomatic HFrEF, BNP elevated. Sacubitril/valsartan has the strongest evidence for mortality reduction.

Why other options are wrong:

- Option A: Digoxin reduces hospitalisations but does **not** reduce mortality (DIG trial).
- Option B: Furosemide relieves symptoms (oedema, dyspnoea) but does not reduce mortality.
- Option C: Nitrates reduce preload and relieve dyspnoea but have no mortality benefit in HFrEF.
- Option D: Sacubitril/Valsartan (ARNi) — **CORRECT.** First-line therapy in HFrEF with demonstrated mortality reduction per ESC/AHA guidelines.

Final Answer: Sacubitril/Valsartan (ARNi) — best mortality-reducing drug in HFrEF $\Rightarrow$ D

Answer: (D) Go Back to Q 132

Q133.

Solution

Concept — Atrial Fibrillation / Rate Control / Anticoagulation: AF ECG shows absent P waves, irregularly irregular QRS, and fibrillatory baseline. With $CHADS_2$-VASc $\geq$2 in men or $\geq$3 in women, oral anticoagulation (OAC) is indicated. NOACs (rivaroxaban, apixaban, dabigatran) are preferred over warfarin. Rate control with beta-blockers or rate-limiting CCBs is first-line.

Step 1 — Identify treatment: $CHADS_2$-VASc = 4 $\Rightarrow$ OAC indicated. Rate control with metoprolol + anticoagulation with rivaroxaban = **Option A.**

Why other options are wrong:

- Option A: Rate control + rivaroxaban — **CORRECT.** Rate control first + NOAC for stroke prevention; CHA_2DS_2-VASc 4 mandates anticoagulation.
- Option B: Amiodarone only without anticoagulation — dangerous; high CHA_2DS_2-VASc score requires OAC regardless of rhythm strategy.
- Option C: Aspirin alone — aspirin is no longer recommended for AF stroke prophylaxis (inferior to OAC, similar bleeding risk).
- Option D: DC cardioversion without anticoagulation — contraindicated if AF duration >48 hours or unknown; thromboembolism risk; must anticoagulate for $\geq$3 weeks before or use TOE to exclude LA thrombus.

Final Answer: Rate control (metoprolol) + anticoagulation (rivaroxaban) $\Rightarrow$ $\boxed{A}$

Answer: (A) Go Back to Q 133

Q134.

Solution

Concept — Severe Mitral Stenosis / BMV: Mitral valve area (MVA) <1.0 cm^2 = very severe MS. The opening snap–A2 interval <80 ms indicates severe MS (short interval = high LA pressure). Balloon Mitral Valvotomy (BMV/PTMC) is the procedure of choice for symptomatic severe MS with favourable valve morphology (Wilkins score $\leq$8, no MR, no LA clot).

Step 1 — Decision: MVA 0.9 cm^2, mean gradient 14 mmHg, symptomatic (NYHA II), no contraindication to BMV mentioned $\Rightarrow$ BMV is the intervention of choice.

Why other options are wrong:

- Option A: Diuretics provide symptomatic relief but do not alter disease course or natural history; not definitive treatment.

- Option B: BMV — **CORRECT.** Definitive treatment for severe symptomatic MS with favourable anatomy.
- Option C: Mitral valve replacement — reserved for unfavourable anatomy (Wilkins >8), significant MR, or failed BMV.
- Option D: Beta-blockers improve haemodynamics by slowing heart rate (longer diastolic filling), but are adjunct therapy, not definitive treatment.

Final Answer: Balloon Mitral Valvotomy (BMV/PTMC) ⇒ B

Answer: (B) Go Back to Q 134

Q135.

Solution

Concept — Hypertension with CKD / ACEi: In patients with hypertension and CKD (especially with proteinuria/microalbuminuria), ACE inhibitors (or ARBs) are the drug class of choice. They reduce intraglomerular pressure, proteinuria, and slow progression of CKD. This benefit is independent of their BP-lowering effect.

Step 1 — Identify best drug: Microalbuminuria + CKD + hypertension ⇒ ramipril (ACE inhibitor) is first-line per JNC 8, KDIGO, and ESC guidelines.

Why other options are wrong:

- Option A: Thiazide diuretic — less effective below eGFR 30; not first-line in CKD with proteinuria.
- Option B: Amlodipine (CCB) — lowers BP effectively but does not reduce proteinuria as effectively as ACE inhibitors in CKD.
- Option C: ACE inhibitor (ramipril) — **CORRECT.** Renoprotective, antiproteinuric; first-line in hypertension with CKD/microalbuminuria.
- Option D: Atenolol (beta-blocker) — no renoprotective advantage in CKD; not preferred as first-line in this scenario.

Final Answer: ACE inhibitor (ramipril) — renoprotective + antihypertensive ⇒ C

Answer: (C) Go Back to Q 135

Q136.

Solution

Concept — Community-Acquired Pneumonia / Most Common Organism: *Streptococcus pneumoniae* is the single most common cause of CAP in adults worldwide. Clinical features of pneumococcal pneumonia: acute onset, rigors, rusty sputum, lobar consolidation, high WBC. Empirical treatment for CAP CURB-65 score 2 (moderate severity): beta-lactam (amoxicillin-clavulanate) ± macrolide.

Step 1 — Identify organism + antibiotic: Rusty sputum + lobar consolidation + acute onset + CURB-65 2 ⇒ *S. pneumoniae*; amoxicillin-clavulanate or penicillin G.

Why other options are wrong:

- Option A: *S. aureus* — causes CAP post-influenza, IV drug users; not the most common; vancomycin is for MRSA, not first-line CAP.
- Option B: *Klebsiella* — causes lobar CAP in alcoholics/diabetics (currant jelly sputum, upper lobe); less typical here.
- Option C: *Legionella* — atypical pneumonia; presents with hyponatraemia, diarrhoea, relative bradycardia; not typical lobar consolidation with rusty sputum.
- Option D: *S. pneumoniae* + amoxicillin-clavulanate — **CORRECT.** Most common CAP organism; beta-lactam is first-line for moderate CAP.

Final Answer: *Streptococcus pneumoniae*; amoxicillin-clavulanate ⇒ D

Answer: (D) Go Back to Q 136

Q137.

Solution

Concept — GOLD Classification of COPD: Based on post-bronchodilator FEV_1 % predicted: GOLD 1 (Mild) ≥80%; GOLD 2 (Moderate) 50–79%; **GOLD 3 (Severe) 30–49%**; GOLD 4 (Very Severe) <30%. This patient has FEV_1 42% = GOLD 3.

Step 1 — Classify: FEV_1/FVC 0.58 confirms obstruction. FEV_1 42% predicted ⇒ GOLD Stage 3 (Severe).

Why other options are wrong:

- Option A: GOLD 3, FEV_1 30–49% — **CORRECT.** 42% falls within the GOLD 3 range.
- Option B: GOLD 2 (50–79%) — 42% is below 50%; does not fit GOLD 2.

- Option C: GOLD 4 (<30%) — 42% is above 30%; does not fit GOLD 4.
- Option D: GOLD 1 (≥80%) — 42% is far below 80%; does not fit GOLD 1.

Final Answer: FEV_1 42% = GOLD 3 (Severe) ⇒ A

Answer: (A) Go Back to Q 137

Q138.

Solution

Concept — Light's Criteria for Exudate: An effusion is an **exudate** if ANY of these are met: (1) Pleural fluid protein/serum protein >0.5; (2) Pleural fluid LDH/serum LDH >0.6; (3) Pleural fluid LDH >2/3 upper limit of normal for serum LDH. Transudate meets none of these.

Step 1 — Apply criteria: PF protein/serum protein = 4.8/6.5 = **0.74** > **0.5** ⇒ exudate. Also PF LDH/serum LDH = 350/280 = **1.25** > **0.6** ⇒ exudate. Both criteria met.

Why other options are wrong:

- Option A: Transudate/CHF — incorrect; the protein and LDH ratios both exceed Light's criteria thresholds.
- Option B: Exudate, PF protein/serum protein >0.5 — **CORRECT.** 4.8/6.5 = 0.74 > 0.5; meets Light's criterion 1.
- Option C: Transudate, LDH ratio <0.6 — incorrect; LDH ratio is 1.25, well above 0.6.
- Option D: Exudate because glucose <60 — glucose is 65 mg/dL (>60), so this criterion is not met; glucose <60 suggests empyema, malignancy or TB, but is not one of Light's three criteria.

Final Answer: Exudate — PF protein/serum protein ratio 0.74 > 0.5 ⇒ B

Answer: (B) Go Back to Q 138

Q139.

Solution

Concept — Acute Severe Asthma / Emergency Management: Markers of acute severe asthma: PEFR 33–50% predicted, RR $\geq$25/min, HR $\geq$110 bpm, unable to complete sentences. Life-threatening: PEFR $<$33%, silent chest, SpO_2 $<$92%. Emergency treatment: continuous nebulised SABA (salbutamol) + ipratropium, systemic corticosteroids, controlled oxygen.

Step 1 — Apply BTS/SIGN guidelines: PEFR 40%, SpO_2 90%, RR 28 = acute severe (not life-threatening yet). Immediate: nebulised salbutamol + ipratropium + IV/oral prednisolone + controlled O_2. If no improvement: IV magnesium sulphate.

Why other options are wrong:

- Option A: Doubling ICS and discharge — inappropriate for an acute severe attack requiring emergency bronchodilator therapy.
- Option B: IV $MgSO_4$ then discharge — magnesium is a second-line agent given after initial bronchodilators; discharging after 30 min is unsafe.
- Option C: Nebulised salbutamol + ipratropium + systemic steroid + O_2 — **CORRECT.** First-line management of acute severe asthma.
- Option D: Immediate intubation — reserved for life-threatening/near-fatal asthma (silent chest, cyanosis, reduced consciousness); not indicated as the first step here.

Final Answer: Nebulised salbutamol + ipratropium + systemic corticosteroid + O_2 $\Rightarrow$ C

Answer: (C) Go Back to Q 139

Q140.

Solution

Concept — *H. pylori* Eradication / Peptic Ulcer Disease: Duodenal ulcer with positive RUT (rapid urease test) confirms *H. pylori* infection. First-line eradication: Triple therapy — PPI + Clarithromycin + Amoxicillin for 14 days (or 10 days). This achieves $>$80% eradication and ulcer healing. Confirm eradication with urea breath test 4–8 weeks after treatment.

Step 1 — Best treatment: RUT positive $\Rightarrow$ eradication mandatory. PPI-based triple therapy for 14 days is the standard first-line regimen.

Why other options are wrong:

- Option A: PPI alone — will heal ulcer but does not eradicate *H. pylori*; high recurrence rate.
- Option B: H_2 blocker alone — largely replaced by PPIs; does not address *H. pylori* infection.
- Option C: Sucralfate + antacids — mucosal protection only; no anti-*H. pylori* activity.
- Option D: Triple therapy (Omeprazole + Clarithromycin + Amoxicillin) — **CORRECT.** Standard first-line *H. pylori* eradication regimen.

Final Answer: Triple therapy — PPI + Clarithromycin + Amoxicillin for 14 days $\Rightarrow$ D

Answer: (D) Go Back to Q 140

Q141.

Solution

Concept — Crohn's Disease vs UC: Crohn's disease (CD) can involve any part of GI tract (mouth to anus), with transmural inflammation, skip lesions, cobblestone mucosa, granulomas, strictures, and fistulas (including perianal). Non-caseating granulomas on biopsy are pathognomonic of CD. UC is limited to the colon, continuous from rectum, with bloody diarrhoea and no granulomas.

Step 1 — Identify features: Skip lesions + cobblestone + terminal ileum involvement + non-caseating granulomas + perianal fistula = **Crohn's disease**.

Why other options are wrong:

- Option A: Crohn's disease — **CORRECT.** All classic features: skip lesions, granulomas, perianal fistula, terminal ileum.
- Option B: Ulcerative colitis — continuous colonic involvement from rectum, no skip lesions, no granulomas, bloody diarrhoea.
- Option C: Intestinal TB — also causes granulomas but these are caseating; ileocaecal involvement most common; AFB stain/culture and CXR can differentiate.
- Option D: Ischaemic colitis — affects watershed areas (splenic flexure, sigmoid); no granulomas; acute onset in older patients with vascular disease.

Final Answer: Crohn's disease — skip lesions + non-caseating granulomas + perianal fistula $\Rightarrow$ A

Answer: (A) Go Back to Q 141

Q142.

Solution

Concept — Child-Pugh Classification of Liver Cirrhosis: Scores 5 parameters (each 1–3 points): serum bilirubin, albumin, PT (INR), ascites, encephalopathy. Class A = 5–6, Class B = 7–9, Class C = 10–15 (worst prognosis).

Step 1 — Calculate score: Bilirubin 3.5 (2 pts), Albumin 2.4 (3 pts), PT prolonged 5 sec (2 pts), Encephalopathy grade II (2 pts), Ascites moderate (2 pts) = Total: 2+3+2+2+2 = **11** $\Rightarrow$ Child C.

Why other options are wrong:

- Option A: Child A (5–6) — incorrect; total score is 11, far above 6.
- Option B: Child C ($\geq$10) — **CORRECT.** Score 11 = Child C; high surgical mortality (>50%).
- Option C: Child B (7–9) — score is 11, above 9.
- Option D: Cannot be calculated — all parameters are available and the score is calculable.

Final Answer: Child-Pugh score 11 = Child C (high risk) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 142

Q143.

Solution

Concept — CT Severity Index (CTSI) / Balthazar Score: Severe acute pancreatitis on CECT is defined by pancreatic necrosis (>30% non-enhancement) and/or peripancreatic complications. CTSI = Balthazar grade (A–E, 0–4 pts) + necrosis score (0–4 pts). CTSI $\geq$7 = severe disease with high morbidity/mortality.

Step 1 — Identify severe finding: >30% non-enhancement of pancreatic parenchyma indicates necrosis (4 points) + peripancreatic fluid collections (Balthazar E = 4 pts) $\Rightarrow$ CTSI = 8 $\Rightarrow$ severe.

Why other options are wrong:

- Option A: CTSI 2 (oedema only, Balthazar B, 0 necrosis) — mild; excellent prognosis.
- Option B: CTSI 3 (fat stranding, Balthazar C, 0 necrosis) — mild-moderate; low morbidity.
- Option C: >30% non-enhancement + fluid collections (CTSI $\geq$7) — **CORRECT.** Indicates pancreatic necrosis; worst prognosis; managed in ICU.

- Option D: CTSI 4 (peripancreatic inflammation, Balthazar D, 0 necrosis) — moderate; better prognosis than necrosis.

Final Answer: >30% pancreatic non-enhancement + fluid collections = CTSI ≥7 (severe) ⇒ C

Answer: (C) Go Back to Q 143

Q144.

Solution

Concept — FENa in AKI Classification: FENa = (Urine Na × Serum Cr)/(Serum Na × Urine Cr) × 100. Pre-renal AKI: FENa <1% (tubules avidly reabsorb Na, kidneys intact). Intra-renal (ATN): FENa >2% (damaged tubules cannot reabsorb Na).

Step 1 — Calculate FENa: FENa = (8 × 3.6)/(140 × 340) × 100 = 28.8/47600 × 100 = **0.06%** < 1% ⇒ **Pre-renal AKI**.

Step 2 — Clinical context: Post-operative emergency laparotomy with oliguria ⇒ hypovolaemia is the primary cause ⇒ pre-renal AKI.

Why other options are wrong:

- Option A: Intra-renal AKI (ATN); FENa >2% — wrong; FENa is 0.06%, indicating intact tubular Na reabsorption.
- Option B: Post-renal; FENa >2% — wrong; post-renal can have variable FENa; no obstruction mentioned.
- Option C: Intra-renal; FENa <1% — contradicts itself; ATN characteristically has FENa >2%.
- Option D: Pre-renal AKI (hypovolaemia); FENa <1% — **CORRECT.** FENa = 0.06% confirms intact tubular function; post-surgical hypovolaemia is the cause.

Final Answer: Pre-renal AKI; FENa <1% (calculated 0.06%) ⇒ D

Answer: (D) Go Back to Q 144

Q145.

Solution

Concept — Anaemia of CKD / Iron Deficiency / ESA Therapy: Anaemia of CKD results from reduced EPO production + functional iron deficiency. Before starting ESA, iron deficiency must be corrected (target ferritin >200 ng/mL, TSAT >20%). IV iron is preferred in CKD (better absorption than oral). If Hb remains <10 g/dL after iron repletion, ESA is added. Target Hb 10–12 g/dL (not higher — thrombosis risk).

Step 1 — Identify deficiency: Ferritin 28 ng/mL + TSAT 12% = absolute iron deficiency. First correct iron deficiency with IV iron, then add ESA if needed.

Why other options are wrong:

- Option A: IV iron first, then ESA if Hb <10 — **CORRECT.** KDIGO guidelines recommend correcting iron deficiency before or alongside ESA.
- Option B: Oral iron only — oral iron has poor absorption in CKD (uraemia, gut changes); IV iron is preferred.
- Option C: Immediate blood transfusion — reserved for symptomatic severe anaemia or haemodynamic instability; not first-line for CKD anaemia with Hb 7.8 (stable).
- Option D: ESA alone — starting ESA without iron in iron-deficient patients causes functional iron deficiency and hyporesponsiveness.

Final Answer: IV iron supplementation first, then ESA if Hb still <10 g/dL ⇒ A

Answer: (A) Go Back to Q 145

Q146.

Solution

Concept — Lupus Nephritis Classification / ISN-RPS 2003: Class IV (Diffuse Proliferative LN) is the most common and most severe form. Characterised by diffuse (>50% of glomeruli) endocapillary/mesangial proliferation, subendothelial deposits causing the classic "wire loop" lesion. Treat with high-dose corticosteroids + cyclophosphamide (NIH protocol) or mycophenolate mofetil (induction).

Step 1 — Match histology to class: Diffuse proliferative GN + subendothelial deposits + wire loops = **ISN/RPS Class IV**.

Why other options are wrong:

- Option A: Class II (mesangial) — minimal mesangial deposits, mild disease,

no wire loops.

- Option B: Class IV — **CORRECT.** Diffuse proliferative LN with wire loop lesions (subendothelial deposits).
- Option C: Class V (membranous) — subepithelial deposits, nephrotic syndrome predominant; no wire loops.
- Option D: Class III (focal) — <50% glomeruli involved; less severe than Class IV.

Final Answer: ISN/RPS Class IV (diffuse proliferative LN); treat with steroids + cyclophosphamide/MMF ⇒ B

Answer: (B) Go Back to Q 146

Q147.

Solution

Concept — Acute Ischaemic Stroke / IV tPA: IV alteplase (0.9 mg/kg, max 90 mg, 10% as bolus then rest over 60 min) is the standard of care for ischaemic stroke within **4.5 hours** of symptom onset, after excluding haemorrhage on non-contrast CT. Warfarin/anticoagulant use is a contraindication only if INR >1.7; INR 1.0 here means no contraindication.

Step 1 — Eligibility check: Symptom onset 1.5 hours ago (within 4.5 hr window); CT shows no haemorrhage; INR 1.0 (no anticoagulation); BP can be managed to <185/110 before tPA. AF is **not** a contraindication to tPA.

Why other options are wrong:

- Option A: Aspirin + clopidogrel — dual antiplatelet not indicated acutely in a tPA candidate (increases haemorrhage risk); anticoagulation started 24 hours after tPA for AF.
- Option B: Heparin immediately — not indicated in acute ischaemic stroke; increases haemorrhagic transformation risk.
- Option C: IV alteplase within 4.5 hours + anticoagulation after 24 hours — **CORRECT.** Standard care for eligible ischaemic stroke.
- Option D: tPA contraindicated in AF — **INCORRECT**; AF is not a contraindication to tPA.

Final Answer: IV alteplase (tPA) 0.9 mg/kg within 4.5-hour window ⇒ C

Answer: (C) Go Back to Q 147

Q148.

Solution

Concept — Bacterial Meningitis / *N. meningitidis*: Gram-negative diplococci in CSF of a young person with purpuric rash (meningococcaemia) = *Neisseria meningitidis*. CSF: turbid, high WBC (polymorphs), high protein, low glucose (<40% of blood glucose). Treatment: IV benzylpenicillin or ceftriaxone. Close contacts should receive rifampicin prophylaxis.

Step 1 — Identify organism: Gram-negative diplococci + purpuric rash + meningism + college student = *N. meningitidis*.

Why other options are wrong:

- Option A: *S. pneumoniae* — Gram-positive diplococci; most common in adults; no purpuric rash; treated with cefotaxime + dexamethasone.
- Option B: *Listeria* — Gram-positive rod; occurs in elderly, immunocompromised, neonates; CSF monocytosis.
- Option C: *Cryptococcus* — fungal; India ink positive; seen in immunocompromised (HIV); CSF lymphocytosis.
- Option D: *N. meningitidis* + ceftriaxone + prophylaxis — **CORRECT.** All clinical features, CSF findings, and Gram stain consistent with meningococcal meningitis.

Final Answer: *Neisseria meningitidis*; IV benzylpenicillin/ceftriaxone + contact prophylaxis ⇒ D

Answer: (D) Go Back to Q 148

Q149.

Solution

Concept — Absence Epilepsy / Drug of Choice: Childhood absence epilepsy (CAE) presents as brief (<30s) staring spells with abrupt onset and offset, no post-ictal confusion, and 3 Hz generalised spike-and-wave on EEG. Drug of choice: **Ethosuximide** (most effective for typical absence). Sodium valproate is an alternative. Carbamazepine and phenytoin can **worsen** absence seizures.

Step 1 — Identify seizure type + drug: 3 Hz GSW + brief staring spells = absence epilepsy. DOC = ethosuximide.

Why other options are wrong:

- Option A: Ethosuximide — **CORRECT.** Drug of choice for typical absence

epilepsy; blocks T-type calcium channels in thalamic neurons.

- Option B: Carbamazepine — used for focal (partial) seizures; **contraindicated** in absence epilepsy (can aggravate).
- Option C: Phenytoin — used for generalised tonic-clonic and focal seizures; not effective for absence and may worsen.
- Option D: Levetiracetam — broad-spectrum AED; not first-line for absence; ethosuximide/valproate are preferred.

Final Answer: Absence epilepsy — DOC is Ethosuximide $\Rightarrow$ A

Answer: (A) Go Back to Q 149

Q150.

Solution

Concept — Parkinson's Disease / Pharmacotherapy: Parkinson's disease is characterised by degeneration of dopaminergic neurons in the substantia nigra. Initial pharmacotherapy for a newly diagnosed patient with functional impairment: **Levodopa/Carbidopa** remains the most effective drug for motor symptoms. Carbidopa inhibits peripheral dopa decarboxylase, reducing peripheral side effects and increasing brain levodopa availability.

Step 1 — Select best initial treatment: Resting tremor + bradykinesia + rigidity + shuffling gait (intact cognition) = idiopathic PD. Levodopa/carbidopa is the gold standard for motor control.

Why other options are wrong:

- Option A: Donepezil — acetylcholinesterase inhibitor for Alzheimer's dementia; not for motor symptoms of PD.
- Option B: Levodopa/Carbidopa — **CORRECT.** Gold standard pharmacotherapy for PD; most effective motor symptom control.
- Option C: Propranolol — used for essential tremor (action tremor), not resting tremor of PD; no dopaminergic effect.
- Option D: Haloperidol — dopamine D2 blocker; causes or worsens parkinsonism; **contraindicated** in PD.

Final Answer: Levodopa/Carbidopa — most effective initial treatment for Parkinson's motor symptoms $\Rightarrow$ B

Answer: (B) Go Back to Q 150

Q151.

Solution

Concept — DKA Management / Fluid First: In DKA, the critical initial step is aggressive IV fluid resuscitation with isotonic saline (0.9% NaCl) 1–1.5 L in the first hour to restore circulating volume. Insulin is started **after** initial fluid resuscitation and **only when $K^+ \geq 3.5$ mEq/L** (to prevent dangerous hypokalaemia when insulin drives K into cells). The serum K of 5.8 appears high but will drop rapidly once insulin is given.

Step 1 — Critical order of management: IV normal saline first (even though glucose is high) $\Rightarrow$ then insulin $\Rightarrow$ then K replacement as needed.

Why other options are wrong:

- Option A: Start insulin immediately — dangerous; must restore volume first and ensure K $\geq$ 3.5 to avoid fatal hypokalaemia.
- Option B: Sodium bicarbonate — only considered if pH $<$6.9; pH 7.12 does not meet this threshold; bicarb can worsen hypokalaemia and cerebral oedema.
- Option C: IV 0.9% normal saline (1–1.5 L first hour) — **CORRECT.** First step in DKA; corrects hypovolaemia, improves perfusion, and helps reduce glucose and ketones.
- Option D: IV KCl 40 mEq/hr immediately — K is already 5.8 (high); adding KCl before volume correction and insulin is dangerous (hyperkalaemia $\Rightarrow$ cardiac arrest).

Final Answer: IV 0.9% normal saline — first step before insulin in DKA $\Rightarrow$ C

Answer: (C) Go Back to Q 151

Q152.

Solution

Concept — Hypothyroidism / Levothyroxine Replacement: Autoimmune hypothyroidism (Hashimoto's thyroiditis) with elevated TSH, low free T4, and positive anti-TPO antibodies. Treatment: **Levothyroxine (T4)** is the drug of choice. Target TSH in replacement therapy for primary hypothyroidism: **0.5–2.5 mIU/L** (or 1.0–2.0 mIU/L in most guidelines). Full replacement dose: 1.6 mcg/kg/day.

Step 1 — Identify correct treatment: Levothyroxine (synthetic T4) is universally recommended; T3 is unnecessary as peripheral conversion of T4$\rightarrow$T3 is adequate.

Why other options are wrong:

- Option A: Liothyronine (T3) — not used as monotherapy for hypothyroidism; short half-life, fluctuating levels; target TSH 0.1–0.5 is too suppressed.
- Option B: Carbimazole — antithyroid drug for **hyperthyroidism**; contraindicated in hypothyroidism.
- Option C: PTU + iodine — used for hyperthyroidism, thyroid storm; opposite indication.
- Option D: Levothyroxine (T4); target TSH 0.5–2.5 mIU/L — **CORRECT.** Standard treatment for primary hypothyroidism.

Final Answer: Levothyroxine (T4); target TSH 0.5–2.5 mIU/L $\Rightarrow$ D

Answer: (D) Go Back to Q 152

Q153.

Solution

Concept — Primary Adrenal Insufficiency (Addison's Disease): Presents with fatigue, weight loss, hyperpigmentation (due to excess ACTH/MSH), postural hypotension, hyponatraemia, and hyperkalaemia (due to mineralocorticoid deficiency). Peak cortisol $<$18 mcg/dL on ACTH stimulation test confirms adrenal insufficiency. Hyperpigmentation distinguishes primary (elevated ACTH) from secondary adrenal insufficiency.

Step 1 — Confirm diagnosis and treatment: Hyperpigmentation + hyponatraemia + hyperkalaemia + failed ACTH test = primary adrenal insufficiency. Treatment: hydrocortisone (glucocorticoid) + fludrocortisone (mineralocorticoid).

Why other options are wrong:

- Option A: Primary AI (Addison's) + hydrocortisone + fludrocortisone — **CORRECT.** Mineralocorticoid replacement (fludrocortisone) is essential in primary AI to correct hyponatraemia and hyperkalaemia.
- Option B: Secondary AI — ACTH is low (pituitary failure); no hyperpigmentation; no mineralocorticoid deficiency (aldosterone regulated by renin-angiotensin, not ACTH); fludrocortisone not needed.
- Option C: Cushing's syndrome — opposite (excess cortisol); presents with weight gain, moon face, buffalo hump; no hyperpigmentation in this pattern.
- Option D: Conn's syndrome (primary hyperaldosteronism) — hypertension + hypokalaemia; opposite electrolyte pattern.

Final Answer: Primary adrenal insufficiency; hydrocortisone + fludrocortisone $\Rightarrow$

A

Answer: (A) Go Back to Q 153

Q154.

Solution

Concept — Iron Deficiency Anaemia (IDA) vs Thalassaemia Trait: Both cause microcytic hypochromic anaemia. **Mentzer index** (MCV/RBC): <13 suggests thalassaemia trait; >13 suggests IDA. Here, Mentzer = 12 (<13) suggests thalassaemia. However, serum ferritin 6 ng/mL (low) and TIBC 480 (raised) strongly confirm absolute iron deficiency. Both can coexist. The **confirmatory test** for thalassaemia is HPLC for Hb analysis (elevated HbA_2 $>3.5\%$ in beta-thalassaemia trait). For IDA: ferritin + TIBC (already diagnostic here).

Step 1 — Ferritin 6 + TIBC 480 = IDA confirmed. But Mentzer 12 suggests concurrent thalassaemia trait; HPLC is needed to clarify.

Why other options are wrong:

- Option A: Anaemia of chronic disease — ferritin is typically NORMAL or elevated in ACD; low ferritin here excludes ACD.
- Option B: IDA — ferritin and TIBC confirm IDA, but Mentzer index 12 prompts investigation for coexisting thalassaemia trait; HPLC is the best confirmatory test here given family history.
- Option C: IDA + HPLC — **CORRECT.** In pregnancy, with family history of thalassaemia and Mentzer <13, HPLC is the confirmatory test to rule out thalassaemia (treat iron deficiency first, then confirm).
- Option D: Sideroblastic anaemia — bone marrow shows ring sideroblasts; ferritin is elevated; not consistent with this presentation.

Final Answer: Iron deficiency anaemia + HPLC to rule out thalassaemia trait $\Rightarrow$ B

Answer: (B) Go Back to Q 154

Q155.

Solution

Concept — SLE Haematological Manifestations / SLICC Criteria: SLICC 2012 haematological criteria include: haemolytic anaemia (Coombs positive), or leukopenia ($<4000/mm^3$), or lymphopenia ($<1000/mm^3$), or thrombocytopenia ($<100{,}000/mm^3$). The patient has a **positive direct Coombs test and Hb 9.4 g/dL** = autoimmune haemolytic anaemia (AIHA) — a specific SLICC haematological criterion.

Step 1 — Identify the specific haematological feature: Direct Coombs test positive + anaemia = AIHA. AIHA is counted as the haematological domain criterion.

Why other options are wrong:

- Option A: Thrombocytopenia $<100{,}000$ — not mentioned in this patient; platelet count not reported as low.
- Option B: Leukopenia <4000 — not reported in this patient.
- Option C: AIHA (Coombs positive) — **CORRECT.** Direct Coombs positive with Hb 9.4 = autoimmune haemolytic anaemia; counts as haematological SLICC criterion.
- Option D: Lymphopenia <1000 — not specifically stated; AIHA is the more specific finding here.

Final Answer: Autoimmune haemolytic anaemia (Coombs positive) = haematological SLICC criterion $\Rightarrow$ C

Answer: (C) Go Back to Q 155

Q156.

Solution

Concept — Biologic DMARDs in RA / EULAR Guidelines: When a patient with seropositive RA fails adequate csDMARD therapy (methotrexate $\pm$ another csDMARD) at 6 months with active disease (DAS28 >3.2), the EULAR 2022 guidelines recommend adding a **biologic DMARD** (bDMARD) — TNF inhibitor (etanercept, adalimumab, infliximab) or a JAK inhibitor (tofacitinib, baricitinib).

Step 1 — Patient has failed MTX + leflunomide = adequate csDMARD trial $\Rightarrow$ escalate to bDMARD/JAKi.

Why other options are wrong:

- Option A: Add hydroxychloroquine — triple csDMARD therapy (MTX + SSZ

+ HCQ); may be tried if bDMARDs unavailable, but less effective than biologics in high-risk seropositive RA.
- Option B: Switch to SSZ + HCQ — step backwards; this combination is less potent than MTX + leflunomide.
- Option C: Increase MTX to 25 mg/week and wait — patient already on 20 mg/week, has failed; waiting another 6 months with active disease causes joint damage.
- Option D: Add TNF-alpha inhibitor or JAK inhibitor — **CORRECT.** EULAR 2022: after failing one or more csDMARDs with poor prognostic features (RF+, anti-CCP+, erosions), add bDMARD or JAKi.

Final Answer: Add biologic DMARD (TNF inhibitor) or JAK inhibitor $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 156

Q157.

Solution

Concept — Acute Gout / ULT: Acute gout is treated with NSAIDs (indomethacin 50 mg TID), colchicine (1 mg then 0.5 mg 1 hour later), or corticosteroids. Urate-lowering therapy (ULT): **allopurinol** (xanthine oxidase inhibitor) is first-line; target serum uric acid <6 mg/dL (<5 mg/dL in tophaceous gout). Do not start allopurinol **during** an acute attack; wait 2–4 weeks after attack resolves.

Step 1 — Identify acute and long-term treatment: Acute: indomethacin or colchicine. Long-term ULT: allopurinol; target SUA <6 mg/dL.

Why other options are wrong:

- Option A: NSAIDs/colchicine acute + allopurinol ULT, target <6 mg/dL — **CORRECT.** Comprehensive management with correct ULT target.
- Option B: Aspirin for acute gout — aspirin at low doses **raises** serum uric acid (reduces renal urate excretion); contraindicated in gout.
- Option C: IV methylprednisolone + no ULT — steroids are used if NSAIDs/colchicine contraindicated; but tophaceous gout with 2+ attacks requires ULT.
- Option D: Probenecid alone without acute treatment — probenecid is a uricosuric (second-line ULT); cannot be used in patients with uric acid nephrolithiasis; not the acute treatment.

Final Answer: NSAIDs/colchicine for acute + allopurinol ULT, target SUA <6 mg/dL $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 157

Q158.

Solution

Concept — TB Diagnosis in HIV / GeneXpert MTB/RIF: In HIV-positive patients, smear-negative TB is common due to lower bacillary load. **GeneXpert MTB/RIF** (CBNAAT) is the WHO-recommended first-line diagnostic test for TB in HIV-positive patients. It simultaneously detects *M. tuberculosis* DNA and rifampicin resistance within 2 hours with high sensitivity (even in smear-negative samples).

Step 1 — Best next test: Smear negative $\times 3$ in an HIV-positive patient with strong clinical/radiological evidence of TB $\Rightarrow$ GeneXpert MTB/RIF of sputum is next.

Why other options are wrong:

- Option A: Mantoux/TST — unreliable in HIV (immunosuppression causes false negatives); not diagnostic.
- Option B: GeneXpert MTB/RIF — **CORRECT.** High sensitivity in smear-negative TB; detects RIF resistance simultaneously; WHO first-line in HIV+ patients.
- Option C: IGRA — better than Mantoux in HIV but still unreliable with low CD4; not a direct diagnostic test for active TB disease.
- Option D: Bronchoscopy for fungal culture only — incomplete; if bronchoscopy done, should include TB culture, GeneXpert of BAL, and fungal culture; doing only fungal culture misses TB.

Final Answer: GeneXpert MTB/RIF (CBNAAT) — WHO-recommended for smear-negative TB in HIV $\Rightarrow$ B

Answer: (B) Go Back to Q 158

Q159.

Solution

Concept — JVP Waveform / Tricuspid Regurgitation: Normal JVP: 'a' wave (atrial systole), 'x' descent (atrial relaxation), 'v' wave (venous filling during ventricular systole), 'y' descent (tricuspid opens). In **tricuspid regurgitation (TR):** During ventricular systole, blood regurgitates back into the right atrium, merging the 'c' and 'v' waves into a large 'cv' wave and obliterating the 'x' descent (normally the 'x' is due to atrial relaxation + descent of AV junction, which is reversed by

TR). This is pathognomonic.

Step 1 — JVP in TR: Large 'cv' wave + obliterated 'x' descent. The 'y' descent is prominent (tricuspid opens to a high-pressure RA).

Why other options are wrong:

- Option A: Absent 'a' wave — seen in atrial fibrillation (no organised atrial activity).
- Option B: Cannon 'a' waves — seen in complete heart block, VT (atria contracting against closed tricuspid valve).
- Option C: Prominent 'cv' wave with obliterated 'x' descent — **CORRECT.** Classic sign of tricuspid regurgitation.
- Option D: Prominent 'y' descent (Friedrich's sign) — seen in constrictive pericarditis (both 'x' and 'y' are prominent, sharp 'y'); not the primary feature of TR.

Final Answer: TR — prominent 'cv' wave + obliterated 'x' descent $\Rightarrow$ C

Answer: (C) Go Back to Q 159

Q160.

Solution

Concept — Mechanism of Amlodipine (Calcium Channel Blocker): Amlodipine is a dihydropyridine **L-type (Long-acting) voltage-gated calcium channel blocker**. It blocks calcium entry into vascular smooth muscle cells, preventing actin-myosin interaction, causing **vasodilatation** and **reduced peripheral vascular resistance (PVR)**, thereby lowering blood pressure. It has minimal effect on the heart (unlike verapamil/diltiazem).

Step 1 — Confirm mechanism: Amlodipine $\Rightarrow$ blocks L-type Ca^{2+} channels in vascular smooth muscle $\Rightarrow$ vasodilatation $\Rightarrow$ reduced PVR $\Rightarrow$ lower BP.

Why other options are wrong:

- Option A: Blocks beta-1 receptors — mechanism of beta-blockers (metoprolol, atenolol); reduces heart rate and cardiac output; not amlodipine.
- Option B: Inhibits ACE — mechanism of ACE inhibitors (ramipril, enalapril); reduces angiotensin II; not amlodipine.
- Option C: Blocks alpha-1 receptors — mechanism of alpha-blockers (prazosin, doxazosin); causes vasodilatation but not amlodipine's mechanism.
- Option D: Blocks L-type voltage-gated Ca^{2+} channels in vascular smooth

muscle — **CORRECT.** Amlodipine's primary antihypertensive mechanism.

Final Answer: Amlodipine blocks L-type Ca^{2+} channels $\Rightarrow$ vasodilatation $\Rightarrow$ reduced PVR $\Rightarrow$ D

Answer: (D) Go Back to Q 160

Q161.

Solution

Concept — Schizophrenia / First-Episode Psychosis: Schizophrenia requires $\geq$2 of the following for $\geq$1 month: delusions, hallucinations, disorganised speech, grossly disorganised or catatonic behaviour, negative symptoms — plus 6 months of continuous disturbance. Third-person auditory hallucinations (voices commenting) are a first-rank symptom (Schneider). Treatment: atypical (second-generation) antipsychotics (risperidone, olanzapine, quetiapine) are first-line for first-episode psychosis.

Step 1 — Identify diagnosis and treatment: 6-month duration + persecutory delusions + third-person auditory hallucinations + social withdrawal + no drug use = schizophrenia. DOC: atypical antipsychotic (risperidone or olanzapine).

Why other options are wrong:

- Option A: Schizophrenia + atypical antipsychotic — **CORRECT.** Duration $\geq$6 months with positive and negative symptoms; risperidone/olanzapine are first-line.
- Option B: Bipolar disorder with psychotic features — bipolar has prominent mood episodes (mania/depression) as primary; this patient has primarily psychotic features without clear mood disturbance.
- Option C: Delusional disorder — only delusions without prominent hallucinations; functioning relatively preserved; duration requirement different.
- Option D: Brief psychotic disorder — duration $<$1 month; this patient has 6 months of illness; does not qualify.

Final Answer: Schizophrenia; atypical antipsychotic (risperidone or olanzapine) $\Rightarrow$ A

Answer: (A) Go Back to Q 161

Q162.

Solution

Concept — Bipolar Disorder Type I / Acute Mania Treatment: Acute mania is treated with mood stabilisers (lithium or valproate) combined with an antipsychotic (olanzapine, quetiapine, risperidone) for rapid behavioural control. Benzodiazepines can be added for agitation. Antidepressants (SSRIs) should not be used in acute mania (can worsen or precipitate switching).

Step 1 — Identify appropriate treatment: Decreased sleep + grandiosity + pressured speech + flight of ideas + disinhibition + spending recklessly $\geq$2 weeks = manic episode. Treatment: lithium or valproate + atypical antipsychotic.

Why other options are wrong:

- Option A: SSRIs (fluoxetine) — **contraindicated** in acute mania; can precipitate or worsen manic episode or cause rapid cycling.
- Option B: Lithium or valproate + antipsychotic — **CORRECT.** First-line combination for acute mania per NICE/BAP guidelines.
- Option C: Benzodiazepine alone — addresses agitation/sleep but does not treat the underlying mania; inadequate as monotherapy.
- Option D: ECT as first-line — ECT is reserved for severe, treatment-resistant mania or mixed states; not first-line pharmacotherapy.

Final Answer: Lithium or valproate + antipsychotic (olanzapine/risperidone) for acute mania $\Rightarrow$ B

Answer: (B) Go Back to Q 162

Q163.

Solution

Concept — Major Depressive Disorder / Moderate-Severe / SSRI + CBT: PHQ-9 score 15–19 = moderate-to-severe MDD. For moderate-to-severe MDD, NICE and APA guidelines recommend **combined antidepressant (SSRI) + psychotherapy (CBT)** as superior to either alone. Passive suicidal ideation without plan requires safety planning and close follow-up, not necessarily immediate hospitalisation.

Step 1 — Identify treatment: PHQ-9 18, passive SI, functional impairment $\Rightarrow$ SSRI + CBT + suicide risk assessment + safety planning.

Why other options are wrong:

- Option A: CBT alone — acceptable for mild-moderate MDD; for PHQ-9 18 (moderate-severe), medication should also be initiated.
- Option B: Benzodiazepine first — not indicated in MDD as primary treatment; may cause dependence; treats only the anxiety component.
- Option C: SSRI + CBT + suicide risk assessment and safety planning — **CORRECT.** Best practice for moderate-to-severe MDD with passive SI.
- Option D: ECT immediately — reserved for severe MDD with psychotic features, treatment resistance, catatonia, or immediate suicide risk with active plan and means; not first-line for passive SI.

Final Answer: SSRI + CBT + safety planning for moderate-to-severe MDD $\Rightarrow$ C

Answer: (C) Go Back to Q 163

Q164.

Solution

Concept — OCD / Defence Mechanisms / Undoing: In OCD, compulsions serve to neutralise or "undo" the anxiety caused by obsessive thoughts. The defence mechanism of **Undoing** involves performing an action or thought to "cancel out" or negate a previous unacceptable thought or action. Handwashing "undoes" the perceived contamination thought. This is distinct from displacement (shifting emotion to another target), repression (pushing into unconscious), or projection (attributing to another person).

Step 1 — Match mechanism: Contamination obsession $\rightarrow$ handwashing compulsion to neutralise = "Undoing." The ritual is meant to reverse or undo the perceived danger of the obsession.

Why other options are wrong:

- Option A: Repression — unconscious pushing away of unacceptable thoughts; the thoughts here are conscious and ego-dystonic.
- Option B: Projection — attributing one's own unacceptable thoughts/feelings to another; the patient knows the thoughts are his own.
- Option C: Displacement — shifting emotion from original source to a substitute; handwashing is specifically linked to the contamination thought, not displaced elsewhere.
- Option D: Undoing — **CORRECT.** Compulsive ritual (handwashing) is used to undo or neutralise the obsessive contamination thought.

Final Answer: Undoing — handwashing neutralises the contamination obsession

$\Rightarrow$ D

Answer: (D) Go Back to Q 164

Q165.

Solution

Concept — Alcohol Withdrawal / Wernicke's Prophylaxis: Alcohol withdrawal symptoms begin 6–24 hours after last drink, peak at 24–72 hours. Delirium tremens (DT) occurs at 72–96 hours. Management: (1) **IV thiamine BEFORE glucose** (to prevent precipitating Wernicke's encephalopathy); (2) **Benzodiazepines** (chlordiazepoxide, diazepam or lorazepam) — drug of choice for withdrawal and seizure prevention. Thiamine 100–500 mg IV TID for 3 days for those at risk of Wernicke's.

Step 1 — Correct management sequence: IV thiamine first (before any glucose/dextrose) $\Rightarrow$ then benzodiazepines for withdrawal seizures and DT prevention.

Why other options are wrong:

- Option A: IV thiamine (before glucose) + benzodiazepines — **CORRECT.** Dual role: prevents Wernicke's + controls withdrawal.
- Option B: Haloperidol + IV glucose — antipsychotics lower seizure threshold; glucose without thiamine risks precipitating Wernicke's; dangerous.
- Option C: Naltrexone — opioid antagonist used for alcohol craving/relapse prevention in **abstinent** patients; not for acute withdrawal.
- Option D: Disulfiram within 24 hours — aversive therapy (Antabuse); contraindicated during active alcohol withdrawal; causes severe acetaldehyde reaction if alcohol consumed; must be used only after detoxification.

Final Answer: IV thiamine first + benzodiazepines (chlordiazepoxide/diazepam) for alcohol withdrawal $\Rightarrow$ A

Answer: (A) Go Back to Q 165

Q166.

Solution

Concept — Delirium vs Dementia / Key Distinguishing Feature: The most important distinguishing feature of delirium from dementia is the **acute onset with fluctuating course and inattention**. Delirium: acute (<hours to days), fluctuating, impaired attention (core feature per CAM criteria), reversible cause (e.g., infection, surgery, drugs). Dementia: gradual onset over months/years, relatively stable course, attention preserved until late, progressive.

Step 1 — Identify delirium marker: Post-operative day 2 + acute confusion + fluctuating consciousness + inattention + visual hallucinations = delirium (Confusion Assessment Method — CAM criteria). In a patient with pre-existing dementia, delirium is superimposed.

Why other options are wrong:

- Option A: Gradually progressive memory loss over 2 years — feature of dementia (Alzheimer's); not delirium.
- Option B: Acute onset with fluctuating consciousness and inattention — **CORRECT.** Core CAM criterion for delirium; distinguishes from dementia's gradual, stable course.
- Option C: Aphasia — a feature of cortical dementias (Alzheimer's, FTD); also seen in stroke; not specific to delirium.
- Option D: Personality change over years — feature of frontotemporal dementia (FTD) or progressive dementia; not delirium.

Final Answer: Acute onset + fluctuating consciousness + inattention = delirium $\Rightarrow$ B

Answer: (B) Go Back to Q 166

Q167.

Solution

Concept — Suicide Risk Assessment / High-Risk Features: High-risk features for completed suicide: male gender, older age, previous attempts (especially with serious methods), specific plan, access to means, hopelessness, social isolation, comorbid substance use (alcohol), recent losses. This patient has multiple high-risk factors including previous serious attempts, specific plan, access to means, and heavy alcohol use. The **MOST appropriate action** for high-risk patients is inpatient psychiatric admission and means restriction.

Step 1 — Risk stratification: Multiple high-risk factors (previous attempt + specific plan + means + hopelessness + alcohol) = very high risk ⇒ inpatient admission.

Why other options are wrong:

- Option A: Outpatient counselling + safety contract — inadequate for a patient with specific plan and means; safety contracts have no proven efficacy in preventing suicide.
- Option B: Antidepressant and review in 1 week — insufficient given immediate risk with means available.
- Option C: Inpatient admission + means removal + treatment — **CORRECT.** Only safe option for a patient with active suicidal ideation, specific plan, and access to means.
- Option D: CBT referral — appropriate long-term; inadequate for an immediate high-risk situation.

Final Answer: Inpatient psychiatric admission + means restriction + treatment ⇒ C

Answer: (C) Go Back to Q 167

Q168.

Solution

Concept — Panic Disorder / Pharmacotherapy + CBT: Panic disorder = recurrent unexpected panic attacks + persistent concern about future attacks + avoidance behaviour (agoraphobia). First-line pharmacotherapy: **SSRIs** (paroxetine, escitalopram, sertraline) — start low and go slow (SSRIs can initially worsen panic). First-line psychotherapy: **CBT with panic-focused exposure**. Benzodiazepines provide short-term relief but are not first-line long-term.

Step 1 — Identify diagnosis and treatment: Recurrent unexpected episodes peaking at 10 min + avoidance + worry about future attacks + normal cardiac evaluation = panic disorder. SSRI + panic-focused CBT.

Why other options are wrong:

- Option A: GAD + buspirone — GAD presents with persistent, generalised worry (not episodic panic attacks); buspirone is for GAD, not panic disorder.
- Option B: Social phobia + beta-blockers — social phobia is situation-specific (social performance); beta-blockers are for performance anxiety, not panic disorder.

- Option C: Specific phobia + desensitisation — phobia is tied to a specific stimulus; panic disorder attacks are unexpected and not situation-specific; CBT for phobia uses gradual exposure, not the same as panic-focused CBT.
- Option D: Panic disorder + SSRI + panic-focused CBT — **CORRECT.** Recurrent unexpected panic attacks + avoidance = panic disorder; SSRI is first-line pharmacotherapy.

Final Answer: Panic disorder; SSRI (paroxetine/escitalopram) + CBT $\Rightarrow$ D

Answer: (D) Go Back to Q 168

Q169.

Solution

Concept — Lepromatous Leprosy (LL) / Classification: Leprosy classified by Ridley-Jopling: TT (tuberculoid) to LL (lepromatous). LL: poor cell-mediated immunity (CMI), high bacillary load (multibacillary, BI $\geq$4), foamy macrophages (Virchow cells) loaded with AFB on biopsy, diffuse skin thickening, madarosis, no/minimal nerve thickening (compared to TT), leonine facies. The biopsy description (foamy macrophages + AFB = Virchow cells) is pathognomonic of LL.

Step 1 — Identify type: Foamy macrophages + high bacillary load (AFB++) = **Lepromatous Leprosy (LL)**, multibacillary, poor CMI.

Why other options are wrong:

- Option A: LL — **CORRECT.** Foamy macrophages (Virchow cells) + high AFB load = lepromatous leprosy; poor CMI; multibacillary.
- Option B: TT — strong CMI; granulomas with Langhans giant cells; paucibacillary; distinct anaesthetic patch with nerve thickening; no foamy macrophages.
- Option C: BL — borderline; intermediate features; moderate bacillary load; both macrophages and some lymphocytes.
- Option D: Pure neuritic leprosy — nerve thickening only, no skin patches; biopsy of affected nerve shows AFB; does not match the skin biopsy finding here.

Final Answer: Lepromatous Leprosy (LL) — foamy macrophages + high AFB load + poor CMI $\Rightarrow$ A

Answer: (A) Go Back to Q 169

Q170.

Solution

Concept — Psoriasis / Systemic Treatment Threshold: For psoriasis with PASI >10 (moderate-to-severe), systemic therapy is indicated. **Methotrexate (MTX)** is the most commonly used first-line systemic treatment in resource-limited settings and many guidelines. Dose: 10–25 mg/week with folic acid supplementation. Monitor LFTs, FBC, renal function. Biologics (anti-TNF, anti-IL-17, anti-IL-23) are used after failure of conventional systemics.

Step 1 — Select first-line systemic: PASI 18 (moderate-to-severe) $\Rightarrow$ systemic needed. MTX is first-line systemic before biologics.

Why other options are wrong:

- Option A: Topical betamethasone indefinitely — topical steroids alone are inadequate for PASI 18; long-term potent topical steroids cause skin atrophy, tachyphylaxis.
- Option B: Systemic MTX + folic acid — **CORRECT.** First-line systemic agent for moderate-to-severe plaque psoriasis; evidence-based and cost-effective.
- Option C: Biologics (adalimumab) as first-line — biologics are second-line (after failure or contraindication to conventional systemics like MTX, cyclosporine, acitretin).
- Option D: Cyclosporine without prior phototherapy — cyclosporine is an option but is not preferred long-term (nephrotoxicity, hypertension); typically after phototherapy failure; also not generally "without prior" phototherapy per guidelines.

Final Answer: Systemic methotrexate 10–25 mg/week + folic acid for PASI >10 psoriasis $\Rightarrow$ B

Answer: (B) Go Back to Q 170

Q171.

Solution

Concept — Pemphigus Vulgaris / Autoimmune Bullous Disease: Pemphigus vulgaris (PV): suprabasal acantholysis, flaccid blisters, positive Nikolsky sign, oral involvement frequently first. DIF: intercellular IgG and C3 deposits (fish-net/chicken wire pattern) around individual keratinocytes. Autoantibody: anti-**desmoglein 3** (Dsg3) — IgG antibody against desmosomal protein desmoglein.

Step 1 — Match clinical + histology + DIF: Suprabasal acantholysis + flaccid

blisters + oral involvement + positive Nikolsky + fishnet IgG DIF = **Pemphigus vulgaris; anti-Dsg3 IgG**.

Why other options are wrong:

- Option A: Bullous pemphigoid — sub-epidermal blisters (not suprabasal acantholysis); tense blisters; Nikolsky negative; DIF linear IgG and C3 at BMZ; anti-BP180/BP230.
- Option B: Dermatitis herpetiformis — intensely pruritic papulovesicles on extensor surfaces; IgA deposits at dermal papillae tips; associated with coeliac disease; no acantholysis.
- Option C: Pemphigus vulgaris; anti-Dsg3 IgG — **CORRECT.** All features match: suprabasal acantholysis, fishnet IgG DIF, oral involvement, flaccid blisters.
- Option D: Linear IgA bullous dermatosis — linear IgA deposits at BMZ on DIF; sub-epidermal; not intercellular.

Final Answer: Pemphigus vulgaris — anti-desmoglein 3 (Dsg3) IgG antibody $\Rightarrow$ C

Answer: (C) Go Back to Q 171

Q172.

Solution

Concept — TEN (Toxic Epidermal Necrolysis) / SJS-TEN Spectrum: BSA classification: SJS <10%, SJS-TEN overlap 10–30%, TEN >30%. TEN: widespread skin detachment >30%, mucosal involvement (eyes, mouth, genitalia), positive Nikolsky sign, drug-induced (carbamazepine, allopurinol, sulfonamides). Management: withdraw causative drug, burn ICU, supportive care, consider IVIG or cyclosporine. Corticosteroids are **controversial** in TEN.

Step 1 — Classify by BSA: 35% BSA skin detachment = **TEN** (detachment >30%). Carbamazepine is a recognised cause (especially in HLA-B*1502 in South/South-East Asians).

Why other options are wrong:

- Option A: SJS (<10% BSA) — this patient has 35% detachment; too extensive for SJS.
- Option B: Erythema multiforme major — target lesions prominent; mucosal involvement; BSA detachment <10%; usually herpes simplex or Mycoplasma triggered; less extensive than SJS.

- Option C: DRESS — drug hypersensitivity syndrome; characterised by widespread erythematous rash + eosinophilia + lymphadenopathy + systemic organ involvement (hepatitis, pneumonitis); NOT skin detachment.
- Option D: TEN; withdraw drug + burns ICU + supportive + IVIG/cyclosporine — **CORRECT.** 35% BSA detachment = TEN; carbamazepine causative; burns unit management.

Final Answer: TEN (>30% BSA skin detachment); carbamazepine causative; burns ICU + IVIG ⇒ D

Answer: (D) Go Back to Q 172

Q173.

Solution

Concept — Scabies / Permethrin 5%: Scabies is caused by *Sarcoptes scabiei* var. *hominis*. Drug of choice: **permethrin 5% cream** applied from neck to toes (infants: include face/scalp), left on for 8–12 hours, washed off, repeated after 1 week. All household contacts must be treated simultaneously to prevent re-infestation. Oral ivermectin (200 mcg/kg) is an alternative (not approved for children <15 kg or <5 years in many guidelines).

Step 1 — Identify correct treatment: Night-time itch + web-space burrows + positive family history + dermoscopy mite sign = scabies. DOC in child: permethrin 5% + treat all contacts.

Why other options are wrong:

- Option A: Permethrin 5% + 8–12 hours + repeat after 1 week + all contacts — **CORRECT.** Standard WHO/AAD recommended treatment for scabies.
- Option B: Topical ivermectin 1% daily for 5 days — topical ivermectin is available but not the standard first-line protocol; daily 5-day application not the recommended schedule.
- Option C: Benzyl benzoate 25% undiluted in children — must be diluted to 12.5% or even 6.25% in children and infants; undiluted BBE is highly irritant and toxic to children.
- Option D: Oral doxycycline 14 days — antibacterial, not scabicidal; used for secondary bacterial infection of scabies lesions, not the primary treatment.

Final Answer: Permethrin 5% cream; 8–12 hours; repeat after 1 week; treat all contacts ⇒ A

Answer: (A) Go Back to Q 173

Q174.

Solution

Concept — Primary Syphilis / Penicillin G: Primary syphilis: painless indurated ulcer (chancre) at site of inoculation + non-tender inguinal lymphadenopathy, 9–90 days after exposure. Serology: VDRL reactive (non-treponemal), FTA-ABS positive (treponemal). Treatment of choice: **Benzathine penicillin G 2.4 million units IM as a single dose** (for primary, secondary, or early latent syphilis $<$1 year duration). This is universally recommended by WHO and CDC.

Step 1 — Confirm diagnosis and DOC: Painless ulcer + VDRL 1:32 + FTA-ABS positive = primary syphilis. DOC = benzathine penicillin G 2.4 MU IM single dose.

Why other options are wrong:

- Option A: Azithromycin 1 g single dose — used for chlamydia and gonorrhoea; not recommended for syphilis due to emerging resistance.
- Option B: Benzathine penicillin G 2.4 MU IM single dose — **CORRECT.** Drug of choice for primary syphilis per WHO/CDC/NACO guidelines.
- Option C: Doxycycline 100 mg BD for 14 days — alternative **only** in case of penicillin allergy; not first-line.
- Option D: Ceftriaxone 1 g IM daily for 10 days — alternative for neurosyphilis in penicillin allergy; not first-line for primary syphilis.

Final Answer: Benzathine penicillin G 2.4 million units IM single dose $\Rightarrow$ B

Answer: (B) Go Back to Q 174

Q175.

Solution

Concept — Vitiligo Pathogenesis / Autoimmune: Vitiligo is an acquired depigmentation disorder caused by the **autoimmune destruction of melanocytes**. The primary mechanism involves autoreactive **CD8+ cytotoxic T cells** targeting melanocyte antigens (tyrosinase, TRP-1, TRP-2) leading to melanocyte apoptosis. Autoantibodies against tyrosinase and melanocyte antigens also contribute. It is associated with other autoimmune diseases (thyroid, pernicious anaemia, T1DM).

Step 1 — Identify correct mechanism: Vitiligo = Type IV (cell-mediated) + autoantibodies; CD8+ T-cell-mediated killing of melanocytes.

Why other options are wrong:

- Option A: IgE-mediated mast cell degranulation — Type I hypersensitivity (allergy/urticaria); not vitiligo.
- Option B: Type II hypersensitivity — antibody-mediated cytotoxicity (e.g., autoimmune haemolytic anaemia); while autoantibodies exist in vitiligo, the primary mechanism is CD8+ T-cell mediated.
- Option C: Autoimmune destruction by CD8+ T cells + autoantibodies against tyrosinase — **CORRECT.** Current consensus on vitiligo pathogenesis.
- Option D: Infectious destruction by spirochaete — leukoderma can follow syphilis (secondary syphilis vitiligo-like patches) but that is not the mechanism of idiopathic vitiligo.

Final Answer: CD8+ T-cell-mediated autoimmune destruction of melanocytes + anti-tyrosinase autoantibodies $\Rightarrow$ C

Answer: (C) Go Back to Q 175

Q176.

Solution

Concept — Atopic Dermatitis / Stepwise Treatment: Stepwise management per NICE/ETFAD/EADV: (1) All patients: regular emollients (moisturisers) as the base of all treatment; (2) Flares: topical corticosteroids (low-to-mid potency for children, mid-to-high for adults); (3) Sensitive areas (face, flexures): **topical calcineurin inhibitors (tacrolimus 0.1%/0.03%, pimecrolimus)** to avoid steroid side effects; (4) Severe: dupilumab (biologic anti-IL-4R). Emollients are the cornerstone of **all** steps.

Step 1 — Select stepwise treatment for moderate AD in a child: Regular emollients + mid-potency topical steroid for flares + tacrolimus/pimecrolimus for face and flexures.

Why other options are wrong:

- Option A: Systemic corticosteroids as first-line — not recommended in children due to HPA axis suppression, growth retardation, and rebound flares; reserved for severe refractory AD.
- Option B: Topical tacrolimus only, avoid moisturisers — **incorrect**; emollients are the mandatory foundation of all AD treatment; tacrolimus alone without emollients is suboptimal.
- Option C: Oral antihistamine alone — antihistamines do not reduce AD inflammation; only provide mild sedation for night-time itch (not evidence-

based as primary treatment).

- Option D: Emollients + mid-potency TCS for flares + tacrolimus/pimecrolimus for face/flexures — **CORRECT.** Evidence-based stepwise management for moderate AD in a child.

Final Answer: Emollients + topical corticosteroid for flares + calcineurin inhibitor for face/flexures ⇒ D

Answer: (D) Go Back to Q 176

Q177.

Solution

Concept — Right Upper Lobe Collapse / CXR Signs: Lobar collapse features: opacification (white out), volume loss signs (tracheal shift *toward* the collapse, elevated hemidiaphragm, mediastinal shift). RUL collapse: opacification of right upper zone, elevated right hilum, tracheal deviation to the right, upper mediastinal shift right. Consolidation (pneumonia) has opacification but **no** volume loss (trachea central or displaced away). Effusion has opacification with trachea displaced *away*. Pneumothorax has absent lung markings with pleural line.

Step 1 — Identify pattern: RUZ opacification + elevated hilum + tracheal shift **toward** lesion (right) = volume loss = **collapse/atelectasis**.

Why other options are wrong:

- Option A: RUL collapse (atelectasis) — **CORRECT.** Tracheal shift *toward* the lesion = volume loss = collapse; elevated hilum confirms RUL.
- Option B: Right pleural effusion — trachea would shift *away* (to the left) due to mass effect; no elevated hilum.
- Option C: RUL consolidation (pneumonia) — similar opacification but trachea remains central (no significant volume loss in consolidation); air bronchograms present.
- Option D: Right tension pneumothorax — hyperinflation of right side; trachea shifts *away* (left); absent lung markings lateral to pleural line; no whiteout.

Final Answer: Right upper lobe collapse — tracheal shift toward opacity + elevated hilum = volume loss ⇒ A

Answer: (A) Go Back to Q 177

Q178.

Solution

Concept — Subarachnoid Haemorrhage / CT Brain / Hyperdense Blood: In SAH, extravasated blood in the subarachnoid space appears **hyperdense (bright white)** on non-contrast CT because fresh blood (haematocrit effect) is denser than CSF. This is most visible in the basal cisterns, Sylvian fissures, and subarachnoid spaces. CT sensitivity for SAH within 6 hours is >98%. If CT is negative at 6 hours, LP for xanthochromia is indicated.

Step 1 — Identify correct CT finding: SAH within 6 hours = hyperdense (white) blood in basal cisterns. Not hypodense (that is ischaemia/infarction).

Why other options are wrong:

- Option A: Hypodense area in basal cisterns — hypodense represents **oedema or infarction** (ischaemic stroke); SAH is hyperdense.
- Option B: Hyperdense (bright white) blood in basal cisterns — **CORRECT.** Fresh subarachnoid blood is hyperdense on NCCT; sensitivity >98% within 6 hours.
- Option C: Midline shift — seen in subdural/epidural haematoma or large infarction; not the hallmark of SAH.
- Option D: Ring-enhancing lesion — seen in brain abscess, metastasis, glioblastoma; requires contrast; not SAH on NCCT.

Final Answer: Hyperdense blood in basal cisterns on NCCT within 6 hours of SAH $\Rightarrow$ B

Answer: (B) Go Back to Q 178

Q179.

Solution

Concept — Contrast Media Pre-medication / ACR Protocol: For patients with prior contrast reaction or high-risk (seafood allergy is NOT a reliable predictor, but prior iodinated contrast reaction is), the **ACR-recommended pre-medication protocol** is: **Prednisolone 50 mg orally at 13, 7, and 1 hours before the procedure + diphenhydramine 50 mg IM/IV 1 hour before**. Seafood allergy was historically considered a risk factor but current evidence shows it does not significantly increase contrast reaction risk (the seafood-iodine myth). However, clinical practice often still pre-medicates these patients.

Step 1 — Identify correct protocol: ACR protocol = 3-dose oral prednisolone

(13h, 7h, 1h) + antihistamine 1h before.

Why other options are wrong:

- Option A: Antihistamine alone 1 hour before — insufficient; ACR protocol requires corticosteroid pre-medication (hours before, not just antihistamine).
- Option B: IV adrenaline prophylactically — adrenaline is used to **treat** anaphylaxis, not as prophylaxis; prophylactic adrenaline is not indicated.
- Option C: Prednisolone 50 mg at 13, 7, 1 hours + diphenhydramine 1 hour before — **CORRECT.** Standard ACR pre-medication protocol.
- Option D: Avoid contrast; MRI without contrast equivalent — for most CT-specific indications (e.g., CT urography, aortic dissection evaluation), non-contrast MRI may not provide equivalent information; pre-medication allows safe administration.

Final Answer: Prednisolone 50 mg at 13h, 7h, 1h + diphenhydramine 1h before (ACR protocol) $\Rightarrow$ C

Answer: (C) Go Back to Q 179

Q180.

Solution

Concept — Radiation Dose Limits / AERB / ICRP: The International Commission on Radiological Protection (ICRP) and AERB (India) set occupational dose limits. ICRP Publication 103 (2007): **20 mSv/year averaged over 5 years** (with no single year exceeding 50 mSv). This is the effective dose limit for radiation workers. The general public limit is 1 mSv/year.

Step 1 — Apply correct limit: AERB/ICRP occupational effective dose limit = **20 mSv/year** averaged over 5 years.

Why other options are wrong:

- Option A: 5 mSv/year — this is the limit for designated radiation areas in some older guidelines; not the occupational effective dose limit.
- Option B: 10 mSv/year — some conservative operational practices use this level; not the AERB/ICRP formal limit.
- Option C: 15 mSv/year — no standard guideline uses this specific value.
- Option D: 20 mSv/year — **CORRECT.** ICRP Publication 103 and AERB: 20 mSv/year averaged over 5 consecutive years (maximum 50 mSv in any single year) for occupational workers.

Final Answer: Occupational radiation limit = 20 mSv/year (averaged over 5 years) per AERB/ICRP $\Rightarrow$ D

Answer: (D) Go Back to Q 180

Q181.

Solution

Concept — Primary Spontaneous Pneumothorax / BTS Guidelines: BTS 2010 guidelines for primary spontaneous pneumothorax (PSP): If the pneumothorax is >2 cm (from lung edge to chest wall at hilum on PA CXR) AND the patient is breathless $\Rightarrow$ needle aspiration or intercostal drain (ICD). If small (≤ 2 cm) and not breathless $\Rightarrow$ observe and discharge with follow-up. Here, the rim is 3 cm (at hilum) $\Rightarrow$ large PSP $\Rightarrow$ intervention needed. Needle aspiration is first-line; ICD if aspiration fails.

Step 1 — Apply BTS criteria: 3 cm rim at hilum = large PSP (>2 cm). Breathless $\Rightarrow$ needle aspiration or ICD.

Why other options are wrong:

- Option A: Needle aspiration or ICD for PSP >2 cm — **CORRECT.** BTS guidelines: aspiration (first) or ICD for moderate/large PSP with symptoms.
- Option B: Immediate thoracotomy — reserved for recurrent PSP (pleurodesis/pleurectomy) or complicated haemopneumothorax; not first-line for uncomplicated PSP.
- Option C: 100% O_2 + observe for 4 hours — appropriate only for small PSP (≤ 2 cm) in an asymptomatic patient; this patient is breathless and has a 3 cm rim.
- Option D: Bilateral ICD empirically — pneumothorax is unilateral (right side); bilateral ICD is excessive and dangerous.

Final Answer: Needle aspiration or ICD for large PSP (>2 cm) per BTS guidelines $\Rightarrow$ A

Answer: (A) Go Back to Q 181

Q182.

Solution

Concept — Gallstones / USG / MRCP for CBD Stones: USG is the best initial investigation for gallbladder pathology. Gallstones on USG: hyperechoic focus + posterior acoustic shadowing + mobile with repositioning. Normal GB wall (<3 mm) and CBD (4 mm = normal) suggests uncomplicated cholelithiasis. If bilirubin rises or CBD dilates (suggestive of CBD stones), **MRCP** is the investigation of choice (non-invasive, no radiation, no contrast, >95% sensitivity for CBD stones). ERCP is both diagnostic and therapeutic if stones confirmed.

Step 1 — Identify diagnosis and next step if needed: Hyperechoic + acoustic shadow + mobile = cholelithiasis. If CBD involvement suspected: MRCP.

Why other options are wrong:

- Option A: GB carcinoma + CT — GB carcinoma: irregular wall thickening, mass replacing GB, infiltration of liver; not suggested by this ultrasound.
- Option B: Cholelithiasis + MRCP — **CORRECT.** Classic USG features of gallstones; MRCP is the non-invasive gold standard for evaluating CBD stones.
- Option C: Acute cholecystitis + HIDA scan — cholecystitis requires GB wall thickening (>3 mm), pericholecystic fluid, Murphy's sign on USG; GB wall is 2 mm here (normal).
- Option D: Porcelain GB + plain X-ray — porcelain GB has calcified GB wall visible on X-ray; this USG shows mobile hyperechoic foci, not calcification of the wall.

Final Answer: Cholelithiasis (gallstones); MRCP for evaluation of CBD stones ⇒ B

Answer: (B) Go Back to Q 182

Q183.

Solution

Concept — ASA Physical Status Classification: ASA I: Normal healthy patient. ASA II: Mild systemic disease, no functional limitation (controlled HTN, well-controlled DM, BMI <40, mild lung disease). ASA III: Severe systemic disease with functional limitation (poorly controlled DM, BP, COPD, morbid obesity BMI>40, active hepatitis, pacemaker, ESRD). ASA IV: Constant threat to life.

Step 1 — Classify this patient: Controlled HTN (BP 136/84) + well-controlled DM (HbA1c 7.4%) + BMI 32 + no functional limitation (climbs 2 flights stairs) =

ASA II (two mild, well-controlled systemic diseases; no functional limitation).

Why other options are wrong:

- Option A: ASA I — patient has two controlled systemic diseases (HTN + DM); ASA I requires no systemic disease.
- Option B: ASA II — **CORRECT.** Mild systemic disease (HTN + DM, both well-controlled, no functional limitation).
- Option C: ASA III — would require **severe** functional limitation or poorly controlled disease; both conditions are well-controlled here.
- Option D: ASA IV — constant threat to life (e.g., severe cardiac failure, sepsis, recent MI <3 months); does not apply.

Final Answer: ASA II — mild controlled systemic disease (HTN + DM), no functional limitation $\Rightarrow$ C

Answer: (C) Go Back to Q 183

Q184.

Solution

Concept — Sugammadex / Reversal of Rocuronium: Sugammadex is a modified cyclodextrin that specifically encapsulates steroidal neuromuscular blocking agents (rocuronium $>$ vecuronium). It is the preferred reversal agent for rocuronium because it provides complete, rapid reversal regardless of depth of blockade. Dose for deep block (TOF 0): 16 mg/kg; for moderate (TOF 1–2): 4 mg/kg; for shallow (TOF $\geq$3): 2 mg/kg. Neostigmine (anticholinesterase) is inadequate for deep residual block and requires anticholinergic (glycopyrrolate) to prevent bradycardia.

Step 1 — Select best reversal agent: TOF ratio 0.2 = residual block from rocuronium $\Rightarrow$ sugammadex 4 mg/kg is most effective.

Why other options are wrong:

- Option A: Neostigmine + glycopyrrolate — can reverse moderate-to-shallow rocuronium block but is less complete than sugammadex; neostigmine has a ceiling effect and cannot reliably reverse deep blockade.
- Option B: Edrophonium + atropine — shorter-acting anticholinesterase; used historically; inferior to sugammadex for reversal of rocuronium.
- Option C: Atropine alone — atropine counteracts muscarinic effects of neostigmine but has no neuromuscular reversal activity; cannot reverse NMB.

- Option D: Sugammadex 4 mg/kg IV — **CORRECT.** Encapsulates rocuronium with near-complete reversal; first-line for reversal of steroidal NMBAs.

Final Answer: Sugammadex 4 mg/kg IV — specific reversal agent for rocuronium $\Rightarrow$ D

Answer: (D) Go Back to Q 184

Q185.

Solution

Concept — Guedel's Stages of Anaesthesia: Stage I: Analgesia (conscious, responsive). Stage II: Excitement/delirium (irregular breathing, risk of laryngospasm and vomiting). Stage III: Surgical anaesthesia (Planes 1–4: progressively deepening loss of reflexes). Stage III Plane 1: regular breathing, eyelash reflex absent, eyelid reflex present, laryngeal and pharyngeal reflexes still present. Stage IV: Medullary depression (respiratory arrest, death).

Step 1 — Match description: Regular breathing + unconscious + laryngeal and pharyngeal reflexes intact + eyelash reflex absent = **Stage III, Plane 1**.

Why other options are wrong:

- Option A: Stage III, Plane 1 — **CORRECT.** Laryngeal and pharyngeal reflexes present in Plane 1; as deepening occurs to Plane 2, these reflexes disappear.
- Option B: Stage I — patient is conscious in Stage I; this patient is unconscious.
- Option C: Stage II — characterised by **irregular** breathing, excitement, risk of laryngospasm; the patient described has regular breathing.
- Option D: Stage IV — medullary depression; respiratory arrest; no spontaneous breathing. This patient is breathing regularly.

Final Answer: Stage III, Plane 1 — regular breathing, laryngeal reflexes present (risk of laryngospasm) $\Rightarrow$ A

Answer: (A) Go Back to Q 185

Q186.

Solution

Concept — Minimum Alveolar Concentration (MAC): MAC is defined as the **end-tidal (alveolar) concentration of an inhalational agent (in % atm at 1 atm) that prevents movement in 50% of patients in response to a standard surgical incision**. It is a measure of potency: **higher MAC = lower potency** (more agent needed). MAC decreases with: increasing age, opioids, hypothermia, pregnancy, hypotension, other CNS depressants. MAC increases with: young age, hyperthermia, chronic alcohol use. Nitrous oxide MAC is 105% (very high = low potency, cannot be used alone for surgical anaesthesia).

Step 1 — Identify correct statement: MAC = end-tidal concentration preventing movement in 50%; higher MAC = lower potency; affected by many factors.

Why other options are wrong:

- Option A: MAC prevents movement in 95% (EC_{95}) — **incorrect**; MAC is EC_{50} (50% of patients); EC_{95} would require a higher concentration (approximately 1.3 $\times$ MAC).
- Option B: MAC is end-tidal concentration preventing movement in 50%; higher MAC = lower potency — **CORRECT.** Complete and accurate definition.
- Option C: MAC is independent of age, opioids, temperature — **incorrect**; MAC is significantly influenced by all these factors.
- Option D: MAC of nitrous oxide $<$1% (very potent) — **incorrect**; N_2O MAC = 105% (cannot achieve MAC at 1 atm; very low potency).

Final Answer: MAC = end-tidal concentration preventing movement in 50%; higher MAC = lower potency $\Rightarrow$ B

Answer: (B) Go Back to Q 186

Q187.

Solution

Concept — Spinal Anaesthesia / Hypotension in Obstetrics: Spinal anaesthesia causes sympathetic blockade $\Rightarrow$ vasodilation + hypotension. In pregnancy, the gravid uterus compresses the inferior vena cava (IVC) in supine position (aortocaval compression), reducing venous return and worsening hypotension. Combined with sympathetic block from spinal: profound hypotension. **Management:** Left uterine displacement (left lateral tilt or wedge under right hip) + IV fluid bolus + vasopressor (phenylephrine preferred for obstetric spinal hypotension;

ephedrine if bradycardia present). Both together address the aortocaval and vasodilatory components.

Step 1 — Identify cause and treatment: Hypotension 20 min post-spinal in a pregnant patient = sympathetic blockade + aortocaval compression. Treat with: left lateral tilt + IV ephedrine/phenylephrine + fluid.

Why other options are wrong:

- Option A: High spinal block + intubation — high spinal causes profound hypotension and may affect breathing, but bradycardia 52 bpm and BP 80/50 without apnoea suggests sympathetic block rather than phrenic nerve/diaphragm paralysis; intubation not the first step.
- Option B: Total spinal + O_2 — total spinal (block reaching brainstem) causes respiratory arrest and unconsciousness; this patient is awake with symptoms suggesting moderate sympathetic block.
- Option C: Sympathetic blockade + aortocaval compression; left tilt + ephedrine/phenylephrine + fluids — **CORRECT.** Most common cause of spinal hypotension in obstetrics; combined treatment.
- Option D: PDPH + blood patch — PDPH occurs 24–48 hours after dural puncture; presents as postural headache; no acute haemodynamic compromise.

Final Answer: Sympathetic block + aortocaval compression; left lateral tilt + vasopressor + IV fluid $\Rightarrow$ C

Answer: (C) Go Back to Q 187

Q188.

Solution

Concept — Malignant Hyperthermia (MH): MH is a rare pharmacogenetic disorder triggered by volatile inhalational agents (halothane, isoflurane, sevoflurane, desflurane) and succinylcholine. Caused by a mutation in **RYR1** (ryanodine receptor), leading to uncontrolled release of calcium from sarcoplasmic reticulum $\Rightarrow$ sustained muscle contraction $\Rightarrow$ hyperthermia, rigidity, rhabdomyolysis, mixed acidosis. **Masseter muscle rigidity** after succinylcholine is an early warning sign. Treatment: **Dantrolene sodium** (blocks RYR1) + stop triggering agents + cooling + correct acidosis.

Step 1 — Identify MH: Succinylcholine + halothane + masseter rigidity + rapidly rising temperature + mixed acidosis + elevated CK = **Malignant Hyperthermia**.

Why other options are wrong:

- Option A: NMS + bromocriptine + dantrolene — NMS is caused by dopamine antagonists (antipsychotics); develops over days (not acutely intraoperatively); slower onset; "lead pipe" rigidity; no succinylcholine trigger.
- Option B: Thyroid storm + PTU + beta-blocker — thyroid storm involves thyrotoxic features (high T4, T3); no muscle rigidity; not triggered by anaesthetic agents.
- Option C: Serotonin syndrome + cyproheptadine — serotonin excess from serotonergic drugs; characterised by hyperreflexia, myoclonus, diarrhoea; not triggered by succinylcholine/halothane.
- Option D: Malignant hyperthermia; dantrolene + stop triggers + cooling + correct acidosis — **CORRECT.** Dantrolene 2.5 mg/kg IV is the specific antidote; repeat until rigidity resolves (max 10 mg/kg).

Final Answer: Malignant hyperthermia; dantrolene sodium + stop triggering agents + cooling $\Rightarrow$ D

Answer: (D) Go Back to Q 188

Q189.

Solution

Concept — O_2-Hb Dissociation Curve / Bohr Effect: Alkalosis (increased pH) causes a **left shift** of the Hb-O_2 dissociation curve (Bohr effect reversed). Left shift: haemoglobin binds O_2 more avidly; P_{50} decreases (P_{50} is the PaO_2 at which Hb is 50% saturated — normally 27 mmHg). Left shift $\Rightarrow$ O_2 is held more tightly by Hb $\Rightarrow$ reduced O_2 unloading to tissues. Causes of left shift: alkalosis (high pH), low CO_2, low temperature, low 2,3-DPG, HbF, CO poisoning.

Step 1 — Apply Bohr effect: Respiratory alkalosis (pH 7.52, low $PaCO_2$) $\Rightarrow$ left shift $\Rightarrow$ increased O_2 affinity $\Rightarrow$ P_{50} decreases $\Rightarrow$ less O_2 released to tissues.

Why other options are wrong:

- Option A: Left shift; P_{50} decreases; reduced tissue O_2 unloading — **CORRECT.** Alkalosis causes left shift; Hb holds O_2 more tightly; tissues receive less O_2.
- Option B: Right shift; P_{50} increases — right shift is caused by acidosis (low pH), high CO_2, high temperature, high 2,3-DPG; opposite of alkalosis.
- Option C: No shift — pH changes are the primary driver of the Bohr effect; alkalosis definitively shifts the curve left.

- Option D: Left shift; tissue oxygenation improved — incorrect interpretation; left shift means O_2 is released **less** readily (reduced offloading), so tissue oxygenation is worsened.

Final Answer: Respiratory alkalosis $\Rightarrow$ left shift $\Rightarrow$ P_{50} decreases $\Rightarrow$ reduced tissue O_2 unloading $\Rightarrow$ A

Answer: (A) Go Back to Q 189

Q190.

Solution

Concept — Spinal vs Epidural Anaesthesia: Spinal: single-shot intrathecal injection into CSF (L3/4 or L4/5); fast onset (2–5 min), dense block, predictable; limited duration. Epidural: injection into epidural space; slower onset (15–20 min), can be continuous via catheter, titratable, used for labour analgesia and post-operative pain. Both avoid general anaesthesia and its respiratory risks in COPD. For TURP: spinal anaesthesia is preferred (allows early detection of TURP syndrome symptoms — confusion, nausea in a conscious patient).

Step 1 — Compare spinal vs epidural: Spinal = faster, denser block; epidural = catheter allows continuous dosing and titration; both avoid GA risks in COPD.

Why other options are wrong:

- Option A: Epidural faster than spinal — **incorrect**; spinal has onset 2–5 min; epidural takes 15–20 min; spinal is much faster.
- Option B: Spinal faster + denser; epidural allows continuous dosing; both avoid GA risks in COPD — **CORRECT.** Accurate comparison of the two techniques.
- Option C: Spinal contraindicated with INR <1.2 — **incorrect**; INR <1.2 is normal; spinal is safe; contraindication would be INR >1.4–1.5 or use of anticoagulants.
- Option D: Epidural always preferred for TURP — spinal is commonly used for TURP as it allows the conscious patient to report early symptoms of TURP syndrome.

Final Answer: Spinal = faster, denser; epidural = catheter allows continuous infusion; both avoid GA $\Rightarrow$ B

Answer: (B) Go Back to Q 190

Q191.

Solution

Concept — Post-Operative Nausea and Vomiting (PONV) / Ondansetron Mechanism: Ondansetron is a selective **5-HT_3 (serotonin) receptor antagonist**. It blocks 5-HT_3 receptors in the chemoreceptor trigger zone (CTZ) and vagal afferents. For refractory PONV, a rescue antiemetic from a **different** class should be chosen: haloperidol (D2 antagonist), dexamethasone (corticosteroid — mechanism unclear), or droperidol. Combining two 5-HT_3 antagonists (granisetron) provides no additional benefit.

Step 1 — Identify correct mechanism and rescue agent: Ondansetron = 5-HT_3 antagonist. For rescue: use different class — haloperidol (D2) or dexamethasone.

Why other options are wrong:

- Option A: Ondansetron = D2 antagonist — **incorrect**; metoclopramide/haloperidol/droperidol are D2 antagonists; ondansetron is 5-HT_3; combining with granisetron (also 5-HT_3) adds no benefit.
- Option B: Ondansetron = NK1 antagonist — **incorrect**; aprepitant is the NK1 antagonist; ondansetron is 5-HT_3.
- Option C: Ondansetron = 5-HT_3 antagonist; rescue = haloperidol (D2) or dexamethasone — **CORRECT.** Correct mechanism + appropriate multimodal rescue antiemetic strategy.
- Option D: Ondansetron = H1 antagonist — **incorrect**; cyclizine/promethazine are H1 antagonists; ondansetron is 5-HT_3.

Final Answer: Ondansetron = 5-HT_3 antagonist; rescue with haloperidol (D2) or dexamethasone $\Rightarrow$ C

Answer: (C) Go Back to Q 191

Q192.

Solution

Concept — Contrast-Induced Nephropathy (CIN) Prevention: CIN prevention strategy (ACR/KDIGO): (1) **IV isotonic saline hydration** — most important; 1 mL/kg/hr for 3–12 hours before and after; (2) Use **iso-osmolar or low-osmolar non-ionic** contrast; (3) Minimise contrast volume; (4) Hold **metformin** 48 hours before and restart 48 hours after; (5) Consider acetylcysteine (evidence conflicting; PRESERVE trial negative). Hydration is the single most evidence-based intervention.

Step 1 — Most important prevention measure: IV isotonic saline (or sodium bicarbonate) hydration is the cornerstone of CIN prevention.

Why other options are wrong:

- Option A: Acetylcysteine only — evidence is conflicting; PRESERVE trial showed no benefit; not the most important measure.
- Option B: Stop metformin 48 hours before (only) — important to prevent lactic acidosis in CKD (metformin accumulates), but stopping metformin alone does not prevent CIN; hydration is more critical.
- Option C: High-osmolar (ionic) contrast — high-osmolar contrast **increases** nephrotoxicity risk; iso-osmolar or low-osmolar non-ionic contrast should be used.
- Option D: IV isotonic saline hydration + iso-osmolar/low-osmolar contrast + minimise volume + hold metformin — **CORRECT.** Comprehensive, evidence-based prevention strategy for CIN in high-risk CKD patient.

Final Answer: IV isotonic saline hydration + iso-osmolar contrast + minimise volume + hold metformin $\Rightarrow$ D

Answer: (D) Go Back to Q 192

Q193.

Solution

Concept — Acute Appendicitis / Alvarado Score: Alvarado score $\geq$7 in males = appendicitis likely; proceed to surgery. Score 8 here is strongly positive. Clinical features: migratory pain (epigastrium $\rightarrow$ RIF), tenderness at McBurney's point, rebound tenderness, guarding, positive Rovsing's sign (palpation of LIF causes pain in RIF due to peritoneal irritation), fever, leukocytosis. Treatment: **laparoscopic appendicectomy** (preferred over open).

Step 1 — Confirm diagnosis and treatment: All classic features of acute appendicitis + Alvarado 8 $\Rightarrow$ surgical intervention.

Why other options are wrong:

- Option A: Acute appendicitis + laparoscopic appendicectomy — **CORRECT.** Alvarado 8 in a male with classic features mandates surgery.
- Option B: Perforated peptic ulcer — presents with sudden severe epigastric pain, board-like rigidity, free air under diaphragm on erect CXR; no localisation to RIF.
- Option C: Acute mesenteric ischaemia — pain disproportionate to exami-

nation, older patient with AF/vascular disease; CT angiography appropriate but not for this clinical picture.
- Option D: Renal colic — colicky pain radiating to groin, haematuria; no peritoneal signs; USG/KUB appropriate.

Final Answer: Acute appendicitis; laparoscopic appendicectomy ⇒ A

Answer: (A) Go Back to Q 193

Q194.

Solution

Concept — Kwashiorkor vs Marasmus: Kwashiorkor: **protein deficiency** with adequate calorie intake; oedema (hallmark), hypoalbuminaemia, skin changes (flaky-paint/crazy pavement dermatosis), hair changes (flag sign, easily plucked), hepatomegaly (fatty liver due to impaired lipoprotein synthesis), miserable affect. Marasmus: **total calorie deficiency** (protein + energy); severe wasting (bag of bones), no oedema, alert and hungry. Distinguishing feature: **oedema + hypoalbuminaemia** = kwashiorkor.

Step 1 — Identify diagnosis: Bilateral pitting oedema + flag sign + flaky-paint dermatosis + serum albumin 1.8 g/dL + hepatomegaly = **kwashiorkor**.

Why other options are wrong:

- Option A: Marasmus — severe wasting without oedema; child is alert and hungry; absent from this presentation (oedema is present here).
- Option B: Kwashiorkor + oedema + hypoalbuminaemia + protein deficiency — **CORRECT.** Classic presentation; oedema distinguishes kwashiorkor from marasmus.
- Option C: Marasmic kwashiorkor — combined features of both (severe wasting + oedema); this child has oedema but weight Z-score –3 suggests moderate wasting; however, the predominant features described are kwashiorkor.
- Option D: Nutritional rickets — bowing of legs, rachitic rosary, widening of wrists; no oedema or protein-deficiency features.

Final Answer: Kwashiorkor — nutritional oedema + hypoalbuminaemia + protein deficiency ⇒ B

Answer: (B) Go Back to Q 194

Q195.

Solution

Concept — Epidemic Curve / Point-Source vs Propagated: Epidemic curve (epi curve) patterns: (1) **Point-source common-vehicle**: single exposure event; sharp symmetric bell-shaped curve; rise and fall within one incubation period. (2) Propagated (person-to-person): successive waves; each peak separated by one incubation period; gradual decline. (3) Continuous common-source: prolonged exposure; plateau or prolonged rise. (4) Mixed: features of both. A wedding function meal = single-point exposure ⇒ point-source common-vehicle epidemic.

Step 1 — Identify curve pattern: Sharp peak at 4–5 hours, rapid rise and fall within hours, all exposure at one event (wedding meal) = **point-source common-vehicle**.

Why other options are wrong:

- Option A: Propagated epidemic — successive peaks; cases generate cases; not seen in a food poisoning scenario with single-event exposure.
- Option B: Continuous common-source — prolonged plateau; seen when exposure to contaminated water supply continues over days/weeks.
- Option C: Point-source common-vehicle — **CORRECT.** Single exposure at the wedding feast; sharp peak within one incubation period of the causative agent.
- Option D: Mixed epidemic — combination features; not applicable when all cases trace to a single event.

Final Answer: Point-source common-vehicle epidemic — single exposure event, sharp peak ⇒ C

Answer: (C) Go Back to Q 195

Q196.

Solution

Concept — UIP India / Birth Dose Vaccines: Under India's Universal Immunisation Programme (UIP), the vaccines given at birth are: (1) **BCG** (single dose, left deltoid), (2) **OPV-0** (zero dose of oral polio vaccine, first dose given at birth), (3) **Hepatitis B birth dose** (within 24 hours of birth). Vitamin K (not a vaccine) is given to newborns as prophylaxis against haemorrhagic disease of newborn, but it is not under the UIP schedule.

Step 1 — Identify correct birth dose schedule: BCG + OPV0 + Hep B (birth

dose) = three vaccines given at birth under UIP.

Why other options are wrong:

- Option A: BCG + OPV0 + Hep B + Vitamin K — Vitamin K is a medication, not a vaccine under UIP; the three UIP birth-dose vaccines are BCG, OPV0, Hep B.
- Option B: OPV0 + DPT only — DPT is given at 6, 10, 14 weeks (not at birth); BCG and Hep B are missing.
- Option C: BCG + Rotavirus — Rotavirus vaccine is given at 6, 10, 14 weeks; not at birth.
- Option D: BCG + OPV0 + Hepatitis B birth dose — **CORRECT.** Three vaccines under UIP given at or within 24 hours of birth.

Final Answer: BCG + OPV0 + Hepatitis B birth dose — three UIP vaccines given at birth $\Rightarrow$ D

Answer: (D) Go Back to Q 196

Q197.

Solution

Concept — Haemophilia A / Diagnosis / Treatment: Haemophilia A: X-linked recessive; Factor VIII deficiency; presents with haemarthrosis, deep tissue bleeds. Labs: prolonged aPTT (intrinsic pathway), normal PT, normal platelet count and bleeding time. Factor VIII activity $<1\%$ = severe haemophilia A (spontaneous bleeds). Treatment: **Recombinant Factor VIII concentrate** (preferred over plasma-derived). DDAVP (desmopressin) is for mild haemophilia A only (releases stored FVIII from endothelium) — not effective in severe haemophilia.

Step 1 — Confirm diagnosis and treatment: Prolonged aPTT + normal PT + haemarthrosis + FVIII 2% = severe Haemophilia A. Treatment = recombinant FVIII.

Why other options are wrong:

- Option A: Haemophilia A; recombinant FVIII — **CORRECT.** FVIII 2% confirms severe Haemophilia A; recombinant FVIII concentrate is the treatment of choice.
- Option B: Haemophilia B; recombinant FIX — Haemophilia B is Factor IX deficiency; labs would show reduced FIX activity; clinically identical to Haemophilia A but different factor.
- Option C: von Willebrand disease; DDAVP — vWD: prolonged bleeding time

+ platelet dysfunction + mildly prolonged aPTT; DDAVP for type 1 vWD; FVIII activity would not be 2% in isolation.
- Option D: ITP; IV immunoglobulin — ITP has low platelet count + prolonged bleeding time; no joint bleeds; normal aPTT and PT.

Final Answer: Haemophilia A; recombinant Factor VIII concentrate ⇒ A

Answer: (A) Go Back to Q 197

Q198.

Solution

Concept — Uncomplicated Lower UTI / Treatment: Uncomplicated lower UTI (cystitis) in a non-pregnant, non-diabetic, immunocompetent woman with no structural abnormality or prior UTI <3 months = **short course antibiotic**. First-line options: **nitrofurantoin** 100 mg (modified-release) BD for 5 days, or **trimethoprim** 200 mg BD for 7 days, or TMP-SMX for 3 days. Fluoroquinolones are not first-line for uncomplicated cystitis (reserved for complicated UTI or pyelonephritis) to preserve effectiveness and reduce resistance.

Step 1 — Classify UTI and select treatment: Uncomplicated lower UTI (no fever, no flank pain, no prior UTI) = short-course oral antibiotic; nitrofurantoin or TMP-SMX.

Why other options are wrong:

- Option A: IV ceftriaxone 14 days — appropriate for complicated UTI/bacteraemia, not uncomplicated cystitis; IV route unnecessary here.
- Option B: Nitrofurantoin 5 days or TMP-SMX 3 days — **CORRECT.** First-line agents for uncomplicated lower UTI per NICE/IDSA guidelines.
- Option C: Ciprofloxacin 14 days — appropriate for pyelonephritis, not simple cystitis; fluoroquinolones are reserved; 14 days is excessive even for pyelonephritis (standard 7 days).
- Option D: No treatment — lower UTI in a symptomatic patient with positive urinalysis requires antibiotic treatment; will not resolve spontaneously in most cases and risks ascending infection.

Final Answer: Nitrofurantoin 100 mg BD × 5 days or TMP-SMX 3 days (uncomplicated lower UTI) ⇒ B

Answer: (B) Go Back to Q 198

Q199.

Solution

Concept — Multiple Sclerosis / MRI Findings / McDonald Criteria: MS diagnosis requires demonstration of **dissemination in space (DIS)** and **dissemination in time (DIT)** by clinical or MRI criteria (McDonald 2017). MRI hallmarks: periventricular **T2/FLAIR hyperintense lesions** (ovoid, perpendicular to corpus callosum = **Dawson's fingers**), juxtacortical, infratentorial, and spinal cord lesions. Active lesions enhance with gadolinium. Dawson's fingers: periventricular lesions extending perpendicular to the lateral ventricles (following perivenular inflammation).

Step 1 — Identify MS-specific MRI finding: Multiple periventricular T2/FLAIR lesions (Dawson's fingers) + Gd-enhancing lesion (active demyelination) + DIS + DIT = MS-specific.

Why other options are wrong:

- Option A: Single enhancing cerebellar lesion — single lesion does not satisfy DIS; could be tumour, abscess, or demyelination.
- Option B: Diffuse cortical atrophy — non-specific; seen in ageing, dementia, prolonged epilepsy; not diagnostic of MS.
- Option C: Multiple periventricular T2/FLAIR lesions (Dawson's fingers) + Gd-enhancing lesion(s) + DIS + DIT — **CORRECT.** Pathognomonic MRI pattern for MS per McDonald 2017 criteria.
- Option D: Basal ganglia T1 hyperintense lesions — suggests manganese deposition (hepatic failure), haemorrhage, or calcification; not MS.

Final Answer: Periventricular Dawson's fingers (T2/FLAIR) + Gd-enhancing lesion = MS pattern $\Rightarrow$ C

Answer: (C) Go Back to Q 199

Q200.

Solution

Concept — Multimodal Analgesia / Opioid-Sparing Strategy: Multimodal analgesia uses drugs with **different mechanisms of action** simultaneously to provide better pain control with fewer opioid-related side effects (nausea, respiratory depression, constipation, ileus). Components: (1) Regular paracetamol (COX-3 centrally); (2) NSAID (COX-1/2 peripherally + centrally); (3) Regional technique (local anaesthetic — neuraxial or wound infiltration); (4) Low-dose opioid for breakthrough. This is the ERAS (Enhanced Recovery After Surgery) approach.

Step 1 — Identify multimodal approach: Regular paracetamol IV + NSAID (ketorolac) + epidural/local anaesthetic infiltration + low-dose opioid PRN = multimodal opioid-sparing.

Why other options are wrong:

- Option A: IV morphine PCA alone — unimodal opioid analgesia; highest opioid consumption; maximum side effects; not multimodal.
- Option B: IV paracetamol alone (regular 4-hourly) — single agent; insufficient for post-operative pain after major abdominal surgery; pain score 8/10 is severe.
- Option C: IM diclofenac PRN only — single NSAID on an as-needed basis; inadequate for severe pain; not multimodal; PRN dosing leads to breakthrough pain.
- Option D: Paracetamol IV + NSAID + epidural/wound infiltration + low-dose opioid PRN — **CORRECT.** True multimodal approach; combines four different analgesic mechanisms; minimises opioid use.

Final Answer: Multimodal analgesia = paracetamol + NSAID + regional technique + low-dose opioid PRN $\Rightarrow$ D

Answer: (D) Go Back to Q 200

Q201.

Solution

Concept — Thyroid surgical anatomy: The recurrent laryngeal nerve (RLN) runs in the tracheoesophageal groove and enters the larynx posterior to the cricothyroid joint. It innervates all intrinsic laryngeal muscles except cricothyroid. Injury causes hoarseness (unilateral) or aphonia/stridor (bilateral).

Step 1 — Identify the at-risk structure: The RLN is the nerve most commonly damaged during thyroid surgery, causing hoarseness. It is in the immediate operative field, particularly near the inferior thyroid artery and Berry's ligament.

Why other options are wrong:

- Option A: Correct — RLN injury is the most common cause of post-thyroidectomy hoarseness.
- Option B: External branch of SLN — injury causes loss of high-pitched phonation (singer's nerve), not frank hoarseness.
- Option C: Internal branch of SLN — sensory to supraglottis; injury leads to aspiration, not hoarseness.

- Option D: Superior thyroid artery — a vascular structure; ligation causes ischaemia to upper pole, but does not cause hoarseness directly.

Final Answer: Recurrent laryngeal nerve $\Rightarrow$ A

Answer: (A) Go Back to Q 201

Q202.

Solution

Concept — Thyroid carcinoma histology: Papillary thyroid carcinoma (PTC) is the most common thyroid malignancy, characterised by Orphan Annie eye nuclei (ground-glass, empty-looking), nuclear grooves, pseudoinclusions, and psammoma bodies (laminated calcifications).

Step 1 — Match histology to type: Ground-glass nuclei + nuclear grooves + psammoma bodies = classic PTC. Normal calcitonin rules out medullary carcinoma.

Why other options are wrong:

- Option A: Follicular carcinoma — shows follicular pattern, vascular/capsular invasion; no psammoma bodies or nuclear grooves.
- Option B: Correct — Papillary carcinoma with all classic features.
- Option C: Medullary carcinoma — calcitonin-secreting, amyloid stroma; calcitonin here is normal.
- Option D: Anaplastic — undifferentiated, rapidly fatal; no such specific cytology.

Final Answer: Papillary carcinoma $\Rightarrow$ B

Answer: (B) Go Back to Q 202

Q203.

Solution

Concept — Breast cancer TNM staging: Tumour 4 cm = T2 (2–5 cm). Four positive axillary nodes = N2a (4–9 ipsilateral axillary nodes positive). No distant metastases = M0. T2N2M0 = Stage IIIA.

Step 1 — Apply TNM: T2 (>2 cm, ≤ 5 cm) + N2a (4–9 positive nodes) + M0 $\rightarrow$ Stage IIIA per AJCC 8th edition.

Why other options are wrong:

- Option A: Stage IIA = T0–2 N1 M0 or T2 N0 M0; N here is N2a, not N1.
- Option B: Stage IIB = T2N1M0 or T3N0M0; incorrect node count here.
- Option C: Correct — Stage IIIA includes T2N2M0 and T3N1–2M0.
- Option D: Stage IIIB = T4(any)N(any)M0 or any T N3M0; tumour is T2, not T4.

Final Answer: Stage IIIA $\Rightarrow$ C

Answer: (C) Go Back to Q 203

Q204.

Solution

Concept — FNAC breast cytology grading: The National Health Service Breast Screening Programme (NHSBSP) cytology grading: C1=inadequate, C2=benign, C3=atypia (probably benign), C4=suspicious of malignancy, C5=definite malignancy.

Step 1 — Grade C5: C5 = definite malignancy on cytology; requires surgical excision/definitive treatment. It does not need histological confirmation before surgery in triple assessment context.

Why other options are wrong:

- Option A: C1 = inadequate; C2 = benign (fibroadenoma).
- Option B: C3 = atypical, possibly benign.
- Option C: C4 = suspicious of malignancy.
- Option D: Correct — C5 = definite malignancy.

Final Answer: Definite malignancy $\Rightarrow$ D

Answer: (D) Go Back to Q 204

Q205.

Solution

Concept — Post-mastectomy complications: Seroma is the most common complication of mastectomy, occurring in up to 80% of cases. It is a collection of serous fluid in the dead space following lymphatic and tissue disruption. It presents as a painless, fluctuant swelling, typically 1–4 weeks post-op, without erythema.

Step 1 — Differentiate complications: Seroma = painless fluctuant swelling in axilla, no erythema, 1–4 weeks post-op. Haematoma = early (<24–48 h), painful, tense. Dehiscence = wound edge separation. Lymphangitis = painful, red streaks.

Why other options are wrong:

- Option A: Correct — seroma fits the clinical picture (painless, fluctuant, axillary, 2 weeks post-op, no erythema).
- Option B: Haematoma — presents early, acutely, with pain and tense swelling.
- Option C: Wound dehiscence — visible wound opening, not a fluctuant swelling.
- Option D: Lymphangitis — painful, red streaking lymphatics, fever.

Final Answer: Seroma $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 205

Q206.

Solution

Concept — Appendicitis management: Classic acute appendicitis with high clinical probability (Alvarado/MANTRELS score) in a young male warrants emergency appendicectomy as the definitive treatment. CT is useful when diagnosis is uncertain but delays surgery in clear-cut cases.

Step 1 — Assess clinical probability: Migration of pain to RIF + tenderness at McBurney's + Rovsing's positive + fever + leukocytosis = high clinical probability. Surgery is the next step, not more imaging.

Why other options are wrong:

- Option A: CT useful when diagnosis uncertain (e.g., atypical presentation, female of childbearing age), but delays definitive treatment in clear-cut cases.
- Option B: Observation appropriate for Alvarado score 4–6 (equivocal), not for high-probability cases.

- Option C: Correct — emergency appendicectomy is the treatment of choice.
- Option D: Colonoscopy contraindicated in acute appendicitis.

Final Answer: Emergency appendicectomy ⇒ B

Answer: (B) Go Back to Q 206

Q207.

Solution

Concept — Small bowel obstruction: Post-operative adhesions are the most common cause of small bowel obstruction (SBO) worldwide, accounting for 60–75% of cases. Previous laparotomy is the key risk factor.

Step 1 — Identify cause: Colicky pain + absolute constipation + central ladder pattern X-ray (small bowel) + prior laparotomy history = adhesive SBO.

Why other options are wrong:

- Option A: Colorectal carcinoma — causes large bowel obstruction; presents with peripheral frame-like pattern on X-ray.
- Option B: Femoral hernia — can cause obstruction, but tender groin lump would be present.
- Option C: Correct — adhesions from prior surgery are the most common cause.
- Option D: Sigmoid volvulus — shows a coffee-bean sign (large bowel), not central ladder pattern.

Final Answer: Adhesions from prior surgery ⇒ C

Answer: (C) Go Back to Q 207

Q208.

Solution

Concept — Colorectal cancer screening: For average-risk individuals aged 50+, colonoscopy every 10 years is considered the gold standard screening test as it detects and removes polyps simultaneously. It is preferred over FOBT or sigmoidoscopy for comprehensive screening.

Step 1 — Apply screening guidelines: Average-risk male, age 58, asymptomatic. Colonoscopy every 10 years starting at age 45–50 is the recommended preferred method per ACS/ACG guidelines.

Why other options are wrong:

- Option A: DRE alone detects only very low rectal lesions; not adequate for CRC screening.
- Option B: Flexible sigmoidoscopy every 5 years is acceptable but evaluates only the left colon; colonoscopy is comprehensive.
- Option C: Annual FOBT is an acceptable option but requires annual compliance; colonoscopy preferred as a single test.
- Option D: Correct — Colonoscopy every 10 years from age 45–50 for average risk.

Final Answer: Colonoscopy every 10 years $\Rightarrow$ D

Answer: (D) Go Back to Q 208

Q209.

Solution

Concept — Direct vs indirect inguinal hernia: A direct inguinal hernia passes through Hesselbach's triangle (bounded by inferior epigastric vessels laterally, inguinal ligament inferiorly, lateral border of rectus medially). It is medial to the inferior epigastric vessels and does not descend into the scrotum.

Step 1 — Classify hernia: Medial to inferior epigastric vessels + doesn't descend into scrotum + empty deep ring = direct hernia $\rightarrow$ passes through posterior wall of inguinal canal (Hesselbach's triangle).

Why other options are wrong:

- Option A: Deep inguinal ring is the entry point for an indirect hernia, which is lateral to inferior epigastric vessels and may descend into scrotum.
- Option B: Femoral canal — femoral hernia, below and lateral to pubic tubercle.
- Option C: Correct — Direct hernia passes through Hesselbach's triangle/posterior wall of inguinal canal.
- Option D: Superficial ring alone is not where any hernia originates; all inguinal hernias pass through it on exit.

Final Answer: Hesselbach's triangle (posterior wall) $\Rightarrow$ C

Answer: (C) Go Back to Q 209

Q210.

Solution

Concept — Femoral hernia: Femoral hernia passes through the femoral canal — below and lateral to the pubic tubercle (unlike inguinal hernia which is above and medial to pubic tubercle). It is more common in multiparous women and has a high risk of strangulation due to the rigid femoral ring.

Step 1 — Identify features: Below and lateral to pubic tubercle + tense irreducible swelling + colicky pain + vomiting + multiparous woman = strangulated femoral hernia.

Why other options are wrong:

- Option A: Lymphadenopathy — multiple, non-tender, compressible nodes; no bowel symptoms.
- Option B: Correct — strangulated femoral hernia with obstruction.
- Option C: Obturator hernia — medial thigh, Howship-Romberg sign; rare.
- Option D: Saphena varix — soft, reducible on coughing, disappears on elevation; no acute symptoms.

Final Answer: Strangulated femoral hernia $\Rightarrow$ B

Answer: (B) Go Back to Q 210

Q211.

Solution

Concept — Perforated peptic ulcer: Free air under the diaphragm on erect CXR confirms hollow viscus perforation (pneumoperitoneum). With a history of peptic ulcer disease, this is perforated peptic ulcer — a surgical emergency requiring immediate laparotomy, oversewing of the perforation, and peritoneal lavage (Oodles procedure / Graham's patch).

Step 1 — Emergency management: Pneumoperitoneum + peritonitis + PUD history = perforated peptic ulcer. Treatment: resuscitation + emergency laparotomy. Endoscopy is contraindicated in perforation.

Why other options are wrong:

- Option A: PPI and NG tube are supportive measures only; perforation requires surgery.
- Option B: H. pylori eradication — appropriate after acute phase management, not in acute perforation.

- Option C: Correct — Emergency laparotomy with peritoneal lavage and closure of perforation.
- Option D: Endoscopy is absolutely contraindicated with pneumoperitoneum.

Final Answer: Emergency laparotomy with peritoneal lavage ⇒ C

Answer: (C) Go Back to Q 211

Q212.

Solution

Concept — Acute cholecystitis investigation: Ultrasound of the abdomen is the first-line imaging for suspected biliary pathology. It detects gallstones (sensitivity >95%), gallbladder wall thickening (>3 mm), pericholecystic fluid, and a sonographic Murphy's sign. It is rapid, non-invasive, and inexpensive.

Step 1 — Choose best initial investigation: RUQ pain + Murphy's sign + mildly raised bilirubin and ALP + fever = acute cholecystitis. Ultrasound is the first-line imaging.

Why other options are wrong:

- Option A: CT useful for complications (abscess, perforation) but not first-line for cholecystitis.
- Option B: HIDA scan useful when USS is equivocal; requires more time and cost.
- Option C: MRCP is for CBD stones/strictures evaluation, not first-line for acute cholecystitis.
- Option D: Correct — Abdominal ultrasound is the best initial investigation.

Final Answer: Ultrasound of the abdomen ⇒ D

Answer: (D) Go Back to Q 212

Q213.

Solution

Concept — Choledocholithiasis management: Charcot's triad (RUQ pain + jaundice + fever) with dilated CBD and elevated liver enzymes confirms choledocholithiasis. ERCP is the treatment of choice — it is both diagnostic and therapeutic (sphincterotomy + stone extraction).

Step 1 — Definitive management: CBD stones + obstructive jaundice + cholangitis = ERCP with sphincterotomy is the gold standard. Open surgery has much higher morbidity.

Why other options are wrong:

- Option A: Correct — ERCP with sphincterotomy and stone extraction is first-line.
- Option B: Open cholecystectomy treats gallbladder but does not remove CBD stones endoscopically.
- Option C: PTC used when ERCP fails or not available; more invasive.
- Option D: MRCP is diagnostic only; does not treat CBD stones.

Final Answer: ERCP with sphincterotomy and stone extraction $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 213

Q214.

Solution

Concept — Variceal bleeding pharmacotherapy: Terlipressin (triglycyl-lysine vasopressin) is a vasopressin analogue that causes splanchnic vasoconstriction, reducing portal pressure and controlling variceal bleeding. It is the only vasoactive drug shown to reduce 7-day mortality and is the first-line pharmacological agent.

Step 1 — Immediate pharmacology: Active variceal bleed $\rightarrow$ terlipressin or somatostatin/octreotide for immediate vasoconstriction. Propranolol is for secondary prophylaxis, not acute bleed.

Why other options are wrong:

- Option A: Propranolol — non-selective beta-blocker for primary/secondary prophylaxis; not for acute bleeding.
- Option B: Correct — Terlipressin is the most effective immediate pharmacological agent.
- Option C: Spironolactone — for ascites management; not for variceal bleed.

- Option D: Omeprazole — for peptic ulcer bleeding; not for variceal bleeding.

Final Answer: Terlipressin ⇒ B

Answer: (B) Go Back to Q 214

Q215.

Solution

Concept — Courvoisier's sign: Courvoisier's law states that in obstructive jaundice, if the gallbladder is palpable and non-tender, it is unlikely to be due to gallstones (because gallstones cause chronic fibrosis of the GB). A distended, non-tender gallbladder with painless jaundice suggests carcinoma of the head of pancreas or periampullary tumour.

Step 1 — Identify eponymous sign: Painless progressive jaundice + palpable non-tender gallbladder + pancreatic head mass = Courvoisier's sign (Courvoisier's law).

Why other options are wrong:

- Option A: Charcot's sign (Charcot's triad) = RUQ pain + jaundice + fever in cholangitis.
- Option B: Grey Turner's sign = flank ecchymosis in pancreatitis.
- Option C: Correct — Courvoisier's sign (palpable non-tender gallbladder + painless jaundice).
- Option D: Trousseau's sign = carpopedal spasm in hypocalcaemia; also Trousseau's sign of malignancy = migratory thrombophlebitis.

Final Answer: Courvoisier's sign ⇒ C

Answer: (C) Go Back to Q 215

Q216.

Solution

Concept — Trendelenburg test (tourniquet test): The Trendelenburg test assesses saphenofemoral junction (SFJ) competence. The leg is elevated, then a tourniquet is placed at the SFJ level. The patient stands — if veins refill from below with tourniquet on, perforators are incompetent. If veins refill only after the tourniquet is released, the SFJ is incompetent.

Step 1 — Interpret test result: Tourniquet at SFJ level prevents reflux → veins

stay collapsed → incompetence is at the SFJ (when releasing the tourniquet causes rapid filling). SFJ incompetence is the most common site.

Why other options are wrong:

- Option A: Saphenopopliteal junction incompetence — short saphenous vein involvement; tourniquet placed at popliteal fossa.
- Option B: Perforator incompetence — veins refill from below even with SFJ tourniquet on.
- Option C: Deep femoral incompetence — not tested by this manoeuvre.
- Option D: Correct — tourniquet at SFJ level positive = SFJ incompetence.

Final Answer: Saphenofemoral junction incompetence ⇒ D

Answer: (D) Go Back to Q 216

Q217.

Solution

Concept — DVT diagnosis: Compression duplex ultrasonography is the gold standard non-invasive diagnostic test for DVT. It combines B-mode ultrasound with Doppler assessment and has sensitivity >95% and specificity >99% for proximal DVT. It is preferred for high-probability cases (Wells score ≥2).

Step 1 — Best investigation: High Wells score (3) + post-operative patient = compression duplex ultrasound is the first-line imaging for confirmation.

Why other options are wrong:

- Option A: Correct — Compression duplex ultrasonography is the gold standard.
- Option B: D-dimer — high sensitivity but low specificity; used to rule OUT DVT in low-probability cases; false positive post-surgery.
- Option C: Venography — gold standard historically but invasive; rarely used now.
- Option D: CT pulmonary angiography — investigates pulmonary embolism, not DVT.

Final Answer: Compression duplex ultrasonography ⇒ A

Answer: (A) Go Back to Q 217

Q218.

Solution

Concept — Fontaine classification of PAD: Stage I: asymptomatic; Stage IIa: claudication >200 m; Stage IIb: claudication <200 m; Stage III: rest pain; Stage IV: ulceration or gangrene. ABI 0.55 indicates moderate-severe PAD (ABI <0.9 = abnormal; <0.4 = severe/critical ischaemia).

Step 1 — Classify: Calf pain at 200 metres (claudication distance = 200 m) = Stage IIb (claudication at $\leq$200 m). ABI 0.55 confirms significant PAD.

Why other options are wrong:

- Option A: Stage I = asymptomatic; this patient has claudication.
- Option B: Correct — Stage IIb = claudication at $\leq$200 m. (IIa is >200 m, IIb is $\leq$200 m).
- Option C: Stage III = rest pain; this patient's pain only on walking.
- Option D: Stage IV = ulceration/gangrene; none present.

Final Answer: Fontaine Stage IIb $\Rightarrow$ B

Answer: (B) Go Back to Q 218

Q219.

Solution

Concept — Ruptured abdominal aortic aneurysm (AAA): Ruptured AAA presents with the classic triad: sudden severe abdominal/back pain + pulsatile abdominal mass + haemodynamic shock. This is a surgical emergency with mortality >80% without immediate operation. Haemodynamic instability (BP 90/60, HR 120) mandates immediate surgery without delay for imaging.

Step 1 — Emergency pathway: Ruptured AAA + haemodynamic instability $\rightarrow$ IMMEDIATE open surgical repair (or EVAR if available and haemodynamically stable briefly). CT angiography should not delay surgery in the unstable patient.

Why other options are wrong:

- Option A: CT angiography appropriate only if haemodynamically stable; delays surgery in instability.
- Option B: IV fluids alone without surgery will not save this patient; however, permissive hypotension while organising theatre is appropriate, but surgery is the definitive answer.
- Option C: Correct — Emergency open surgical repair is life-saving and im-

mediately required.

- Option D: Elective EVAR — not appropriate for a ruptured, haemodynamically unstable patient.

Final Answer: Emergency open surgical repair ⇒ C

Answer: (C) Go Back to Q 219

Q220.

Solution

Concept — Burst abdomen: Complete wound dehiscence (burst abdomen / evisceration) is a full-thickness disruption of the wound with protrusion of abdominal contents. It typically occurs 7–10 days post-op, preceded by serosanguinous (salmon-pink) discharge, and is associated with obesity, diabetes, malnutrition, wound infection, and excessive coughing.

Step 1 — Classify complication: Serosanguinous discharge on day 7 + visible bowel loops through wound = complete wound dehiscence with evisceration (burst abdomen).

Why other options are wrong:

- Option A: Seroma — fluid collection, no evisceration; does not involve bowel loops.
- Option B: Haematoma — blood collection; would be tense, dark, not visible bowel.
- Option C: Anastomotic leak — faecal/peritoneal contamination from internal anastomosis; different presentation.
- Option D: Correct — Burst abdomen with evisceration.

Final Answer: Burst abdomen (complete wound dehiscence with evisceration) ⇒ D

Answer: (D) Go Back to Q 220

Q221.

Solution

Concept — Rectal cancer TNM staging: T2 = invasion of muscularis propria (not beyond). N1 = 1–3 positive regional lymph nodes. M0 = no distant metastasis. T2N1M0 = Stage IIIA (N1 upstages even T2 tumours to Stage III).

Step 1 — Apply TNM: Invades muscularis propria = T2. One positive mesorectal node = N1. No distant metastases = M0 → T2N1M0 = Stage IIIA.

Why other options are wrong:

- Option A: Correct — T2N1M0 is Stage IIIA.
- Option B: T3N0M0 = Stage IIA (tumour beyond muscularis propria, no nodes); this tumour is T2.
- Option C: T4aN1M0 = Stage IIIC (T4a = perforation through visceral peritoneum); not applicable here.
- Option D: T2N0M0 = Stage I; but here node is positive (N1).

Final Answer: T2N1M0 — Stage IIIA ⇒ A

Answer: (A) Go Back to Q 221

Q222.

Solution

Concept — Colostomy vs ileostomy: A colostomy (usually sigmoid) produces formed or semi-formed stool and is flush with the skin surface. An ileostomy produces liquid, caustic effluent (high enzyme content) and requires a raised spout (2–3 cm) to protect the peristomal skin from enzymatic damage.

Step 1 — Characteristic of colostomy: Colostomy = flush with skin, formed stool, located in left iliac fossa. Ileostomy = spouted, liquid output, in right iliac fossa.

Why other options are wrong:

- Option A: Raised spout is characteristic of ileostomy, not colostomy.
- Option B: Correct — Colostomy is flush with skin and produces formed stool.
- Option C: Higher output and electrolyte imbalance are features of ileostomy.
- Option D: Ileostomy is typically in the right iliac fossa; sigmoid colostomy is in the left iliac fossa.

Final Answer: Flush with skin, producing formed stool ⇒ B

Answer: (B) Go Back to Q 222

Q223.

Solution

Concept — Antenatal booking visit investigations: At the first ANC visit: Blood group + Rh typing, CBC, VDRL, HIV, urine culture, rubella serology, HBsAg, Pap smear. The glucose challenge test (GCT) is offered at 24–28 weeks to screen for gestational diabetes, not at the first visit (10 weeks).

Step 1 — Timing of GCT: GCT/OGTT for GDM is done at 24–28 weeks of gestation (or earlier if high risk). At 10 weeks, it is too early to screen for GDM. The question asks what is NOT routinely done at first visit.

Why other options are wrong:

- Option A: Blood group and Rh typing — routinely done at booking.
- Option B: VDRL — routinely done at booking to screen for syphilis.
- Option C: Correct — GCT is NOT done at the first visit (10 weeks); it is offered at 24–28 weeks.
- Option D: HIV testing — mandatory at booking under PPTCT programme.

Final Answer: Glucose challenge test at first visit $\Rightarrow$ C

Answer: (C) Go Back to Q 223

Q224.

Solution

Concept — Placenta praevia management: Central (Grade IV) placenta praevia causing antepartum haemorrhage at 34 weeks requires hospitalisation. Digital vaginal examination is absolutely contraindicated (risk of massive haemorrhage). Management: bed rest, IV access, crossmatch, betamethasone for fetal lung maturity if preterm, and planned LSCS.

Step 1 — Emergency management: Painless APH + central placenta praevia + 34 weeks = hospitalise, avoid DRE, plan for LSCS. Tocolysis not appropriate with active bleeding from praevia.

Why other options are wrong:

- Option A: Digital vaginal examination ABSOLUTELY contraindicated — risks massive haemorrhage.

- Option B: Induction of labour contraindicated in central placenta praevia.
- Option C: Tocolysis may worsen bleeding; not the priority management.
- Option D: Correct — Hospitalisation, bed rest, IV access, crossmatch, and plan for LSCS.

Final Answer: Hospitalisation, bed rest, IV access, crossmatch, plan for LSCS ⇒ D

Answer: (D) Go Back to Q 224

Q225.

Solution

Concept — Pre-eclampsia seizure prophylaxis: Magnesium sulphate (MgSO4) is the drug of choice for eclampsia prophylaxis and treatment. It works as an anticonvulsant by blocking NMDA receptors and causing cerebral vasodilation. The Pritchard regime is used most commonly. HELLP syndrome features confirm severe disease.

Step 1 — Immediate drug: BP 170/115 + 3+ proteinuria + HELLP = severe pre-eclampsia. Magnesium sulphate IV is the standard immediate anticonvulsant. Delivery planning follows.

Why other options are wrong:

- Option A: Correct — Magnesium sulphate is the drug of choice for prevention and treatment of eclamptic seizures.
- Option B: Phenobarbitone — historically used, now superseded by MgSO4; not first-line.
- Option C: Diazepam — inferior to MgSO4 for eclampsia; causes neonatal respiratory depression.
- Option D: Nifedipine alone — antihypertensive only; does not prevent seizures.

Final Answer: Magnesium sulphate IV ⇒ A

Answer: (A) Go Back to Q 225

Q226.

Solution

Concept — GDM diagnosis (IADPSG 2010): GDM is diagnosed by 75 g OGTT at 24–28 weeks if ANY one value meets or exceeds: Fasting $\geq$5.1 mmol/L, OR 1-hour $\geq$10.0 mmol/L, OR 2-hour $\geq$8.5 mmol/L. The patient's 1-hour value (10.5 mmol/L) exceeds 10.0 mmol/L, confirming GDM.

Step 1 — Apply IADPSG thresholds: Patient: 1-hour 10.5 mmol/L $\geq$ 10.0 mmol/L threshold $\rightarrow$ GDM diagnosed. The IADPSG criteria use any single abnormal value.

Why other options are wrong:

- Option A: Correct — IADPSG criteria: fasting $\geq$5.1, 1-hr $\geq$10.0, or 2-hr $\geq$8.5 mmol/L.
- Option B: Fasting $\geq$7.0 or 2-hr $\geq$11.1 mmol/L are thresholds for overt diabetes, not GDM.
- Option C: 1-hr $\geq$11.1 mmol/L alone is not the IADPSG threshold; IADPSG uses $\geq$10.0 mmol/L at 1 hour.
- Option D: Random glucose $\geq$11.1 mmol/L diagnoses overt DM, not GDM specifically.

Final Answer: Fasting $\geq$5.1 or 1-hour $\geq$10.0 or 2-hour $\geq$8.5 mmol/L $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 226

Q227.

Solution

Concept — Ruptured ectopic pregnancy: Positive βhCG + no IUP on USS + free fluid in POD + haemodynamic shock = ruptured ectopic pregnancy. This is a surgical emergency. Methotrexate is contraindicated when haemodynamically unstable.

Step 1 — Emergency action: BP 80/50, HR 130 = haemodynamic shock from intraperitoneal haemorrhage. Immediate surgical intervention (laparotomy or laparoscopy) is mandatory.

Why other options are wrong:

- Option A: Methotrexate — for unruptured, haemodynamically stable ectopic with βhCG <5000 IU/L; absolutely contraindicated here.
- Option B: Serial βhCG — appropriate only for stable, unruptured ectopic

under watchful waiting; not in shock.

- Option C: Correct — Emergency laparotomy/laparoscopy for ruptured ectopic with shock.
- Option D: Elective diagnostic laparoscopy — patient is in haemodynamic shock; elective is inappropriate.

Final Answer: Emergency laparotomy/laparoscopy ⇒ C

Answer: (C) Go Back to Q 227

Q228.

Solution

Concept — Bishop score components: Bishop score assesses cervical readiness for induction. Five parameters: (1) Dilation, (2) Effacement (length), (3) Station, (4) Consistency (firm/medium/soft), (5) Position (posterior/mid/anterior). Fetal heart rate pattern is NOT a component.

Step 1 — Identify the excluded parameter: The 5 Bishop score parameters are dilation, effacement, station, consistency, and position. FHR is monitored by CTG, not included in Bishop score.

Why other options are wrong:

- Option A: Cervical dilation — IS included (0=closed, 1=1–2 cm, 2=3–4 cm, 3=5+ cm).
- Option B: Fetal station — IS included (scores from -3 to +1/2).
- Option C: Cervical effacement — IS included (0=0–30%, 1=40–50%, 2=60–70%, 3=80%).
- Option D: Correct — Fetal heart rate is NOT part of the Bishop score.

Final Answer: Fetal heart rate pattern ⇒ D

Answer: (D) Go Back to Q 228

Q229.

Solution

Concept — Causes of PPH (4 T's): Primary PPH (blood loss >500 mL within 24 h of delivery) causes — Tone (uterine atony 70–80%), Tissue (retained placenta 10%), Trauma (lacerations 10%), Thrombin (coagulopathy). Uterine atony is by far the most common cause.

Step 1 — Identify cause: Soft boggy uterus = uterine atony. This is the most common cause of PPH (accounts for 70–80% of cases). Retained placenta compounds the bleeding here but atony is the primary driver.

Why other options are wrong:

- Option A: Correct — Uterine atony is the most common cause of PPH.
- Option B: Retained placenta — second most common cause; contributes here but atony is primary.
- Option C: Vaginal lacerations — cause bright red pulsatile bleeding with well-contracted uterus.
- Option D: Coagulopathy — less common; follows massive haemorrhage or DIC.

Final Answer: Uterine atony ⇒ A

Answer: (A) Go Back to Q 229

Q230.

Solution

Concept — Obstructed labour management: Bandl's ring (pathological retraction ring) = a visible/palpable groove between the thinned-out lower uterine segment and thickened upper segment, indicating impending uterine rupture. Management is emergency caesarean section immediately — adding oxytocin would be catastrophic.

Step 1 — Emergency decision: Fully dilated cervix + 0 station after 4 hours with adequate contractions + Bandl's ring = obstructed labour with impending rupture. Emergency LSCS is mandatory.

Why other options are wrong:

- Option A: Additional oxytocin is ABSOLUTELY contraindicated — will cause uterine rupture.
- Option B: Correct — Emergency caesarean section to prevent uterine rupture

and save mother and baby.

- Option C: Vacuum delivery — requires fetal head at perineum (+3/+4); head at 0 station makes this unsafe.
- Option D: Symphysiotomy — can be considered in resource-limited settings if CS not available, but LSCS is preferred.

Final Answer: Emergency caesarean section ⇒ B

Answer: (B) Go Back to Q 230

Q231.

Solution

Concept — Rotterdam criteria for PCOS: Diagnosis requires 2 of 3: (1) Oligo/anovulation, (2) Clinical/biochemical hyperandrogenism, (3) Polycystic ovarian morphology on USS. At least 2 criteria must be met after excluding other causes.

Step 1 — Apply Rotterdam: Oligomenorrhoea (oligo-anovulation) + clinical hyperandrogenism (acne, hirsutism) + PCO morphology ($\geq$12 follicles 2–9 mm) = 3/3 criteria met, PCOS confirmed.

Why other options are wrong:

- Option A: Elevated LH + low FSH + PCO — these are features of PCOS but NOT the Rotterdam criteria definition.
- Option B: Oligomenorrhoea alone with high androgens = only 2 criteria; this statement is incomplete and incorrect as it misrepresents the diagnosis.
- Option C: Correct — Rotterdam criteria: oligo-anovulation + hyperandrogenism + PCO morphology (any 2 of 3).
- Option D: Infertility + obesity + acne — not the diagnostic criteria; infertility is a consequence, not a criterion.

Final Answer: Oligo-anovulation, hyperandrogenism, and PCO morphology (any 2 of 3) ⇒ C

Answer: (C) Go Back to Q 231

Q232.

Solution

Concept — Fibroid types and symptoms: Submucosal fibroids (FIGO Type 0–2, intracavitary or indenting endometrial cavity) are the most common cause of heavy menstrual bleeding in fibroid uterus. Subserosal and pedunculated fibroids cause bulk symptoms (pressure), not HMB.

Step 1 — Identify fibroid responsible for HMB: Submucosal fibroid distorts the endometrial cavity and impairs uterine contractility, leading to profuse menstrual bleeding and dysmenorrhoea.

Why other options are wrong:

- Option A: Subserosal fibroid — projects outward; causes bulk symptoms (urinary frequency, constipation); rarely causes HMB.
- Option B: Pedunculated fibroid — hangs on a stalk; causes torsion, not HMB.
- Option C: Cervical fibroid — rare; causes dyspareunia, outlet obstruction.
- Option D: Correct — Submucosal fibroid is the type most responsible for heavy menstrual bleeding.

Final Answer: Submucosal fibroid $\Rightarrow$ D

Answer: (D) Go Back to Q 232

Q233.

Solution

Concept — Endometriosis diagnosis: Diagnostic laparoscopy with histological confirmation of endometrial glands and stroma outside the uterus is the gold standard for diagnosing endometriosis. No non-invasive test is sufficiently sensitive or specific for definitive diagnosis.

Step 1 — Gold standard: Powder-burn lesions on laparoscopy + histology confirming endometrial glands/stroma = definitive diagnosis. CA-125 and MRI have limited accuracy; USS detects endometriomas but not peritoneal implants.

Why other options are wrong:

- Option A: Correct — Diagnostic laparoscopy with histology is the gold standard.
- Option B: CA-125 — elevated in many conditions (PID, ovarian cancer, etc.); not specific; used for monitoring, not diagnosis.
- Option C: MRI — useful for deep infiltrating endometriosis and endometri-

omas but cannot detect superficial peritoneal lesions.
- Option D: Transvaginal USS — detects endometriomas (chocolate cysts) but not peritoneal implants reliably.

Final Answer: Diagnostic laparoscopy with histological confirmation $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 233

Q234.

Solution

Concept — Cervical cancer screening: WHO and current Indian NHM guidelines recommend HPV DNA testing as the preferred primary screening method (co-testing with cytology or primary HPV test alone) starting from age 25–30 years, with a 5-year interval. HPV testing has higher sensitivity than Pap smear alone.

Step 1 — Preferred screening method: For a 30-year-old sexually active woman with no prior abnormal smear, HPV DNA testing (primary or co-testing) every 5 years is recommended as the preferred method.

Why other options are wrong:

- Option A: Annual Pap smear from age 18 — outdated; current guidelines start at 21–25 years and recommend longer intervals.
- Option B: Correct — HPV DNA testing every 5 years (primary HPV or co-testing) from age 25–30.
- Option C: Colposcopy — a diagnostic procedure, not a screening test for the general population.
- Option D: VIA — acceptable in low-resource settings as an alternative, not the preferred method when HPV testing is available.

Final Answer: HPV DNA testing every 5 years $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 234

Q235.

Solution

Concept — Ovarian carcinoma histology: High-grade serous carcinoma (HGSC) is the most common and most lethal subtype of ovarian cancer, accounting for 70–80% of ovarian cancer deaths. It is often associated with BRCA1/2 mutations and commonly presents at advanced stage (Stage III–IV).

Step 1 — Most common type: Postmenopausal + bilateral masses + ascites + peritoneal deposits = advanced epithelial ovarian cancer. HGSC is the most common histological type.

Why other options are wrong:

- Option A: Mucinous cystadenocarcinoma — less common; typically large unilateral tumour; better prognosis.
- Option B: Endometrioid carcinoma — second most common epithelial type; associated with endometriosis; often lower grade.
- Option C: Correct — High-grade serous carcinoma is the most common ovarian cancer histotype.
- Option D: Clear cell carcinoma — associated with endometriosis; relatively resistant to platinum chemotherapy.

Final Answer: High-grade serous carcinoma $\Rightarrow$ C

Answer: (C) Go Back to Q 235

Q236.

Solution

Concept — PALM-COEIN AUB classification: PALM = structural causes (Polyp, Adenomyosis, Leiomyoma, Malignancy/hyperplasia); COEIN = non-structural causes (Coagulopathy, Ovulatory dysfunction, Endometrial, Iatrogenic, Not yet classified). Simple endometrial hyperplasia is classified under Endometrial (AUB-E).

Step 1 — Classify: Endometrial biopsy showing simple hyperplasia without atypia = endometrial cause = AUB-E (part of PALM structural causes — specifically the malignancy/hyperplasia category, often termed AUB-M/E).

Why other options are wrong:

- Option A: AUB-P (Polyp) — endometrial or cervical polyp on USS or hysteroscopy.

- Option B: AUB-A (Adenomyosis) — diffuse uterine enlargement, USS/MRI features of adenomyosis.
- Option C: AUB-L (Leiomyoma) — fibroid present on USS.
- Option D: Correct — AUB-E (Endometrial) includes endometrial hyperplasia and other primary endometrial disorders.

Final Answer: Endometrial (AUB-E) $\Rightarrow$ D

Answer: (D) Go Back to Q 236

Q237.

Solution

Concept — Contraception in breastfeeding with DVT history: Combined oral contraceptives are contraindicated in breastfeeding (first 6 months) and in DVT history (oestrogen-related thrombosis risk). Copper IUD is non-hormonal, highly effective (>99%), does not affect lactation, and is safe in DVT history. Levonorgestrel IUS and DMPA are progestogen-only but still hormonal.

Step 1 — Choose non-hormonal highly effective method: DVT history = contraindication to oestrogen (OCP) and concern for any thromboembolic progestogen risk. Non-hormonal = Cu-IUD. It does not interfere with breastfeeding.

Why other options are wrong:

- Option A: Correct — Copper IUD: non-hormonal, highly effective, safe in DVT, does not affect lactation.
- Option B: COCP — contraindicated: DVT history + breastfeeding (thrombosis risk, reduces breast milk).
- Option C: LNG-IUS — progestogen-containing; generally safer than COCP, but UKMEC category 2 for DVT history.
- Option D: DMPA — progestogen; safe in lactation but injectable, and some concerns with thrombosis risk in very high-risk patients.

Final Answer: Copper intrauterine device (Cu-IUD) $\Rightarrow$ A

Answer: (A) Go Back to Q 237

Q238.

Solution

Concept — Semen analysis interpretation: WHO 2021 reference values — Count $\geq$16 million/mL, motility $\geq$42% (total) or $\geq$30% progressive, morphology $\geq$4% (Kruger strict criteria). This patient: count 10 million/mL (oligospermia), motility 30% (asthenospermia), morphology 3% (teratospermia) = OAT syndrome.

Step 1 — Classify: All three parameters are below normal thresholds = Oligoasthenoteratozoospermia (OAT syndrome). This is the most common cause of male infertility.

Why other options are wrong:

- Option A: Azoospermia = zero sperm; count here is 10 million/mL.
- Option B: Correct — OAT syndrome (low count + low motility + poor morphology).
- Option C: Asthenospermia only would have normal count and morphology.
- Option D: Normospermia requires all values within normal limits; all three here are abnormal.

Final Answer: Oligoasthenoteratozoospermia (OAT syndrome) $\Rightarrow$ B

Answer: (B) Go Back to Q 238

Q239.

Solution

Concept — CTG classification: NICE/FIGO CTG classification: Normal = baseline 110–160 bpm + moderate variability (6–15 bpm) + accelerations + no/early decelerations. Variable decelerations that are uncomplicated (short duration, recover quickly) can make the trace suspicious/non-reassuring but need evaluation. Presence of accelerations is reassuring.

Step 1 — Classify CTG: Baseline 130 bpm (normal) + moderate variability + accelerations (reassuring features) + variable decelerations (one non-reassuring feature) = Suspicious/non-reassuring (one non-reassuring feature with otherwise normal features). Requires further assessment (e.g., fetal blood sampling).

Why other options are wrong:

- Option A: Fully normal CTG requires no variable decelerations. Variable decelerations make it suspicious.

- Option B: Pathological = 2+ non-reassuring features or any abnormal feature; only 1 non-reassuring feature here.
- Option C: Correct — Suspicious/non-reassuring (one non-reassuring feature — variable decelerations — with otherwise normal features).
- Option D: Sinusoidal pattern = smooth sine wave, absent variability, no accelerations; not present here.

Final Answer: Suspicious/non-reassuring — requires further evaluation $\Rightarrow$ C

Answer: (C) Go Back to Q 239

Q240.

Solution

Concept — Neonatal resuscitation (NRP algorithm): After initial steps (warm, dry, stimulate, position, clear airway), if the neonate is not breathing or HR <100 bpm $\rightarrow$ start Positive Pressure Ventilation (PPV). PPV is the single most important intervention in neonatal resuscitation. Cardiac compressions are only initiated if HR remains <60 bpm after 30 seconds of effective PPV.

Step 1 — NRP algorithm: APGAR 2 + HR 60 bpm + gasping = requires PPV immediately after initial stabilisation steps. Do NOT start compressions or epinephrine without adequate PPV first.

Why other options are wrong:

- Option A: Epinephrine — only after 60 seconds of adequate PPV + compressions if HR <60 bpm persists.
- Option B: Cardiac compressions — only if HR <60 bpm after 30 seconds of effective PPV.
- Option C: Correct — Start PPV immediately; it is the most critical next step after initial stabilisation.
- Option D: Surfactant via ETT — for respiratory distress syndrome in preterm, not for birth asphyxia resuscitation immediately.

Final Answer: Start positive pressure ventilation (PPV) $\Rightarrow$ C

Answer: (C) Go Back to Q 240

Q241.

Solution

Concept — Eclampsia management: Eclampsia = convulsions in a woman with pre-eclampsia. Immediate priorities: (1) Protect airway/position, (2) Control seizures with IV MgSO4, (3) Control BP, (4) Assess fetal condition, (5) Deliver — timing based on gestation and maternal/fetal status. MgSO4 is first priority.

Step 1 — Immediate priority: Convulsions + severe hypertension + 32 weeks = eclampsia. Stop seizures with MgSO4 (loading dose 4–6 g IV over 15–20 min) and stabilise before planning delivery.

Why other options are wrong:

- Option A: Correct — Control seizures with IV MgSO4 first, then stabilise BP, then plan delivery.
- Option B: Immediate LSCS without seizure control risks complications during anaesthesia; stabilise first.
- Option C: Diazepam — inferior to MgSO4 and associated with neonatal respiratory depression.
- Option D: Corticosteroids for lung maturity are important at 32 weeks but NOT the immediate priority in active seizures.

Final Answer: Control seizures with IV MgSO4 and stabilise $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 241

Q242.

Solution

Concept — HELLP syndrome: HELLP = Haemolysis (elevated LDH, schistocytes, anaemia) + Elevated Liver enzymes (AST/ALT) + Low Platelets ($<100{,}000/\mu$L). It is a severe variant of pre-eclampsia. Occurs typically in the third trimester.

Step 1 — Diagnose HELLP: Haemolysis (schistocytes, Hb 9.8 g/dL, LDH 1020) + EL (AST 185 U/L) + LP (platelets $62{,}000/\mu$L) + hypertension + proteinuria = HELLP syndrome. Definitive.

Why other options are wrong:

- Option A: Acute fatty liver of pregnancy — profound hypoglycaemia, coagulopathy, hepatic encephalopathy; different from HELLP.
- Option B: Correct — HELLP syndrome (all three criteria met).
- Option C: ITP — thrombocytopenia only; no haemolysis, no elevated liver

enzymes, no hypertension.

- Option D: HUS — thrombocytopenia + microangiopathic haemolytic anaemia + acute kidney injury; more prominent renal failure.

Final Answer: HELLP syndrome $\Rightarrow$ B

Answer: (B) Go Back to Q 242

Q243.

Solution

Concept — Anti-D prophylaxis: Anti-D immunoglobulin 300 μg (1500 IU) is given to Rh-negative mothers within 72 hours of delivery of an Rh-positive baby to prevent sensitisation. The 50 μg dose is used for threatened abortion or procedures before 12 weeks (not for delivery).

Step 1 — Correct dose and timing: O negative mother + Rh positive baby = give anti-D 300 μg IM within 72 hours of delivery. First pregnancy can cause sensitisation if inadequately managed.

Why other options are wrong:

- Option A: 50 μg anti-D — used for events before 12 weeks gestation (threatened miscarriage, termination); inadequate for delivery.
- Option B: Correct — 300 μg anti-D within 72 hours of delivery of Rh-positive baby.
- Option C: Blood transfusion — not indicated prophylactically here.
- Option D: No action — incorrect; sensitisation can occur in the first pregnancy, especially at delivery.

Final Answer: Anti-D immunoglobulin 300 μg within 72 hours $\Rightarrow$ C

Answer: (C) Go Back to Q 243

Q244.

Solution

Concept — Active phase arrest (Friedman's curve): In active labour ($\geq$6 cm), normal progress is $\geq$1 cm dilation per hour. Arrest of active phase = no cervical change for $\geq$4 hours with adequate contractions, or $\geq$6 hours with inadequate contractions. Here: 1 cm in 4 hours (4 h with adequate contractions, no descent) = active phase arrest.

Step 1 — Classify progress: Active phase (5 cm $\rightarrow$ 6 cm in 4 hours = 0.25 cm/h) with adequate contractions (3/10 min $\times$ 40 sec) = active phase arrest. This requires evaluation and likely intervention.

Why other options are wrong:

- Option A: Friedman's curve normal progress in active phase requires $\geq$1 cm/h in primigravida; 0.25 cm/h is abnormal.
- Option B: Latent phase prolongation — latent phase is before 6 cm; she is in active phase.
- Option C: Precipitate labour — very rapid progress; opposite of what is described.
- Option D: Correct — Active phase arrest (failure to progress): 1 cm in 4 hours with adequate contractions.

Final Answer: Active phase arrest (failure to progress) $\Rightarrow$ D

Answer: (D) Go Back to Q 244

Q245.

Solution

Concept — Developmental milestones (15 months): By 15 months: walks independently, says 3–5 words, feeds self with fingers, uses pincer grasp, waves bye-bye, points to desired objects. Full sentences (4–5 words) appear at 2.5–3 years; triangle drawing at 5 years; tricycle at 3 years.

Step 1 — Match milestone to age: Walking independently = milestone expected by 12–15 months. All other options describe milestones of much older children.

Why other options are wrong:

- Option A: Correct — Walking independently is an appropriate milestone at 15 months.
- Option B: Full sentences of 4–5 words — milestone at 2.5–3 years.

- Option C: Drawing a triangle — milestone at 5 years.
- Option D: Riding a tricycle — milestone at 3 years.

Final Answer: Walking independently $\Rightarrow$ A

Answer: (A) Go Back to Q 245

Q246.

Solution

Concept — Physiological jaundice of newborn: Physiological jaundice: appears day 2–3, peaks day 4–5, resolves by day 7–10 (term) or day 14 (preterm). Total bilirubin <15 mg/dL at day 3, predominantly unconjugated (indirect), no anaemia, no haemolysis. DCT negative. ABO incompatibility: DCT positive, anaemia, rapid rise in bilirubin from day 1.

Step 1 — Diagnose: Day 3, bilirubin 14 mg/dL, direct 0.5 (indirect/unconjugated), DCT negative, breastfeeding well, normal stool/urine = physiological jaundice.

Why other options are wrong:

- Option A: ABO incompatibility — DCT positive, haemolytic; jaundice appears on day 1. DCT negative here.
- Option B: Correct — Physiological jaundice: day 3, indirect bilirubin 14 mg/dL, DCT negative, no haemolysis.
- Option C: Biliary atresia — presents with persistent conjugated jaundice, pale stools, dark urine. Direct bilirubin would be markedly elevated.
- Option D: Breast milk jaundice — appears after day 5–7, peaks at 2 weeks, persists up to 12 weeks; too early at day 3.

Final Answer: Physiological jaundice of newborn $\Rightarrow$ B

Answer: (B) Go Back to Q 246

Q247.

Solution

Concept — Kwashiorkor vs Marasmus: Kwashiorkor: protein-energy malnutrition predominantly protein deficient. Features: oedema (pitting), moon face (generalised oedema), flaky paint dermatosis, flag sign (alternating bands in hair), hypoalbuminaemia (<2.8 g/dL), hepatomegaly. Some preservation of subcutaneous fat. Marasmus: severe caloric deficiency — severe wasting, old man face, NO oedema.

Step 1 — Classify: Oedema + moon face + flaky paint dermatosis + flag sign + albumin 1.8 g/dL = kwashiorkor. Weight 7 vs expected 12 kg = severe.

Why other options are wrong:

- Option A: Marasmus — severe wasting, NO oedema, "old man face" (NOT moon face), normal albumin relatively.
- Option B: Marasmic-kwashiorkor — features of both; severe wasting + oedema.
- Option C: Correct — Kwashiorkor: oedema + moon face + skin changes + hypoalbuminaemia.
- Option D: Iron deficiency anaemia — pallor, koilonychia; not oedema or skin changes.

Final Answer: Kwashiorkor $\Rightarrow$ C

Answer: (C) Go Back to Q 247

Q248.

Solution

Concept — Tetralogy of Fallot (TOF): TOF = 4 defects: (1) VSD, (2) Overriding aorta, (3) Pulmonary stenosis, (4) Right ventricular hypertrophy. Presents with cyanosis, hypercyanotic spells relieved by squatting (increases SVR), boot-shaped heart, decreased pulmonary markings. Single S2 (pulmonary component absent due to PS).

Step 1 — Diagnose: Cyanotic spells relieved by squatting + boot-shaped heart + decreased pulmonary markings + systolic murmur + single S2 = classic TOF.

Why other options are wrong:

- Option A: Isolated VSD — acyanotic; increased pulmonary markings; pansystolic murmur; cardiomegaly.

- Option B: TGA — presents with severe cyanosis from birth; egg-on-side cardiac silhouette; no boot-shaped heart.
- Option C: Large ASD — acyanotic; fixed split S2; right axis deviation.
- Option D: Correct — Tetralogy of Fallot (boot-shaped heart, decreased pulmonary markings, hypercyanotic spells, squatting relief).

Final Answer: Tetralogy of Fallot (TOF) $\Rightarrow$ D

Answer: (D) Go Back to Q 248

Q249.

Solution

Concept — UIP India immunisation schedule at 6 weeks: At 6 weeks: OPV-1, Pentavalent-1 (DPT + HepB + Hib), Rotavirus vaccine-1 (RVV-1), PCV-1, and fractional IPV-1 (fIPV-1 — intradermal). BCG, OPV-0, and HepB birth dose are given at birth.

Step 1 — Match schedule: 6-week vaccines = OPV-1 + Pentavalent-1 + RVV-1 + PCV-1 + fIPV-1. BCG is at birth, not 6 weeks.

Why other options are wrong:

- Option A: Correct — OPV-1, Pentavalent-1, RVV-1, PCV-1, fIPV-1 are all due at 6 weeks.
- Option B: OPV-0, BCG, HepB birth dose — given at birth (0 weeks), not at 6 weeks.
- Option C: MMR-1 and Varicella — given at 9–12 months and 15 months respectively.
- Option D: OPV-3, Pentavalent-3, RVV-3 — given at 14 weeks (3rd dose schedule).

Final Answer: OPV-1, Pentavalent-1, RVV-1, PCV-1, fIPV-1 $\Rightarrow$ A

Answer: (A) Go Back to Q 249

Q250.

Solution

Concept — WHO IMCI dehydration plans: Plan A = mild/no dehydration — ORS at home. Plan B = moderate dehydration — ORS 75 mL/kg over 4 hours in a health facility; reassess. Plan C = severe dehydration/shock — IV fluids (Ringer's lactate 100 mL/kg). This child has moderate dehydration (sunken eyes, reduced turgor, dry mucosa, tachycardia — no shock).

Step 1 — Classify and treat: Moderate dehydration = 2+ signs without shock. WHO Plan B: ORS 75 mL/kg over 4 hours in health facility with close monitoring.

Why other options are wrong:

- Option A: Plan C (IV Ringer's lactate 100 mL/kg) — for severe dehydration/shock; no shock present here.
- Option B: Correct — Plan B: ORS 75 mL/kg over 4 hours in health facility for moderate dehydration.
- Option C: Plan A (ORS at home) — for no dehydration or minimal dehydration only; moderate dehydration needs supervised ORT.
- Option D: Normal saline bolus — for shock (Plan C); no shock here.

Final Answer: ORS 75 mL/kg over 4 hours (Plan B) $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 250

Q251.

Solution

Concept — Febrile seizures management: Simple febrile seizures (single episode, <15 min, generalised, no focal deficit) in a child aged 6 months to 5 years require no anticonvulsant prophylaxis and no immediate lumbar puncture (unless clinical signs of meningitis). Management: fever control (paracetamol/ibuprofen), reassurance, parental education about recurrence (30%).

Step 1 — Classify and manage: Single episode, 2 minutes, generalised, no meningism = simple febrile seizure. No prophylaxis needed. Paracetamol for fever + education.

Why other options are wrong:

- Option A: LP immediately — only if signs of meningitis (neck stiffness, bulging fontanelle, purpuric rash); not for simple febrile seizure.
- Option B: Long-term anticonvulsant — not recommended for simple febrile

seizure; risks outweigh benefits.

- Option C: Correct — Fever control, reassurance, and parental education are standard management.
- Option D: MRI brain — not indicated in a first simple febrile seizure with no focal signs.

Final Answer: Paracetamol for fever, reassurance, parental education $\Rightarrow$ C

Answer: (C) Go Back to Q 251

Q252.

Solution

Concept — Measles complications: In malnourished children, measles carries high mortality from complications. The most serious preventable complications are pneumonia (most common cause of death) and encephalitis (post-measles encephalomyelitis). Vitamin A supplementation reduces mortality by 50%.

Step 1 — Most serious complication: Pneumonia accounts for 60–70% of measles deaths in developing countries. Encephalitis (1/1000 cases) is the most severe neurological complication. Together these are the most serious and preventable.

Why other options are wrong:

- Option A: Otitis media — common, less serious, treatable with antibiotics.
- Option B: Febrile convulsions — transient, rarely fatal.
- Option C: Parotitis — complication of mumps, not measles.
- Option D: Correct — Pneumonia and encephalitis are the most serious (and potentially fatal) complications.

Final Answer: Pneumonia and/or encephalitis $\Rightarrow$ D

Answer: (D) Go Back to Q 252

Q253.

Solution

Concept — Whooping cough (Pertussis): Caused by Bordetella pertussis. Drug of choice: azithromycin (or erythromycin). Azithromycin is preferred in infants <6 months (erythromycin associated with infantile hypertrophic pyloric stenosis). It eradicates organisms and reduces infectivity but does not reliably shorten the clinical course in the paroxysmal phase.

Step 1 — Identify drug of choice: Pertussis confirmed (paroxysmal cough + whoop + lymphocytosis). Drug of choice = azithromycin (especially in infants).

Why other options are wrong:

- Option A: Correct — Azithromycin is the drug of choice for pertussis in infants.
- Option B: Amoxicillin — no activity against Bordetella pertussis.
- Option C: Co-trimoxazole — alternative agent but not preferred; less effective than macrolides.
- Option D: Cephalexin — inactive against B. pertussis.

Final Answer: Azithromycin $\Rightarrow$ A

Answer: (A) Go Back to Q 253

Q254.

Solution

Concept — Large VSD features: Large VSD causes significant left-to-right shunt $\rightarrow$ pulmonary overflow $\rightarrow$ cardiomegaly and increased pulmonary vascular markings on CXR. Pansystolic murmur at lower left sternal border + mid-diastolic murmur (mitral flow murmur from increased pulmonary return) + failure to thrive = classic large VSD.

Step 1 — Diagnose: Pansystolic murmur LLSB + mid-diastolic apex murmur + cardiomegaly + increased pulmonary markings + failure to thrive = large VSD with haemodynamic significance.

Why other options are wrong:

- Option A: ASD (ostium secundum) — fixed widely split S2, ejection systolic murmur at pulmonary area, no failure to thrive in infancy usually.
- Option B: Correct — Large VSD with left-to-right shunt causing the described features.

- Option C: PDA — continuous machine-like murmur at left infraclavicular area; not pansystolic at LLSB.
- Option D: Pulmonary stenosis — ejection systolic murmur, thrill at pulmonary area; decreased pulmonary markings.

Final Answer: Large VSD with left-to-right shunt ⇒ B

Answer: (B) Go Back to Q 254

Q255.

Solution

Concept — Early-onset neonatal sepsis (EOS) pathogens: In India, the most common causative organisms of EOS are Gram-negative organisms — Klebsiella pneumoniae, E. coli, and Acinetobacter — unlike in Western countries where GBS predominates. Klebsiella is the most common cause of EOS in many Indian studies.

Step 1 — Most common in Indian context: Prolonged rupture of membranes (28 hours) + early sepsis in India = Klebsiella pneumoniae (Gram-negative) is the most common causative organism.

Why other options are wrong:

- Option A: Staphylococcus aureus — causes late-onset nosocomial neonatal sepsis; less common in EOS.
- Option B: Listeria monocytogenes — important in Western countries; rare in India.
- Option C: Correct — Klebsiella pneumoniae is the most common cause of EOS in India.
- Option D: GBS — predominant cause of EOS in Western developed countries; less common in India due to lower GBS colonisation rates.

Final Answer: Klebsiella pneumoniae ⇒ C

Answer: (C) Go Back to Q 255

Q256.

Solution

Concept — Diphtheria complications: Corynebacterium diphtheriae produces exotoxin causing two major life-threatening complications: (1) Laryngeal obstruction (laryngeal diphtheria → membranous croup → asphyxia) requiring emergency tracheostomy, and (2) Myocarditis (exotoxin causes cardiomyopathy, heart block, arrhythmias — leading cause of death in diphtheria).

Step 1 — Most dangerous acute complications: Stridor present = laryngeal extension → airway obstruction imminent. Myocarditis develops at 1–2 weeks. Both together are the most dangerous.

Why other options are wrong:

- Option A: Parotitis — complication of mumps, not diphtheria.
- Option B: Otitis media — common, minor complication.
- Option C: Peritonsillar abscess — complication of tonsillitis; not specific to diphtheria.
- Option D: Correct — Laryngeal obstruction (immediate) and myocarditis (delayed) are the most dangerous complications.

Final Answer: Laryngeal obstruction and myocarditis ⇒ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 256

Q257.

Solution

Concept — Gomez classification: Weight-for-age as % of expected median: Grade I (mild): 75–90%; Grade II (moderate): 60–74%; Grade III (severe): <60%. This child: $7/12 \times 100 = 58.3\%$ = Grade III (severe). No oedema = marasmus, not kwashiorkor.

Step 1 — Apply Gomez: 7 kg / 12 kg expected = 58.3% → Grade III malnutrition. No oedema + severe wasting + "old man's face" = marasmus pattern (severe caloric restriction).

Why other options are wrong:

- Option A: Correct — Grade III (severe) malnutrition, clinical picture of marasmus (severe wasting, no oedema).
- Option B: Grade II malnutrition = 60–74%; kwashiorkor has oedema (absent here).

- Option C: Grade I = 75–90%; weight is well below this.
- Option D: Normal = >90%; this child's weight is profoundly below normal.

Final Answer: Grade III malnutrition (severe) — marasmus ⇒ A

Answer: (A) Go Back to Q 257

Q258.

Solution

Concept — PDA pharmacological closure: Indomethacin (COX-1/2 inhibitor) or Ibuprofen (COX inhibitor) inhibit prostaglandin E2 synthesis, which normally maintains ductal patency. Prostaglandin inhibition closes the ductus in preterm neonates. Indomethacin has 70% success rate.

Step 1 — Drug of choice: Preterm PDA with significant shunting → indomethacin or ibuprofen IV. Surgical ligation is reserved if medical therapy fails.

Why other options are wrong:

- Option A: Furosemide — reduces pulmonary oedema symptoms but does not close PDA; counterproductive (increases prostaglandins).
- Option B: Correct — Indomethacin or ibuprofen (COX inhibitors) are the drugs of choice for PDA closure.
- Option C: Digoxin — treats heart failure; does not close PDA.
- Option D: Sildenafil — used in pulmonary arterial hypertension; dilates pulmonary vasculature; not for PDA closure.

Final Answer: Indomethacin or Ibuprofen (COX inhibitor) ⇒ B

Answer: (B) Go Back to Q 258

Q259.

Solution

Concept — Primary open-angle glaucoma (POAG): POAG: insidious onset, bilateral, open angles on gonioscopy, elevated IOP (normal >21 mmHg), progressive visual field loss (arcuate scotoma, nasal step), optic disc changes (increased CDR, neuroretinal rim notching/thinning, cup asymmetry). Painless.

Step 1 — Diagnose: IOP 26 mmHg + open angles + CDR 0.8 + inferior notching + arcuate scotoma = POAG. All classic features present.

Why other options are wrong:

- Option A: Acute angle-closure glaucoma — painful red eye, corneal oedema, fixed mid-dilated pupil, closed angle; acute presentation. Not painless chronic.
- Option B: Ocular hypertension — elevated IOP with normal disc and visual fields; no field defect or disc changes.
- Option C: Correct — POAG: open angles + elevated IOP + progressive field loss + disc changes.
- Option D: Secondary glaucoma from uveitis — history of uveitis, inflammatory cells, posterior synechiae.

Final Answer: Primary open-angle glaucoma (POAG) $\Rightarrow$ C

Answer: (C) Go Back to Q 259

Q260.

Solution

Concept — Posterior capsule opacification (PCO) treatment: PCO (secondary cataract) is the most common late complication of cataract surgery, occurring in 20–50% within 5 years. Nd:YAG laser posterior capsulotomy creates a hole in the opacified posterior capsule, restoring vision immediately. It is safe, quick, and outpatient-based.

Step 1 — Treatment of PCO: Elschnig's pearls + wrinkled posterior capsule + painless vision loss 2 months post-phaco = PCO. Nd:YAG laser capsulotomy is the definitive treatment.

Why other options are wrong:

- Option A: Repeat cataract surgery — unnecessary; the original IOL is in place; only the capsule is opacified.
- Option B: Topical corticosteroids — for inflammation, not capsular opacification.
- Option C: Anterior vitrectomy — not indicated for PCO.
- Option D: Correct — Nd:YAG laser posterior capsulotomy is the treatment of choice for PCO.

Final Answer: Nd:YAG laser posterior capsulotomy $\Rightarrow$ D

Answer: (D) Go Back to Q 260

Q261.

Solution

Concept — Diabetic retinopathy (DR) classification (ETDRS): Severe NPDR (pre-proliferative): any one of the "4-2-1 rule" — haemorrhages/MAs in all 4 quadrants, OR venous beading in 2+ quadrants, OR IRMA in 1+ quadrant. Also: cotton wool spots, hard exudates. No new vessels yet.

Step 1 — Classify: CWS + hard exudates (CSME risk) + IRMA + venous beading = at least one criterion of "4-2-1 rule" met = Severe NPDR. No new vessels (NVD/NVE) = not proliferative.

Why other options are wrong:

- Option A: Correct — Severe NPDR (pre-proliferative DR): IRMA + venous beading + CWS + hard exudates, no new vessels.
- Option B: Mild NPDR — only microaneurysms; none of the severe features.
- Option C: Moderate NPDR — some haemorrhages/MAs, CWS, hard exudates but not meeting 4-2-1 rule criteria.
- Option D: PDR — requires new vessels (NVD/NVE); none described here.

Final Answer: Severe non-proliferative diabetic retinopathy (NPDR) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 261

Q262.

Solution

Concept — Rhegmatogenous retinal detachment (RRD): RRD is caused by a break (tear or hole) in the retina through which fluid enters the subretinal space. Most common type in high myopes. Presents with photopsia, floaters (vitreous haemorrhage/pigment), then a dark shadow advancing from periphery centrally as more retina detaches.

Step 1 — Classify RD: Myope + photopsia + floaters + peripheral shadow + break at superotemporal quadrant = rhegmatogenous (break-induced) retinal detachment.

Why other options are wrong:

- Option A: Exudative/secondary RD — caused by sub-retinal fluid from choroidal disease (tumour, choroiditis); no break; retina is smooth and convex.
- Option B: Correct — Rhegmatogenous RD: break in retina + photopsia +

floaters + shadow.

- Option C: Tractional RD — caused by fibrovascular membranes pulling retina (diabetic, sickle cell); no break; concave surface; no photopsia.
- Option D: Retinoschisis — splitting of retinal layers; not a true detachment; rarely symptomatic.

Final Answer: Rhegmatogenous retinal detachment $\Rightarrow$ B

Answer: (B) Go Back to Q 262

Q263.

Solution

Concept — Vitamin A deficiency eye disease: Vitamin A deficiency causes xerophthalmia: night blindness (earliest), Bitot's spots (conjunctival metaplasia/keratin debris, triangular, foamy, temporal), xerosis, corneal ulceration $\rightarrow$ keratomalacia. Bitot's spots on both nasal and temporal conjunctiva with corneal dryness confirms VAD.

Step 1 — Identify deficiency: Night blindness + Bitot's spots + dry cornea in a malnourished tribal child = Vitamin A deficiency (xerophthalmia).

Why other options are wrong:

- Option A: Vitamin D deficiency — rickets; no eye manifestations.
- Option B: Vitamin C deficiency — scurvy; perifollicular haemorrhages, gum disease; no Bitot's spots.
- Option C: Correct — Vitamin A deficiency causes xerophthalmia (night blindness, Bitot's spots, corneal xerosis).
- Option D: Vitamin B12 deficiency — megaloblastic anaemia, subacute combined degeneration; optic neuropathy (rarely); no Bitot's spots.

Final Answer: Vitamin A deficiency $\Rightarrow$ C

Answer: (C) Go Back to Q 263

Q264.

Solution

Concept — Bacterial corneal ulcer treatment: Contact lens wearers are at high risk for Pseudomonas aeruginosa keratitis (Gram-negative rods, dense stromal infiltrate, rapid progression, hypopyon). Treatment: intensive topical fluoroquinolone (ciprofloxacin 0.3% or moxifloxacin) every 15–30 minutes initially.

Step 1 — Match organism to treatment: Gram-negative rods (Pseudomonas) + hypopyon + contact lens = bacterial keratitis. Topical fluoroquinolone is first-line.

Why other options are wrong:

- Option A: Natamycin — topical antifungal; for fungal keratitis (Aspergillus, Fusarium), not for Gram-negative bacteria.
- Option B: Topical acyclovir — for herpes simplex keratitis (dendritic ulcer, HSV); not bacterial.
- Option C: Oral acyclovir + steroids — for viral keratitis; steroids contraindicated in bacterial/fungal ulcer without coverage.
- Option D: Correct — Intensive topical fluoroquinolone (ciprofloxacin/moxifloxacin) for bacterial corneal ulcer.

Final Answer: Intensive topical fluoroquinolone (ciprofloxacin/moxifloxacin) $\Rightarrow$ D

Answer: (D) Go Back to Q 264

Q265.

Solution

Concept — Visual field defects and anatomical localisation: Bitemporal hemianopia = loss of both temporal visual fields = damage at the optic chiasm (where nasal fibres from both eyes cross). Pituitary tumours expand upward compressing the chiasm from below, causing bitemporal hemianopia.

Step 1 — Localise lesion: Bitemporal hemianopia = optic chiasm. Pituitary macroadenoma compresses chiasm from below (inferior chiasmal compression $\rightarrow$ superior temporal fields first affected).

Why other options are wrong:

- Option A: Correct — Optic chiasm compression causes bitemporal hemianopia.
- Option B: Right optic nerve — causes right monocular visual loss only (uni-

lateral).

- Option C: Right optic tract — causes left homonymous hemianopia (contralateral to the tract).
- Option D: Left occipital cortex — causes right homonymous hemianopia with macular sparing.

Final Answer: Optic chiasm (compression by pituitary tumour) ⇒ A

Answer: (A) Go Back to Q 265

Q266.

Solution

Concept — Anterior uveitis (iridocyclitis): HLA-B27 is strongly associated with anterior uveitis in seronegative spondyloarthropathies (ankylosing spondylitis, reactive arthritis, psoriatic arthritis). Anterior uveitis presents with acute painful red eye, photophobia, lacrimation, ciliary injection (limbal flush), and slit-lamp findings: fine KPs on posterior corneal surface, cells and flare in anterior chamber, posterior synechiae.

Step 1 — Diagnose: HLA-B27 positive + ankylosing spondylitis + painful red eye + ciliary injection + fine KPs + posterior synechiae = anterior uveitis (iridocyclitis).

Why other options are wrong:

- Option A: Bacterial conjunctivitis — mucopurulent discharge, no KPs, no photophobia, no posterior synechiae.
- Option B: Correct — Anterior uveitis with HLA-B27 association and slit-lamp findings.
- Option C: Posterior uveitis — choroiditis; no anterior segment involvement; fundus shows lesions.
- Option D: Scleritis — deep boring pain, scleral tenderness, violaceous hue; no KPs.

Final Answer: Anterior uveitis (iridocyclitis) ⇒ B

Answer: (B) Go Back to Q 266

Q267.

Solution

Concept — Cover-uncover test for manifest squint: In manifest (tropia) squint, the deviated eye moves to take up fixation when the fixing eye is covered. When cover is removed, the deviated eye drifts back. The fixing eye does not move when the deviated eye is covered. This confirms unilateral manifest squint (esotropia of the left eye).

Step 1 — Interpret test: Left eye moves outward to fix when right eye is covered = left eye was deviated inward (esotropia). Right eye doesn't move when left is covered = right eye is the preferred fixing eye. = Unilateral manifest esotropia (concomitant squint).

Why other options are wrong:

- Option A: Paralytic (non-comitant) squint — angle changes with direction of gaze; tested by alternate cover test; diplopia present.
- Option B: Intermittent squint of the right eye — right eye shows no movement in this test; left eye is the squinting eye.
- Option C: Correct — Unilateral concomitant manifest esotropia of the left eye.
- Option D: Pseudosquint — normal cover test; apparent squint due to epicanthal folds, no movement on cover test.

Final Answer: Unilateral concomitant squint (manifest esotropia of the left eye) $\Rightarrow$ C

Answer: (C) Go Back to Q 267

Q268.

Solution

Concept — Wet vs Dry ARMD: Wet (neovascular/exudative) ARMD: choroidal neovascularisation (CNV) grows under the retina $\rightarrow$ subretinal fluid/haemorrhage $\rightarrow$ rapid central vision loss, metamorphopsia. OCT shows subretinal/intraretinal fluid. Treated with anti-VEGF injections. Dry ARMD: geographic atrophy, drusen, slow progressive loss, no fluid.

Step 1 — Classify: Subretinal neovascular membrane + subretinal haemorrhage + OCT fluid + metamorphopsia = wet (neovascular) ARMD.

Why other options are wrong:

- Option A: Dry ARMD — no CNV, no subretinal fluid or haemorrhage; geographic atrophy; slow vision loss.
- Option B: Central serous retinopathy — subretinal fluid without CNV or haemorrhage; typically young males under stress; self-limiting.
- Option C: Macular hole — full-thickness hole in the fovea; distinct OCT appearance; no subretinal haemorrhage.
- Option D: Correct — Wet (neovascular/exudative) ARMD with CNV and subretinal haemorrhage.

Final Answer: Wet (neovascular/exudative) ARMD $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 268

Q269.

Solution

Concept — Acute suppurative otitis media (ASOM) treatment: ASOM in children: first-line treatment is amoxicillin for 5–7 days (or 10 days in <2 years or severe cases) along with analgesics. Spontaneous resolution occurs in 60% but antibiotic reduces duration and complications. Myringotomy is for non-resolving/recurrent cases.

Step 1 — First-line treatment: Bulging TM + loss of landmarks + fever + otalgia after URTI = ASOM. Amoxicillin is first-line antibiotic.

Why other options are wrong:

- Option A: Correct — Amoxicillin for 5–7 days is first-line for ASOM.
- Option B: Myringotomy + grommets — for recurrent AOM ($\geq$3 episodes in 6 months) or persistent otitis media with effusion, not for a single acute episode.
- Option C: Topical antibiotic drops — for otitis externa or post-perforation discharge; not for intact TM ASOM.
- Option D: Mastoidectomy — for mastoiditis complication; not first-line for simple ASOM.

Final Answer: Amoxicillin for 5–7 days plus analgesics $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 269

Q270.

Solution

Concept — Tuning fork tests interpretation: Rinne's test: AC > BC = positive (normal or SNHL); AC < BC = negative (conductive HL). Weber's test: lateralises to the better ear in SNHL; lateralises to the worse ear in conductive HL. Pattern here: Rinne's negative right + Weber's to right = conductive HL in the right ear.

Step 1 — Interpret: Rinne's negative (AC < BC) right = bone conduction better than air in right ear = conductive HL right. Weber lateralises to right = right ear is the conductively impaired ear (paradoxically "hears" bone conduction better in the affected side).

Why other options are wrong:

- Option A: SNHL right — Rinne's positive (AC > BC); Weber lateralises to the LEFT (better ear) in SNHL.
- Option B: Correct — Conductive HL right: Rinne's negative right + Weber lateralises to right.
- Option C: SNHL left — Weber would lateralise to right (better ear), but Rinne's would be positive on left; here Rinne's is negative on right.
- Option D: Mixed HL bilaterally — would require bilateral abnormalities; Weber's strongly lateralises to one side.

Final Answer: Conductive hearing loss in the right ear $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 270

Q271.

Solution

Concept — Noise-induced hearing loss (NIHL): NIHL is bilateral, symmetrical sensorineural hearing loss (AC = BC, no air-bone gap) with a characteristic audiometric notch at 4000 Hz (4 kHz notch). Caused by chronic exposure to loud noise (factory, machinery). Poor speech discrimination due to cochlear hair cell damage.

Step 1 — Diagnose: Factory noise exposure + bilateral AC and BC elevated equally at 4 kHz (no air-bone gap) + 4 kHz notch = NIHL.

Why other options are wrong:

- Option A: Otosclerosis — conductive HL; air-bone gap present; maximum at 2 kHz; Carhart's notch.

- Option B: Presbycusis — age-related SNHL; progressive high-frequency loss; no specific 4 kHz notch; no occupational noise history.
- Option C: Correct — NIHL: bilateral SNHL with 4 kHz notch in a factory worker.
- Option D: Meniere's disease — low-frequency fluctuating SNHL, tinnitus, vertigo, aural fullness; episodic.

Final Answer: Noise-induced hearing loss (NIHL) $\Rightarrow$ C

Answer: (C) Go Back to Q 271

Q272.

Solution

Concept — Cholesteatoma complications: Cholesteatoma (keratinising stratified squamous epithelium in the middle ear/mastoid) destroys bone by enzymatic erosion (collagenase). Untreated, it can erode into intracranial structures $\rightarrow$ meningitis, extradural abscess, subdural abscess, brain abscess, sigmoid sinus thrombosis, and facial nerve palsy.

Step 1 — Most serious complication: Bone erosion by cholesteatoma $\rightarrow$ lateral wall of mastoid $\rightarrow$ mastoiditis (subperiosteal abscess, post-auricular swelling). Further erosion $\rightarrow$ tegmen (meningitis) or dural sinuses (sigmoid sinus thrombosis) or temporal lobe (brain abscess).

Why other options are wrong:

- Option A: Serous otitis media — middle ear effusion without infection; not a complication of cholesteatoma.
- Option B: Eustachian tube dysfunction — predisposes to cholesteatoma formation; not its complication.
- Option C: Tympanosclerosis — calcification of middle ear structures; different entity; non-dangerous.
- Option D: Correct — Intracranial extension (meningitis or brain abscess) is the most serious complication.

Final Answer: Intracranial extension — meningitis or brain abscess $\Rightarrow$ D

Answer: (D) Go Back to Q 272

Q273.

Solution

Concept — Nasal polyp treatment: Nasal polyps arise from eosinophilic inflammation of the nasal mucosa. First-line treatment: topical intranasal corticosteroids (INCS — mometasone, fluticasone, beclomethasone) — they reduce polyp size, improve symptoms, and prevent recurrence. Systemic steroids for acute exacerbation. Surgery (FESS) for refractory cases.

Step 1 — Drug of choice: Nasal polyps + Samter's triad (asthma + aspirin sensitivity + nasal polyps) = topical INCS is first-line medical treatment.

Why other options are wrong:

- Option A: Correct — Topical intranasal corticosteroids (mometasone) are the drug of choice for nasal polyps.
- Option B: Systemic antihistamines — for allergic rhinitis; no role in reducing polyp size.
- Option C: Antibiotics — for bacterial sinusitis; not for inflammatory nasal polyps.
- Option D: Topical decongestant long-term — contraindicated (rhinitis medicamentosa); not for polyps.

Final Answer: Topical intranasal corticosteroids (mometasone) $\Rightarrow$ A

Answer: (A) Go Back to Q 273

Q274.

Solution

Concept — Anterior epistaxis management: Little's area (Kiesselbach's plexus) on the anteroinferior nasal septum is the most common site of epistaxis (90%). First-line: direct pressure (compress soft part of nose for 10–20 minutes, lean forward). If bleeding point visible and accessible: silver nitrate chemical cauterisation. Posterior packing and embolisation are for posterior epistaxis.

Step 1 — Initial management: Anterior epistaxis, haemodynamically stable, identifiable bleeding point = compression + silver nitrate cauterisation.

Why other options are wrong:

- Option A: Posterior nasal packing — for posterior epistaxis (above/behind posterior choana); not for anterior epistaxis.
- Option B: Correct — Compression of the soft nose for 10–20 minutes + silver

nitrate cauterisation for Little's area bleeding.
- Option C: Internal maxillary artery embolisation — for refractory posterior epistaxis not controlled by packing.
- Option D: IV tranexamic acid + transfusion — for massive, life-threatening epistaxis with haemodynamic instability; not first-line.

Final Answer: Compression and silver nitrate cauterisation ⇒ B

Answer: (B) Go Back to Q 274

Q275.

Solution

Concept — Orbital complications of sinusitis (Chandler classification): Chandler stages: I=Pre-septal cellulitis (periorbital), II=Orbital cellulitis, III=Subperiosteal abscess, IV=Orbital abscess, V=Cavernous sinus thrombosis. Proptosis + restricted EOM + fever with CT showing collection between periorbita and orbital walls = subperiosteal abscess (Stage III).

Step 1 — Classify: Proptosis + restricted EOM + eyelid swelling + CT evidence of subperiosteal collection → Chandler Stage III (subperiosteal abscess). Requires surgical drainage.

Why other options are wrong:

- Option A: Pre-septal cellulitis — eyelid oedema without proptosis or restricted EOM; CT shows no collection.
- Option B: Cavernous sinus thrombosis — bilateral eye involvement, high fever, meningism, toxaemia; later stage.
- Option C: Correct — Subperiosteal abscess: proptosis + restricted EOM + CT showing periorbital collection.
- Option D: Mucocele — painless, slowly progressive proptosis in adults; no acute infectious features.

Final Answer: Subperiosteal abscess of the orbit ⇒ C

Answer: (C) Go Back to Q 275

Q276.

Solution

Concept — Laryngeal cancer lymphatic drainage: Glottis (true vocal cords) has virtually no lymphatic drainage — hence carcinoma here presents early with hoarseness but late nodal spread. Supraglottis has rich bilateral lymphatic drainage → early bilateral nodal metastasis. This is why supraglottic carcinoma has a worse prognosis despite appearing less obvious initially.

Step 1 — Drainage of supraglottis: Supraglottic larynx → rich bilateral lymphatic channels → deep cervical nodes (jugulodigastric and others) → early bilateral nodal spread possible even with small primary tumour.

Why other options are wrong:

- Option A: No lymphatic drainage — characteristic of glottis, not supraglottis.
- Option B: Sparse drainage/late metastasis — this describes glottis, not supraglottis.
- Option C: Submental nodes — for lower lip/floor of mouth lesions; not supraglottis.
- Option D: Correct — Supraglottis: rich bilateral lymphatic drainage to deep cervical nodes = early nodal spread.

Final Answer: Rich bilateral lymphatic drainage to deep cervical nodes ⇒ D

Answer: (D) Go Back to Q 276

Q277.

Solution

Concept — Tracheostomy indications: Tracheostomy is indicated for: (1) Prolonged mechanical ventilation (>7–14 days) to facilitate weaning, secretion clearance, patient comfort, and allow oral feeding; (2) Upper airway obstruction (acute or chronic); (3) To bypass laryngeal/pharyngeal obstruction (tumour, burns); (4) Neurological conditions with poor airway protection. In this case, prolonged ventilation (14 days) for GBS is the primary indication.

Step 1 — Primary indication: GBS with respiratory failure, intubated 14 days = prolonged mechanical ventilation requiring long-term airway access — classic indication for tracheostomy.

Why other options are wrong:

- Option A: Correct — Prolonged mechanical ventilation (>7–14 days) is the

most important indication here.
- Option B: Acute upper airway obstruction alone can be managed with emergency tracheostomy but is not this scenario.
- Option C: Tracheostomy facilitates tube feeding but is not done primarily for oral feeding.
- Option D: Laryngeal tumour resection — would be an indication, but not the scenario described.

Final Answer: Prolonged mechanical ventilation requiring long-term airway access $\Rightarrow$ A

Answer: (A) Go Back to Q 277

Q278.

Solution

Concept — Foreign body airway — conscious child: For a conscious child (>1 year) with partial airway obstruction: 5 back blows between shoulder blades followed by 5 abdominal thrusts (Heimlich manoeuvre). Do NOT perform blind finger sweeps. Rigid bronchoscopy is done for complete/persistent obstruction under controlled setting.

Step 1 — Immediate manoeuvre: Conscious child, partial obstruction (can cry) $\rightarrow$ 5 back blows + 5 abdominal thrusts (Heimlich). Never perform blind finger sweeps (can push FB deeper).

Why other options are wrong:

- Option A: Blind finger sweep — contraindicated in children; can push foreign body deeper.
- Option B: Correct — 5 back blows + 5 abdominal thrusts for conscious child with partial obstruction.
- Option C: Emergency tracheostomy — for complete, irremovable airway obstruction; not first-line in partial obstruction.
- Option D: Rigid bronchoscopy — for persistent or complete obstruction not cleared by BLS manoeuvres; not immediate first-line in a conscious partial obstruction.

Final Answer: 5 back blows alternating with 5 abdominal/chest thrusts $\Rightarrow$ B

Answer: (B) Go Back to Q 278

Q279.

Solution

Concept — Spiral fracture: A spiral fracture results from a torsional (twisting) force applied to the long bone. The fracture line spirals around the bone shaft in a helical pattern over a long segment. Common in skiing, indirect twisting injuries.

Step 1 — Match mechanism to fracture type: Twisting injury + oblique spiralling fracture line over long segment = spiral fracture. Transverse = direct blow perpendicular to bone. Comminuted = multiple fragments.

Why other options are wrong:

- Option A: Transverse fracture — perpendicular line across the bone; from direct impact.
- Option B: Comminuted fracture — bone shattered into multiple (>2) fragments; from high-energy injury.
- Option C: Correct — Spiral fracture from twisting mechanism; characteristic helical oblique line.
- Option D: Greenstick fracture — incomplete fracture in children (cortex broken on one side only); not adults.

Final Answer: Spiral fracture $\Rightarrow$ C

Answer: (C) Go Back to Q 279

Q280.

Solution

Concept — Fat embolism syndrome (FES): FES (Gurd's criteria): MAJOR — petechial rash (pathognomonic: over chest, axillae, conjunctivae), respiratory failure, cerebral dysfunction. MINOR — fever, tachycardia, jaundice, renal failure. Typically 24–72 hours after long bone fracture or intramedullary procedures. Bilateral pulmonary infiltrates = ARDS from fat emboli.

Step 1 — Diagnose FES: 48 hours post-femur fracture + acute confusion + petechial rash on chest/axillae + hypoxia + bilateral pulmonary infiltrates = Fat embolism syndrome (Gurd's criteria: 2 major criteria).

Why other options are wrong:

- Option A: Pulmonary thromboembolism — sudden onset dyspnoea, pleuritic chest pain; petechial rash NOT a feature.
- Option B: ARDS from sepsis — would have fever, source of sepsis, leukocy-

tosis; no petechial rash.

- Option C: Haemothorax — unilateral dullness, absent breath sounds; requires rib fractures, not isolated femur.
- Option D: Correct — Fat embolism syndrome: petechial rash + confusion + hypoxia + bilateral infiltrates 48 h post-femoral fracture.

Final Answer: Fat embolism syndrome (Gurd's criteria) $\Rightarrow$ D

Answer: (D) Go Back to Q 280

Q281.

Solution

Concept — Colles' fracture: Colles' fracture = fracture of the distal radius with dorsal displacement, dorsal (posterior) angulation, radial deviation, and impaction (dinner-fork deformity). Classic mechanism: fall on outstretched hand (FOOSH) in older osteoporotic women. Smith's fracture = "reverse Colles'" — volar/anterior displacement.

Step 1 — Match features: Dinner-fork deformity + dorsal displacement/angulation + radial deviation + FOOSH mechanism = Colles' fracture.

Why other options are wrong:

- Option A: Correct — Colles' fracture: dorsal displacement + angulation + radial deviation + FOOSH.
- Option B: Smith's fracture — volar displacement (garden-spade deformity); from FOOSH with wrist flexed.
- Option C: Barton's fracture — intra-articular fracture-dislocation of distal radius; either direction; subluxation of wrist joint.
- Option D: Monteggia fracture-dislocation — fracture of proximal ulna + dislocation of radial head; not wrist.

Final Answer: Colles' fracture $\Rightarrow$ A

Answer: (A) Go Back to Q 281

Q282.

Solution

Concept — Osteosarcoma: Most common primary malignant bone tumour in adolescents (10–25 years). Site: metaphysis of long bones, especially distal femur/proximal tibia (around knee). Features: Codman's triangle (periosteal elevation), sunburst pattern, elevated ALP, malignant osteoid production on biopsy.

Step 1 — Diagnose: 16-year-old + distal femur metaphysis + Codman's triangle + sunburst + elevated ALP + malignant osteoid = osteosarcoma. Classic presentation.

Why other options are wrong:

- Option A: Giant cell tumour — epiphysis, 20–40 years, soap-bubble appearance, no ALP elevation.
- Option B: Correct — Osteosarcoma: metaphysis, adolescent, Codman's triangle + sunburst + malignant osteoid.
- Option C: Ewing's sarcoma — diaphysis, younger children/teens, onion-skin periosteal reaction, no Codman's or sunburst; question indicates osteoid production (not Ewing's).
- Option D: Chondrosarcoma — cartilaginous tumour, older adults, axial skeleton; chondroid matrix, not osteoid.

Final Answer: Osteosarcoma $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 282

Q283.

Solution

Concept — Compartment syndrome: Compartment syndrome = elevated pressure within a closed fascial compartment compromising perfusion. Diagnosis: compartment pressure >30 mmHg or delta pressure (diastolic BP - compartment pressure) <30 mmHg. The 6 P's: Pain (out of proportion), Pressure, Paraesthesia, Pallor, Pulselessness, Paralysis. Treatment: EMERGENCY fasciotomy — all four compartments of the leg.

Step 1 — Treat: Delta pressure <30 mmHg = critical ischaemia. Immediate emergency fasciotomy of all four leg compartments (anterior, lateral, superficial posterior, deep posterior) is mandatory.

Why other options are wrong:

- Option A: Elevate + morphine — elevation reduces arterial perfusion in compartment syndrome; worsens ischaemia; contraindicated.
- Option B: Ice + observe — observation delays decompression; risks permanent nerve/muscle damage.
- Option C: Correct — Emergency fasciotomy of all four compartments is the only effective treatment.
- Option D: Angiography — vascular injury is a differential, but compartment pressure criteria are met; fasciotomy should not be delayed.

Final Answer: Emergency fasciotomy of all four compartments $\Rightarrow$ C

Answer: (C) Go Back to Q 283

Q284.

Solution

Concept — Avascular necrosis (AVN) of femoral head: AVN results from interruption of blood supply to the femoral head $\rightarrow$ ischaemic necrosis $\rightarrow$ subchondral collapse. Causes: corticosteroids, trauma (femoral neck fracture), alcohol, sickle cell disease, decompression sickness. MRI is gold standard early; subchondral crescent sign = pathognomonic of AVN.

Step 1 — Diagnose: Long-term prednisolone + progressive hip pain + subchondral crescent sign + femoral head collapse on MRI = AVN. Classic steroid-induced AVN.

Why other options are wrong:

- Option A: Osteoarthritis — joint space narrowing, osteophytes, subchondral sclerosis; no crescent sign; different age group.
- Option B: Septic arthritis — acute, fever, very restricted movement in all directions; joint fluid infected.
- Option C: Rheumatoid arthritis — symmetric polyarthritis; erosions; not typical isolated hip in SLE.
- Option D: Correct — AVN: steroid use + crescent sign + femoral head collapse on MRI.

Final Answer: Avascular necrosis (AVN) of the femoral head $\Rightarrow$ D

Answer: (D) Go Back to Q 284

Q285.

Solution

Concept — Acute haematogenous osteomyelitis pathogen: In children >1 year, the most common causative organism of acute haematogenous osteomyelitis is Staphylococcus aureus (responsible for 70–80% of cases). It seeds in the metaphysis of long bones (proximal tibia, distal femur) via haematogenous spread during bacteraemia.

Step 1 — Most common organism: 10-year-old + tibial metaphysis + fever + elevated inflammatory markers = acute haematogenous osteomyelitis. S. aureus is the most common cause in all age groups except neonates (also GBS) and sickle cell (Salmonella).

Why other options are wrong:

- Option A: Correct — Staphylococcus aureus is the most common causative organism (70–80%).
- Option B: Streptococcus pyogenes — second most common in children; less frequent than S. aureus.
- Option C: Salmonella typhi — most common in sickle cell disease patients; not in this normal child.
- Option D: Pseudomonas aeruginosa — in IV drug users or puncture wound foot osteomyelitis; not typical haematogenous in children.

Final Answer: Staphylococcus aureus $\Rightarrow$ A

Answer: (A) Go Back to Q 285

Q286.

Solution

Concept — Radial nerve palsy in mid-shaft humeral fracture: The radial nerve winds around the posterior aspect of the humerus in the spiral groove at the level of the mid-shaft. It is the most commonly injured nerve in humeral shaft fractures. Injury causes: wrist drop (paralysis of wrist extensors: ECRL, ECRB, ECU), finger drop (MCP extension), loss of thumb abduction, and sensory loss over the anatomical snuff box and dorsum of thumb web space.

Step 1 — Identify nerve: Mid-shaft humerus fracture + wrist drop + loss of MCP extension + sensory loss over dorsum of hand = radial nerve palsy in the spiral groove.

Why other options are wrong:

- Option A: Median nerve — causes loss of thumb opposition, thenar wasting, sensory loss over palm and radial 3.5 digits; no wrist drop.
- Option B: Correct — Radial nerve palsy: wrist drop + finger drop + dorsal hand sensory loss from mid-shaft humeral fracture.
- Option C: Ulnar nerve — causes claw hand (ring and little finger), sensory loss over ulnar 1.5 digits; no wrist drop.
- Option D: Musculocutaneous nerve — causes weakness of elbow flexion + sensory loss over lateral forearm; no wrist drop.

Final Answer: Radial nerve $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q 286

Q287.

Solution

Concept — Shoulder dislocation: Anterior glenohumeral dislocation accounts for 95–97% of all shoulder dislocations. Mechanism: forced abduction + external rotation (FOOSH or tackle). Humeral head dislocates anteriorly and inferiorly (subcoracoid position). Features: loss of shoulder contour (deltoid flattening), arm held in slight abduction and external rotation, axillary fullness (palpable humeral head).

Step 1 — Diagnose: Forced abduction + external rotation + arm held in abduction/ER + loss of shoulder contour + axillary fullness = anterior dislocation (subcoracoid).

Why other options are wrong:

- Option A: Posterior dislocation — rare (<3%); caused by seizure, electrocution; arm held in adduction + internal rotation; lightbulb sign on AP X-ray.
- Option B: Inferior (luxatio erecta) — extremely rare; arm locked overhead in full abduction.
- Option C: Correct — Anterior glenohumeral dislocation is the most common type (95–97%).
- Option D: Superior dislocation — very rare; associated with massive rotator cuff tear.

Final Answer: Anterior glenohumeral dislocation $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q 287

Q288.

Solution

Concept — Ewing's sarcoma: Second most common primary malignant bone tumour in children/adolescents (5–25 years). Site: diaphysis of long bones (femur, tibia) and flat bones (pelvis, ribs, scapula). Features: onion-skin periosteal reaction (lamellated), permeative lytic lesion, soft tissue mass, systemic features (fever, elevated ESR, LDH). Histology: small round blue cells (CD99 positive, t(11;22) translocation).

Step 1 — Diagnose: 14-year-old + femoral diaphysis + onion-skin periosteal reaction + soft tissue mass + systemic symptoms = Ewing's sarcoma.

Why other options are wrong:

- Option A: Osteosarcoma — metaphysis, sunburst pattern, Codman's triangle, osteoid production; not diaphysis or onion-skin.
- Option B: Acute osteomyelitis — can mimic Ewing's, but no onion-skin pattern on X-ray; bone scan/MRI and biopsy differentiate.
- Option C: Giant cell tumour — epiphysis, soap-bubble appearance, 20–40 years; not diaphysis in a child.
- Option D: Correct — Ewing's sarcoma: diaphysis + onion-skin + soft tissue mass + systemic features in a teenager.

Final Answer: Ewing's sarcoma $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q 288

Q289.

Solution

Concept — Mallampati grading: Class I: soft palate, uvula, faucial pillars fully visible. Class II: soft palate, uvula visible, faucial pillars obscured. Class III: soft palate and base of uvula visible only. Class IV: only hard palate visible. Class III and IV predict difficult intubation. Class III = soft palate visible, uvula not seen (only base or none).

Step 1 — Apply grading: Only soft palate visible, NO uvula, NO faucial pillars = Mallampati Class III (difficult intubation predicted).

Why other options are wrong:

- Option A: Correct — Mallampati Class III: soft palate visible, uvula base partially or not visible, faucial pillars not seen.

- Option B: Class I — all structures visible; easy intubation.
- Option C: Class II — soft palate + uvula visible, faucial pillars obscured; moderate difficulty.
- Option D: Class IV — only hard palate visible; most difficult. Here soft palate IS visible → Class III not IV.

Final Answer: Class III (difficult intubation predicted) ⇒ A

Answer: (A) Go Back to Q 289

Q290.

Solution

Concept — LMA vs ETT for laparoscopy: Laparoscopic cholecystectomy involves: (1) Trendelenburg position, (2) CO2 pneumoperitoneum (increases aspiration risk), (3) Raised intraabdominal pressure pushing stomach contents upward. ETT with cuffed tube provides a sealed airway, protects against aspiration, and allows controlled ventilation against raised intraabdominal pressure. LMA does not seal the oesophagus and risks aspiration.

Step 1 — Choose airway device: Laparoscopy + CO2 pneumoperitoneum + aspiration risk = cuffed ETT is mandatory for airway protection.

Why other options are wrong:

- Option A: LMA — supraglottic device; does not protect against aspiration; higher risk of gastric regurgitation under pneumoperitoneum.
- Option B: Correct — Cuffed ETT provides airway protection against aspiration during laparoscopy.
- Option C: Nasopharyngeal airway — not adequate for general anaesthesia or laparoscopy.
- Option D: Guedel airway — only maintains oropharyngeal patency; no aspiration protection.

Final Answer: Endotracheal tube (ETT) with cuffed tube ⇒ B

Answer: (B) Go Back to Q 290

Q291.

Solution

Concept — PONV prophylaxis: The 5HT3 receptor antagonists (ondansetron, granisetron) are the most effective and best-tolerated first-line agents for PONV prophylaxis. For high Apfel risk score (3–4), multimodal prophylaxis with 2–3 drugs is recommended: ondansetron + dexamethasone ± scopolamine. Ondansetron IV is given at end of surgery.

Step 1 — First-line antiemetic: Apfel score 4 (highest risk) = multimodal PONV prophylaxis. Ondansetron (5HT3 antagonist) is the most commonly recommended first-line drug.

Why other options are wrong:

- Option A: Haloperidol — dopamine antagonist; second-line; used for breakthrough PONV.
- Option B: Metoclopramide — dopamine antagonist; weak antiemetic; not preferred first-line.
- Option C: Correct — Ondansetron IV (5HT3 antagonist) is first-line for PONV prophylaxis.
- Option D: Promethazine IM — antihistamine + anticholinergic; causes sedation; not first-line in modern PONV protocols.

Final Answer: Ondansetron IV (5HT3 antagonist) ⇒ C

Answer: (C) Go Back to Q 291

Q292.

Solution

Concept — Thoracic epidural analgesia for abdominal surgery: Thoracic epidural analgesia (TEA) provides superior post-operative pain control for open abdominal and thoracic surgery compared to systemic opioids alone. It reduces pulmonary complications, facilitates early mobilisation, decreases ICU stay, and attenuates the stress response. Gold standard for major open abdominal surgery.

Step 1 — Best analgesia for open AAA repair: Open abdominal aortic surgery → thoracic epidural is gold standard. IV morphine PCA alone is inferior.

Why other options are wrong:

- Option A: Femoral nerve block — for lower limb surgery (hip/knee); not for abdominal surgery.

- Option B: IV morphine PCA — effective but inferior to epidural for open abdominal surgery; more side effects.
- Option C: Intercostal nerve block at one level — single dermatome coverage; insufficient for major abdominal surgery.
- Option D: Correct — Thoracic epidural catheter provides excellent continuous multilevel analgesia for open abdominal surgery.

Final Answer: Epidural analgesia (thoracic epidural catheter) $\Rightarrow$ D

Answer: (D) Go Back to Q 292

Q293.

Solution

Concept — Pneumoperitoneum on erect CXR: The most specific sign of perforated hollow viscus on erect CXR is free air (pneumoperitoneum) visible as a crescentic lucency beneath the diaphragm — most often under the right hemidiaphragm (more easily seen against liver). Even 1 mL of free gas can be detected. Double bubble sign = duodenal atresia. Sail sign = thymic sail. Air-fluid levels in colon = large bowel obstruction (not perforation sign).

Step 1 — Identify the sign: Free air under right hemidiaphragm = pneumoperitoneum = most specific sign of perforated hollow viscus on erect CXR.

Why other options are wrong:

- Option A: Correct — Air under the right hemidiaphragm (pneumoperitoneum) is the pathognomonic sign of hollow viscus perforation on erect CXR.
- Option B: Air-fluid levels in colon — bowel obstruction, not perforation specifically.
- Option C: Double bubble sign — on AXR in neonates; indicates duodenal atresia; not perforation.
- Option D: Sail sign — thymic sail sign in children; not related to perforation.

Final Answer: Air under the right hemidiaphragm (pneumoperitoneum) $\Rightarrow$ A

Answer: (A) Go Back to Q 293

Q294.

Solution

Concept — Snowman/figure-of-8 heart sign: Total anomalous pulmonary venous connection (TAPVC) supracardiac type: the anomalous vertical vein + left SVC form a large structure above the heart, creating the upper ball of the "snowman." The heart and cardiac shadow form the lower ball. This is the "snowman" or "figure-of-8" appearance on CXR. TAPVC causes cyanosis from birth as all pulmonary venous return drains into systemic veins.

Step 1 — Match X-ray sign to diagnosis: Snowman/figure-of-8 cardiac silhouette + neonatal cyanosis = TAPVC (supracardiac type). The anomalous connection above the heart creates the snowman appearance.

Why other options are wrong:

- Option A: TOF — boot-shaped heart (coeur en sabot); decreased pulmonary markings.
- Option B: Correct — TAPVC supracardiac: snowman sign from anomalous vertical vein + cardiac shadow.
- Option C: TGA — egg-on-side appearance; narrow superior mediastinum; no snowman.
- Option D: Coarctation — rib notching in older children; figure-3 sign on barium swallow; not snowman.

Final Answer: TAPVC (supracardiac type) $\Rightarrow$ B

Answer: (B) Go Back to Q 294

Q295.

Solution

Concept — Double bubble sign: The double bubble sign on AXR = two gas-filled structures (stomach + proximal duodenum separated by the pylorus) with no distal bowel gas. It is **pathognomonic** of duodenal atresia. Associated with Down syndrome (trisomy 21) in 30% of cases. Caused by failure of recanalisation of the duodenal lumen during fetal development.

Step 1 — Diagnose: Polyhydramnios + bilious vomiting from first feed + double bubble AXR + no distal gas + Down syndrome features = duodenal atresia.

Why other options are wrong:

- Option A: Pyloric stenosis — presents at 3–6 weeks with non-bilious projec-

tile vomiting; string sign on barium; no double bubble.
- Option B: Jejunal atresia — multiple air-fluid levels (triple bubble or more), not double bubble; no Down syndrome association.
- Option C: Correct — Duodenal atresia: double bubble sign + Down syndrome association + bilious vomiting from birth.
- Option D: Malrotation with midgut volvulus — double bubble possible but bile-stained vomiting + ischaemic bowel; urgent surgical emergency; USS with Doppler (whirlpool sign); less classic double bubble.

Final Answer: Duodenal atresia ⇒ C

Answer: (C) Go Back to Q 295

Q296.

Solution

Concept — Severe contrast reaction treatment: Severe anaphylactoid contrast reactions (urticaria + angioedema + stridor + hypotension) = anaphylaxis. Treatment: IM adrenaline (epinephrine) 0.5 mg 1:1000 (first and most important), IV fluids for hypotension, oxygen, antihistamines + corticosteroids (secondary). Epinephrine reverses bronchospasm, laryngeal oedema, and hypotension.

Step 1 — Immediate treatment: Anaphylaxis from contrast = IM adrenaline 0.5 mg (1:1000) immediately. Hydrocortisone and antihistamines are adjuncts, not primary treatment.

Why other options are wrong:

- Option A: IV hydrocortisone + antihistamine — adjuncts that take time to work; not adequate for acute anaphylaxis with hypotension and stridor.
- Option B: IV normal saline alone — for mild hypotension only; does not treat anaphylaxis.
- Option C: Oral prednisolone — completely inadequate for severe anaphylaxis; too slow onset.
- Option D: Correct — IM adrenaline 0.5 mg (1:1000) + IV fluids is the immediate life-saving treatment.

Final Answer: IM adrenaline (epinephrine) 0.5 mg + IV fluids ⇒ D

Answer: (D) Go Back to Q 296

Q297.

Solution

Concept — Thymic sail sign: The sail sign (thymic sail sign) is a normal radiological finding in infants and young children. The thymus occupies the anterior superior mediastinum; on CXR it appears as a triangular opacity in the right upper mediastinum with a wavy (undulating) lateral border due to indentation by ribs — the "thymic wave sign." It is a normal variant and should not be confused with mediastinal pathology.

Step 1 — Identify sign: Triangular opacity in right upper mediastinum + wavy lateral border in a young child = sail sign (thymic sail sign). Normal variant.

Why other options are wrong:

- Option A: Correct — Sail sign (thymic sail sign): triangular right upper mediastinal opacity with wavy border in children.
- Option B: Hampton's hump — wedge-shaped pleural-based opacity in pulmonary embolism; not mediastinal.
- Option C: Westermark's sign — oligaemia (decreased vascular markings) distal to a pulmonary embolism; not this.
- Option D: Air bronchogram sign — branching air-filled bronchi within opacified lung; indicates alveolar consolidation; not mediastinal.

Final Answer: Sail sign (thymic sail sign) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q 297

Q298.

Solution

Concept — Pancreatic head carcinoma surgery: Carcinoma of the head of pancreas is treated surgically by Whipple's procedure (pancreaticoduodenectomy) — resection of the head of pancreas, duodenum, distal common bile duct, gallbladder, and distal stomach. It is the only potentially curative surgery. Only 10–20% are resectable at presentation.

Step 1 — Surgical procedure: Resectable pancreatic head carcinoma = Whipple's procedure (pancreaticoduodenectomy). ERCP stenting alone is palliative for obstructed jaundice.

Why other options are wrong:

- Option A: Correct — Whipple's procedure (pancreaticoduodenectomy) is the

curative surgery for resectable pancreatic head carcinoma.

- Option B: Distal pancreatectomy — for carcinoma of the body and tail, not head of pancreas.
- Option C: Billroth II gastrectomy — for gastric resection; not for pancreatic head lesions.
- Option D: ERCP with stenting — palliative measure to relieve jaundice; not curative.

Final Answer: Whipple's procedure (pancreaticoduodenectomy) $\Rightarrow$ A

Answer: (A) Go Back to Q 298

Q299.

Solution

Concept — Aortic dissection investigation: Classic presentation of acute aortic dissection (Type A): tearing chest pain radiating to the back + unequal blood pressures + aortic regurgitation murmur + normal ECG (unlike MI). CT angiography of the aorta (CT aortogram) is the gold standard investigation — shows the intimal flap, true and false lumen, and extent of dissection.

Step 1 — Best investigation: Tearing chest pain + unequal BP + AR murmur + normal ECG = aortic dissection. CT aortogram is the gold standard — fast, non-invasive, widely available.

Why other options are wrong:

- Option A: ECG + troponin — for myocardial infarction; ECG normal here; troponin may be mildly elevated in dissection.
- Option B: Correct — CT angiography of the aorta is the gold standard for diagnosing aortic dissection.
- Option C: Coronary angiography — for CAD/MI; aortic dissection presenting as MI requires CT first to rule out dissection before angio.
- Option D: Transthoracic echo — can show aortic regurgitation and proximal dissection but misses distal dissection; not gold standard alone.

Final Answer: CT angiography of the aorta (CT aortogram) $\Rightarrow$ B

Answer: (B) Go Back to Q 299

Q300.

Solution

Concept — Sepsis-6 bundle management: The Sepsis-6 (Surviving Sepsis Campaign) bundle to be completed within 1 hour: (1) Measure lactate, (2) Blood cultures $\times 2$, (3) IV broad-spectrum antibiotics, (4) IV crystalloid 30 mL/kg for hypotension/lactate ≥ 4 mmol/L, (5) Oxygen, (6) Measure urine output. Antibiotics within 1 hour reduce mortality by 7% per hour of delay.

Step 1 — Critical immediate intervention: Lactate 6.2 + BP 80/50 + fever + abdominal source = septic shock. Priority = IV broad-spectrum antibiotics within 1 hour + IV fluid resuscitation + oxygen — the Sepsis-6 core interventions.

Why other options are wrong:

- Option A: CT abdomen before antibiotics — diagnostic imaging should NEVER delay antibiotic administration; give antibiotics first then image.
- Option B: Norepinephrine first without antibiotics — vasopressors are for refractory hypotension; antibiotics should be given simultaneously with resuscitation.
- Option C: Central line for CVP monitoring — CVP monitoring is no longer a sepsis bundle requirement; focus is on clinical assessment, lactate clearance. Antibiotics take priority.
- Option D: Correct — IV broad-spectrum antibiotics within 1 hour + IV fluid resuscitation + oxygen = the critical immediate Sepsis-6 interventions.

Final Answer: IV broad-spectrum antibiotics + IV fluids + oxygen $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q 300

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	2	B	3	A	4	C	5	D
6	A	7	C	8	B	9	D	10	A
11	C	12	D	13	A	14	B	15	C
16	D	17	B	18	A	19	B	20	C
21	D	22	A	23	C	24	B	25	C
26	D	27	A	28	B	29	C	30	D
31	A	32	B	33	C	34	D	35	A
36	B	37	C	38	D	39	A	40	C
41	B	42	A	43	C	44	B	45	D
46	A	47	C	48	B	49	D	50	C
51	A	52	C	53	B	54	D	55	A
56	C	57	D	58	A	59	B	60	C
61	A	62	D	63	B	64	C	65	A
66	D	67	B	68	C	69	A	70	D
71	B	72	C	73	A	74	D	75	B
76	C	77	A	78	D	79	B	80	C
81	D	82	A	83	B	84	C	85	D
86	A	87	C	88	B	89	D	90	C
91	A	92	B	93	D	94	C	95	A
96	D	97	B	98	C	99	A	100	D
101	B	102	C	103	A	104	D	105	C
106	B	107	C	108	D	109	A	110	B
111	C	112	A	113	D	114	B	115	C
116	B	117	D	118	B	119	C	120	A
121	A	122	B	123	C	124	D	125	A
126	B	127	D	128	D	129	A	130	B
131	C	132	D	133	A	134	B	135	C
136	D	137	A	138	B	139	C	140	D
141	A	142	B	143	C	144	D	145	A
146	B	147	C	148	D	149	A	150	B
151	C	152	D	153	A	154	B	155	C
156	D	157	A	158	B	159	C	160	D
161	A	162	B	163	C	164	D	165	A
166	B	167	C	168	D	169	A	170	B
171	C	172	D	173	A	174	B	175	C
176	D	177	A	178	B	179	C	180	D
181	A	182	B	183	C	184	D	185	A
186	B	187	C	188	D	189	A	190	B
191	C	192	D	193	A	194	B	195	C
196	D	197	A	198	B	199	C	200	D
201	A	202	B	203	C	204	D	205	A
206	B	207	C	208	D	209	C	210	B
211	C	212	D	213	[illegible]	214	B	215	C
216	D	217	A	218	[illegible]	219	C	220	D
221	A	222	B	223	C	224	D	225	A