Physiology

Sample Paper – 10

Questions:	25	**Duration:**	45 min
Total Marks:	100	**Marking:**	+4 / −1

Topics: Respiratory · Digestive · Musculoskeletal · Renal · Cardiovascular · Reproductive · Biochemistry

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Quick Reference

Instructions

- This paper has **25 questions** carrying **100 marks**.
- Each correct answer: **+4 marks**. Each wrong answer: **−1 mark**. Unattempted: **0**.
- Suggested time: **45 minutes**. All questions are single-correct MCQs.
- Covers: Respiratory, Digestive, Musculoskeletal, Renal, Cardiovascular, Reproductive systems, Biochemistry.
- Click a question number in the answer key to jump to its solution.

Q1. Total lung capacity (TLC) is a fundamental respiratory volume. Which of the following statements most accurately defines TLC and its normal value in an average adult male?

(A) TLC is the volume of air remaining in the lungs after a maximal forced expiration; it equals residual volume (RV) alone, approximately 1.2 L.

(B) TLC is the volume of air inhaled in a single normal breath at rest (tidal volume), approximately 0.5 L.

(C) TLC is the maximum volume of air that can be forcibly exhaled after a maximal inspiration (vital capacity), approximately 4.8 L, excluding residual volume.

(D) TLC is the total volume of gas in the lungs after a maximal inspiration; it equals the sum of all lung volumes: tidal volume (TV) + inspiratory reserve volume (IRV) + expiratory reserve volume (ERV) + residual volume (RV), approximately 6 L in an average adult male.

Q2. Pepsin is the principal proteolytic enzyme of the stomach, secreted as the inactive zymogen pepsinogen by chief cells. At which pH does pepsin exhibit its optimal proteolytic activity?

(A) pH 7.0–8.0 — the neutral to mildly alkaline range typical of pancreatic secretions in the duodenum.

(B) pH 4.0–5.0 — mildly acidic conditions such as those in the distal stomach after partial neutralisation.

(C) pH 1.5–2.0 — the highly acidic gastric lumen where pepsin (activated from pepsinogen by gastric HCl) has peak proteolytic activity; it is irreversibly denatured above pH 5, so protein digestion ceases once chyme is neutralised in the duodenum.

(D) pH 6.5–7.0 — the near-neutral pH of saliva, where salivary proteases function optimally.

Q3. The classic liver lobule is the histological unit of the liver, with the central vein at its centre and portal triads at its corners. Which of the following correctly lists the structures that constitute the **portal triad**?

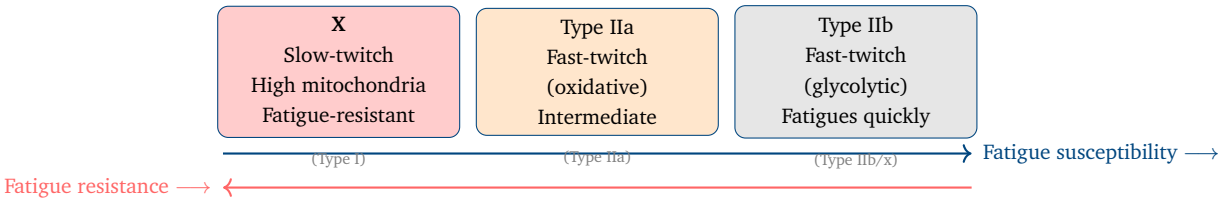
(A) Central vein (hepatic venule), hepatic sinusoid, and Kupffer cell cluster.

(B) Hepatic vein, lymphatic vessel, and nerve fibre bundle.

(C) Portal arteriole, bile canaliculus, and hepatic sinusoid.

(D) A branch of the portal vein, a branch of the hepatic artery (portal arteriole), and a bile ductule — the portal triad supplies oxygenated blood and nutrients to hepatocytes and drains bile; the central vein (hepatic venule) is at the lobule centre.

Q4. The diagram below illustrates three major skeletal muscle fibre types arranged by their contractile and metabolic properties. Which fibre type, labelled **X** in the diagram, has the highest mitochondrial density, greatest capillary supply, abundant myoglobin, and highest fatigue resistance, making it ideal for sustained aerobic activity?



(A) Fast-twitch glycolytic (Type IIb) fibres — they rely on anaerobic glycolysis, have few mitochondria, and generate powerful but short bursts of force before fatigue (sprinting).

(B) Fast-twitch oxidative (Type IIa) fibres — intermediate between Type I and Type IIb, they use both aerobic and anaerobic metabolism and are moderately fatigue-resistant.

(C) Slow-twitch (Type I, oxidative) fibres — they have the highest mitochondrial density, greatest capillary supply, abundant myoglobin (red colour), high fatigue resistance, and are specialised for sustained aerobic activity (e.g., postural muscles, marathon running).

(D) Intermediate fibres — a hypothetical fourth fibre type with equal aerobic and anaerobic capacity found only in cardiac muscle.

Q5. The spleen plays a critical role in the quality control of circulating red blood cells (RBCs). Which of the following best describes the primary splenic function in RBC biology?

(A) The spleen synthesises new erythrocytes from haematopoietic stem cells throughout adult life, replacing those lost to normal ageing.

(B) The spleen stores excess RBCs in a reservoir and releases them during exercise or haemorrhage to increase oxygen-carrying capacity rapidly.

(C) The spleen removes old, damaged, and abnormally shaped red blood cells through phagocytosis by resident macrophages in the red pulp; normal RBC lifespan is approximately 120 days; haemoglobin is recycled — globin yields amino acids, haem yields bilirubin, which is excreted in bile.

(D) The spleen coats aged RBCs with complement proteins, triggering their lysis directly in the circulation without macrophage involvement.

Q6. Antidiuretic hormone (ADH, vasopressin) regulates water reabsorption in the renal collecting duct. When ADH secretion is completely suppressed — for example, following a large water load — what is the expected character of the urine produced?

(A) Hypo-osmolar (dilute) urine is produced — when ADH is absent or suppressed, the collecting duct remains impermeable to water, and the dilute tubular fluid (approximately

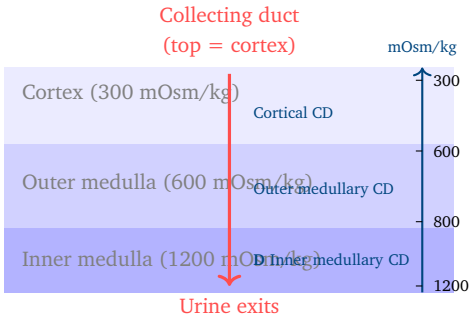
100 mOsm/kg leaving the ascending limb of the loop of Henle) is excreted unchanged, producing large volumes of dilute urine.

(B) Hyperosmolar (concentrated) urine is produced — the absence of ADH paradoxically activates aquaporin-2 channels, concentrating urine to 1200 mOsm/kg.

(C) Isotonic urine identical in osmolality to plasma (approximately 290 mOsm/kg) is always produced regardless of ADH levels.

(D) No urine is produced because suppression of ADH halts glomerular filtration via tubuloglomerular feedback.

Q7. The diagram below shows the renal medullary osmotic gradient from cortex to inner medulla, and the path of the collecting duct. In which tubular segment does the **greatest final concentration of urine** occur when ADH is maximally active?



(A) The cortical collecting duct — here, isotonic fluid equilibrates with the 300 mOsm/kg cortical interstitium; urine concentration is minimal at this level.

(B) The proximal convoluted tubule — most water reabsorption occurs here, so the greatest concentration is achieved at this early segment.

(C) The descending limb of the loop of Henle — fluid becomes concentrated as water leaves, but this is not the final point of concentration for urine.

(D) The inner medullary collecting duct — as tubular fluid travels through the collecting duct from cortex to inner medulla, water is progressively reabsorbed by osmosis (when ADH is present) into the hyperosmotic interstitium, concentrating urine to a maximum of approximately 1200 mOsm/kg at the inner medulla.

Q8. The smooth endoplasmic reticulum (SER) is a membranous organelle lacking ribosomes. In which of the following cell types is the SER **most abundant**, reflecting its key metabolic functions?

(A) Neurons — the SER produces myelin sheaths and neurotransmitter vesicles in large quantities.

(B) Erythrocytes — the SER handles haemoglobin synthesis and oxygen binding reactions in mature red blood cells.

(C) Steroidogenic cells (adrenal cortex, gonads) and hepatocytes — the SER houses enzymes for steroidogenesis, lipid synthesis, and cytochrome P450-mediated drug/xenobiotic metabolism; it also sequesters intracellular Ca^{2+} in muscle cells (sarcoplasmic reticulum).

(D) Goblet cells of the intestinal mucosa — the SER packages mucin glycoproteins into secretory vesicles for mucus secretion.

Q9. A patient develops sudden-onset bronchospasm, urticaria, hypotension, and angioedema within minutes of receiving a penicillin injection. This life-threatening systemic reaction is an example of which type of hypersensitivity?

(A) Type IV (delayed-type / cell-mediated) hypersensitivity — mediated by sensitised T lymphocytes; reaction peaks at 48–72 hours (e.g., tuberculin skin test, contact dermatitis).

(B) Type I (immediate / IgE-mediated) hypersensitivity — prior sensitisation causes mast cells and basophils to display antigen-specific IgE; re-exposure cross-links IgE triggering immediate degranulation releasing histamine, tryptase, and leukotrienes, producing vasodilation, bronchospasm, angioedema, and shock within minutes.

(C) Type II (antibody-dependent cytotoxic) hypersensitivity — IgG or IgM antibodies bind to cell-surface antigens and trigger complement-mediated lysis (e.g., haemolytic transfusion reactions, Goodpasture syndrome).

(D) Type III (immune complex-mediated) hypersensitivity — antigen-antibody complexes deposit in tissues, activate complement, and cause inflammation (e.g., serum sickness, lupus nephritis).

Q10. In a healthy individual, glucose does not normally appear in the urine despite being freely filtered at the glomerulus. Above which plasma glucose concentration does glucosuria (glucose in urine) first occur?

(A) Approximately 180 mg/dL (10 mmol/L) — this is the plasma glucose level above which the renal tubular maximum (T_m) for glucose reabsorption is exceeded and glucose appears in urine; in diabetes mellitus, persistent hyperglycaemia above this threshold causes osmotic diuresis.

(B) Approximately 90 mg/dL (5 mmol/L) — normal fasting blood glucose; the kidney begins to spill glucose at this physiological range.

(C) Approximately 300 mg/dL (16.7 mmol/L) — a severely hyperglycaemic level that must be sustained for several days before the renal threshold is reached.

(D) Approximately 70 mg/dL (3.9 mmol/L) — the lower limit of normal blood glucose; glucose appears in urine whenever blood glucose falls below this level.

Q11. During clinical examination, a patient is found to have markedly elevated jugular venous pressure (JVP), visible as distension of the internal jugular vein with the patient at 45 degrees. Which of the following conditions is the **most common** cause of elevated JVP?

(A) Severe anaemia — reduced oxygen-carrying capacity causes compensatory tachycardia and increased cardiac output, which raises venous return and JVP.

(B) Left heart failure — failure of the left ventricle causes pulmonary oedema that backs up fluid into the pulmonary veins, ultimately raising JVP.

(C) Hypovolaemia (dehydration) — reduced circulating blood volume increases venous tone, paradoxically raising JVP despite low filling pressures.

(D) Right heart failure — when the right ventricle fails to pump blood effectively into the pulmonary circulation, venous pressure backs up into the great veins and right atrium, raising JVP; other causes include cardiac tamponade, constrictive pericarditis, and superior vena cava obstruction.

Q12. The sinoatrial (SA) node generates the cardiac pacemaker potential through a spontaneous diastolic depolarisation called the "funny current" (I_f). Which ion channel and ionic mechanism underlies I_f?

(A) HCN (hyperpolarisation-activated cyclic nucleotide-gated) channels allow mixed Na^+/K^+ influx during diastole — this inward current slowly depolarises the SA node membrane from about -60 mV toward threshold (≈ -40 mV), producing the pacemaker potential; sympathetic stimulation increases I_f (faster rate), parasympathetic decreases it.

(B) L-type voltage-gated Ca^{2+} channels carry I_f — they open at -60 mV and allow large Ca^{2+} influx that alone drives the pacemaker potential independently of Na^+.

(C) Inward-rectifier K^+ channels (I_{K1}) generate I_f by allowing K^+ to flow inward during diastole, making the cell progressively more positive.

(D) Voltage-gated Na^+ channels (Nav1.5) produce I_f — they activate at -90 mV, the same as in ventricular myocytes, driving phase 0 depolarisation rather than the slow pacemaker potential.

Q13. Gluconeogenesis is the metabolic pathway that synthesises glucose from non-carbohydrate precursors, primarily in the liver and kidney cortex during fasting. Which of the following correctly lists the principal substrates (precursors) for gluconeogenesis?

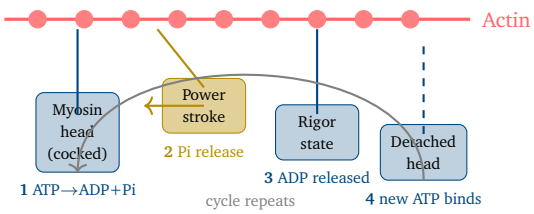
(A) Fatty acids and ketone bodies — beta-oxidation of long-chain fatty acids directly feeds carbon skeletons into gluconeogenesis, making fat the primary gluconeogenic fuel.

(B) Lactate, glycerol, and glucogenic amino acids — lactate (from anaerobic glycolysis in muscle and RBCs via the Cori cycle), glycerol (from triglyceride hydrolysis in adipose tissue), and most amino acids except leucine and lysine (which are purely ketogenic) can be converted to glucose by the liver and kidney cortex.

(C) Glucose-6-phosphate and glycogen — glycogenolysis and gluconeogenesis use the same substrates; glucose-6-phosphate from muscle glycogen is the primary gluconeogenic precursor.

(D) Succinyl-CoA and fumarate — these TCA cycle intermediates are the exclusive carbon sources for hepatic gluconeogenesis during prolonged fasting.

Q14. The diagram below shows four numbered steps of the crossbridge cycle in skeletal muscle contraction. Which step, labelled **2**, represents the **power stroke** that generates force and shortens the sarcomere?



(A) Step 1 — ATP hydrolysis (ATP $\rightarrow$ ADP + Pi) cocks (energises) the myosin head into a high-energy conformation; no force is generated yet.

(B) Step 2 — The power stroke occurs after myosin head binds actin and Pi is released; the myosin head pivots, pulling the actin filament toward the M-line and generating force; ADP then dissipates, and a new ATP must bind for the cycle to repeat.

(C) Step 3 — ADP release from the rigor complex generates the power stroke by a conformational change after the head has already pivoted to its low-energy position.

(D) Step 4 — New ATP binding to the myosin head generates the power stroke by providing energy for the actin–myosin interaction to produce force.

Q15. Vitamin B_{12} (cobalamin) is an essential water-soluble vitamin required for DNA synthesis and neurological function. What is the unique structural feature that distinguishes it from all other vitamins?

(A) It contains a porphyrin ring identical to the haem group in haemoglobin, but substituted with zinc instead of iron, making it the only metalloenzyme cofactor derived from a vitamin.

(B) It is the only vitamin that is a lipid (fat-soluble steroid), making it structurally distinct from all other B-group vitamins, which are water-soluble.

(C) Cobalt (Co) — vitamin B_{12} is the only vitamin containing a metal ion at its core; the cobalt atom is co-ordinated within a corrin ring (similar to porphyrin) and is essential for the vitamin's enzymatic activity (methionine synthase and methylmalonyl-CoA mutase); deficiency causes megaloblastic anaemia and subacute combined degeneration of the spinal cord.

(D) It contains a selenium atom at its active site, making it the only seleno-vitamin and explaining its role as an antioxidant similar to glutathione peroxidase.

Q16. The large intestine (colon) receives approximately 1.5 litres of liquid chyme from the ileum each day but produces only about 100–200 mL of faeces. What is the **primary physiological function** of the colon?

(A) Enzymatic digestion of complex carbohydrates and proteins — the colon contains numerous digestive enzymes secreted by colonocytes that complete the digestion begun in the small intestine.

(B) Absorption of water and electrolytes (Na^+, Cl^-) from the remaining liquid chyme, converting it to semi-solid faeces for storage and elimination; the colon also houses the gut microbiome which ferments undigested fibres, producing short-chain fatty acids and vitamins K and B_{12}, but nutrient digestion and absorption are minimal compared to the small intestine.

(C) Secretion of bile salts and bicarbonate — the colon is the primary site of bile salt synthesis and alkaline mucus secretion, neutralising acids produced by bacterial fermentation.

(D) Active absorption of fat-soluble vitamins (A, D, E, K) and long-chain fatty acids via specialised absorptive cells (colonocytes) using chylomicron formation.

Q17. The zona pellucida is the glycoprotein coat surrounding the oocyte. What critical event involving the zona pellucida occurs immediately **after** the first sperm successfully fertilises the egg?

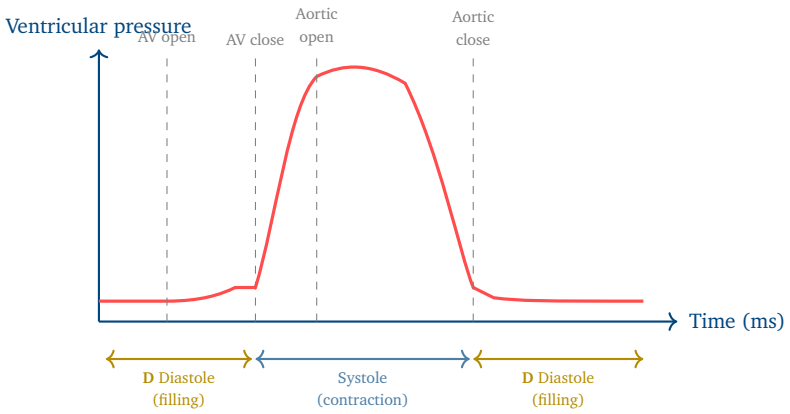
(A) The zona pellucida dissolves entirely within seconds of sperm-egg fusion, allowing the fertilised egg to immediately implant in the endometrium without further barriers.

(B) Prevention of polyspermy — the zona pellucida undergoes the "zona reaction" after the first sperm fuses with the egg: cortical granule enzymes (released by exocytosis) harden the zona pellucida and inactivate sperm receptors, preventing additional sperm from penetrating and ensuring normal diploid fertilisation.

(C) The zona pellucida thickens to protect the embryo from immune rejection by the maternal immune system during the entire first trimester.

(D) The zona pellucida triggers meiosis II completion in the oocyte by releasing zona proteins that act as second messengers inside the egg cytoplasm.

Q18. The diagram below illustrates the phases of the cardiac cycle. During which phase, labelled **D**, does ventricular filling occur?



(A) Systole — ventricular filling is an active process driven by myocardial contraction; it occurs simultaneously with the ejection of blood into the aorta.

(B) Isovolumetric contraction — the brief phase between mitral valve closure and aortic valve opening during which ventricular volume increases to fill the chamber.

(C) Isovolumetric relaxation — ventricular filling begins immediately when ventricular pressure starts to fall, before any valves open.

(D) Diastole — ventricular filling occurs during diastole: first rapid filling (when AV valves open after ventricular pressure falls below atrial pressure), then slow filling (diastasis), and finally atrial systole (atrial kick, approximately 20% of filling); total filling time is approximately 500 ms at resting heart rate; tachycardia reduces diastolic filling time.

Q19. When performing a lumbar puncture (spinal tap), clinicians insert the needle below the termination of the spinal cord to avoid injuring it. At which vertebral level does the adult spinal cord (conus medullaris) normally terminate?

(A) C7–T1 — the cervical enlargement, where the brachial plexus originates; puncture below this level is necessary for safe access.

(B) L1–L2 (the conus medullaris) — the spinal cord ends here in adults; below this level the vertebral canal contains only the cauda equina (bundle of nerve roots); lumbar punctures are safely performed at L3–L4 or L4–L5 to avoid injuring the cord.

(C) T12 — the thoracolumbar junction; the cord ends at the same level as the last thoracic vertebra in all adults.

(D) S2–S3 — the sacral level, where the filum terminale anchors the cord to the coccyx; punctures at this level are needed for caudal anaesthesia.

Q20. In endocrinology, "trophic" (or "tropic") hormones are defined by their target and mechanism of action. Which of the following correctly defines trophic hormones and gives accurate examples?

(A) Trophic (tropic) hormones stimulate other endocrine glands to produce and secrete their hormones: TSH stimulates the thyroid, ACTH stimulates the adrenal cortex, FSH and LH

stimulate the gonads — all are secreted by the anterior pituitary in response to hypothalamic releasing hormones.

(B) Trophic hormones promote cell growth and hypertrophy in non-endocrine target tissues; growth hormone (GH) is the prototype, acting directly on bone and muscle without an intermediate endocrine gland.

(C) Trophic hormones are lipid-soluble steroid hormones that cross cell membranes and bind nuclear receptors to regulate gene transcription; cortisol and oestrogen are primary examples.

(D) Trophic hormones are released from the posterior pituitary (neurohypophysis) in response to osmotic signals; ADH and oxytocin are the two trophic hormones that regulate endocrine gland function.

Q21. The atrioventricular (AV) node is a specialised conducting structure at the junction between the atria and ventricles. It intentionally slows electrical conduction — a physiologically important delay. Approximately how long is the AV node conduction delay, and what is its clinical significance?

(A) Approximately 0.4 seconds — this long delay allows the atria to complete two full contractions before ventricular systole begins, doubling the time for ventricular filling.

(B) Approximately 0.01 seconds — the AV node conducts almost instantaneously; the ECG PR interval is instead caused by slow conduction in the bundle of His.

(C) Approximately 0.5 seconds — a half-second delay corresponding to the QRS complex duration on the ECG, representing total ventricular activation time.

(D) Approximately 0.1 seconds (100 ms) — the AV node deliberately slows conduction from atria to ventricles, allowing atrial systole to complete ventricular filling before ventricular contraction begins; on the ECG, this manifests as the PR segment between the P wave and QRS complex.

Q22. Swallowing (deglutition) is a complex coordinated reflex involving multiple cranial nerves. Which of the following correctly identifies the cranial nerves involved in the swallowing reflex?

(A) Cranial nerves V (trigeminal — jaw/tongue muscles), VII (facial — buccinator), IX (glossopharyngeal — pharyngeal sensation/stylopharyngeus), X (vagus — pharyngeal/laryngeal muscles, oesophagus), and XII (hypoglossal — tongue propulsion) all participate in the coordinated act of swallowing.

(B) Only cranial nerves IX (glossopharyngeal) and X (vagus) are involved in swallowing; all other cranial nerve contributions are negligible and not clinically relevant.

(C) Cranial nerves II (optic), III (oculomotor), and IV (trochlear) coordinate the visual and motor aspects of swallowing by directing food toward the pharynx.

(D) Cranial nerves XI (accessory) and XII (hypoglossal) alone control swallowing; the accessory nerve drives the pharyngeal constrictors and the hypoglossal drives the larynx.

Q23. The glomerular filtration barrier normally prevents plasma proteins such as albumin from passing into the filtrate, even though albumin is a relatively small protein. What is the principal mechanism that prevents albumin filtration?

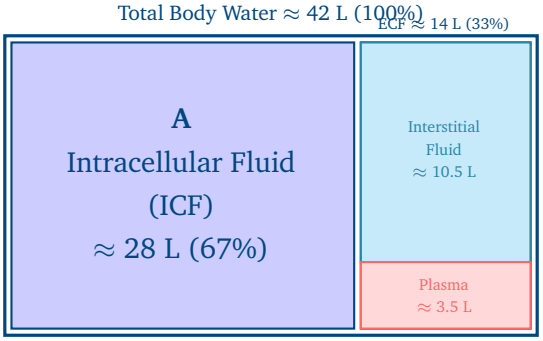
(A) Albumin is too large to pass through the glomerular pores under any circumstances; the molecular weight of 69 kDa is above the absolute size cut-off of the filtration barrier regardless of charge.

(B) Albumin binds to specific carrier proteins in the afferent arteriole that prevent it from reaching the glomerular filtration surface.

(C) The glomerular filtration barrier has three layers: the fenestrated endothelium (prevents cells), the glomerular basement membrane (GBM), and podocyte slit diaphragms; the negatively charged glycocalyx of the GBM electrostatically repels anionic albumin (which is also negatively charged), preventing its filtration — loss of this charge barrier causes nephrotic syndrome.

(D) Peritubular capillary oncotic pressure is so high that it pulls all filtered albumin back from Bowman's space before it can enter the tubular lumen.

Q24. The diagram below represents the distribution of total body water (TBW) among fluid compartments in a 70 kg adult male (TBW ≈ 42 L). Which compartment, labelled **A**, constitutes the **largest** single fluid compartment?



(A) Intracellular fluid (ICF) accounts for approximately 2/3 (about 67%) of total body water — in a 70 kg adult with ≈42 L TBW: ICF ≈ 28 L (2/3), extracellular fluid (ECF) ≈ 14 L (1/3), of which interstitial fluid ≈ 10.5 L and plasma ≈ 3.5 L.

(B) Plasma is the largest fluid compartment, accounting for approximately 60% of TBW; it circulates continuously through the cardiovascular system and is the primary reservoir of body water.

(C) Interstitial fluid is the largest body fluid compartment, comprising approximately 50% of TBW; it surrounds all cells and is the immediate environment of every cell in the body.

(D) Transcellular fluid (CSF, synovial fluid, aqueous humour, pleural fluid) is the largest compartment at approximately 40% of TBW, forming the essential "third space" of fluid distribution.

Q25. Histamine is a potent vasoactive amine mediator of inflammation and allergic reactions. In which cells is histamine stored preformed, and what triggers its release?

(A) Mast cells (in tissues, especially skin, gut, and airways) and basophils (circulating granulocytes) store histamine preformed in cytoplasmic granules; upon activation (IgE cross-linking, complement, trauma), they degranulate and release histamine, causing vasodilation, increased capillary permeability, bronchoconstriction, and itch.

(B) Platelets and eosinophils are the principal histamine-storing cells; histamine is released when platelets aggregate at sites of vascular injury during haemostasis.

(C) Neutrophils and monocytes synthesise histamine de novo (from histidine) upon activation by bacterial lipopolysaccharide; no preformed stores exist in these cells.

(D) Enterochromaffin cells (ECL cells) of the gastric mucosa store histamine and release it only in response to gastrin to stimulate acid secretion; circulating histamine in allergy is produced elsewhere.

Answer Key — Physiology Sample Paper 10

1	D	2	C	3	D	4	C	5	C
6	A	7	D	8	C	9	B	10	A
11	D	12	A	13	B	14	B	15	C
16	B	17	B	18	D	19	B	20	A
21	D	22	A	23	C	24	A	25	A

Detailed Solutions

Solution

Q1. (D) TLC = TV + IRV + ERV + RV ≈ 6 L

Total lung capacity (TLC) is the volume of gas in the lungs at the end of a maximal inspiration. It is the sum of all four lung volumes: Tidal Volume (TV, ≈0.5 L) + Inspiratory Reserve Volume (IRV, ≈3.0 L) + Expiratory Reserve Volume (ERV, ≈1.2 L) + Residual Volume (RV, ≈1.2 L) ≈ **6 L**. Vital capacity (VC = TV + IRV + ERV ≈ 4.8 L) does *not* include RV; RV cannot be measured by simple spirometry (requires gas dilution or body plethysmography). TLC is reduced in restrictive lung disease (fibrosis) and may be increased in emphysema (air trapping).

Solution

Q2. (C) pH 1.5–2.0

Pepsin is the major gastric protease. Pepsinogen (inactive) is secreted by chief cells of gastric glands and is autocatalytically cleaved to pepsin in the highly acidic gastric lumen (pH 1.5–2.0 maintained by HCl from parietal cells). Pepsin is optimally active at **pH 1.5–2.0** and hydrolyses peptide bonds adjacent to aromatic/large aliphatic residues, initiating protein digestion. It is *irreversibly denatured* above pH 5 — so protein digestion ceases as chyme is alkalinised by pancreatic bicarbonate in the duodenum. Protein digestion is completed by pancreatic proteases (trypsin, chymotrypsin, elastase) at the near-neutral duodenal pH.

Solution

Q3. (D) Portal vein branch + hepatic arteriole + bile ductule

The **portal triad** (also called portal tract) is located at the corners of each classic hexagonal liver lobule. It contains: (1) a *branch of the portal vein* (draining nutrient-rich but relatively deoxygenated blood from the gut), (2) a *hepatic arteriole* (branch of the hepatic artery, supplying oxygenated blood), and (3) a *bile ductule* (draining bile produced by hepatocytes, flowing in the opposite direction to blood). Blood flows from the portal triad *inward* through sinusoids lined by Kupffer cells toward the *central vein* (zone 1 $\rightarrow$ zone 2 $\rightarrow$ zone 3 in acinar classification). This architecture means periportal hepatocytes (zone 1) receive the most oxygenated blood and are most vulnerable to hepatotoxins entering via the portal vein; centrilobular hepatocytes (zone 3) are most susceptible to ischaemic injury.

Solution

Q4. (C) Slow-twitch (Type I) oxidative fibres

Skeletal muscle fibres are classified into three main types based on contractile speed and metabolic profile:

- **Type I (slow-twitch, oxidative) — fibre X**: High mitochondrial density, rich capillary network, abundant *myoglobin* (gives red colour), low myosin-ATPase activity (slow twitch), high fatigue resistance. Used in sustained aerobic activity: postural muscles, endurance exercise (marathon, cycling).
- **Type IIa (fast-twitch, oxidative-glycolytic)**: Intermediate properties; moderate mitochondria; used in middle-distance activities.
- **Type IIb/IIx (fast-twitch, glycolytic)**: Few mitochondria, little myoglobin (pale), fast myosin-ATPase, powerful but rapidly fatigue; used in sprinting and heavy lifting.

Type I fibres are recruited first (Henneman size principle) for low-force, sustained tasks; Type II fibres are recruited for high-force, rapid movements.

Solution

Q5. (C) Spleen removes aged/damaged RBCs by macrophage phagocytosis

The **spleen** serves as the quality-control filter for circulating erythrocytes. In the *red pulp*, macrophage-lined splenic sinusoids trap RBCs that are: old (>120 days), deformed (spherocytes, sickle cells), opsonised by IgG or complement, or have rigid membranes. Macrophages phagocytose these cells and recycle their components: *globin* chains are broken into amino acids returned to the amino acid pool; *haem* is converted to biliverdin $\rightarrow$ bilirubin (transported to liver, conjugated, excreted in bile); *iron* is retained and recycled via transferrin for new erythropoiesis. In haemolytic anaemia, splenic sequestration is accelerated, causing splenomegaly. Splenectomy leads to persistence of Howell-Jolly bodies and target cells in the blood.

Solution

Q6. (A) Hypo-osmolar (dilute) urine

ADH (arginine vasopressin) acts on principal cells of the collecting duct by binding V2 receptors → cAMP → PKA → insertion of aquaporin-2 (AQP2) water channels into the apical membrane → water reabsorption into the hyperosmotic medullary interstitium. When ADH is **absent** (suppressed by water loading): AQP2 channels are internalised, the collecting duct is impermeable to water, and the dilute fluid leaving the ascending limb (≈100 mOsm/kg) passes through unchanged → large volumes of dilute, hypo-osmolar urine (up to 18 L/day). This is the basis of *primary polydipsia* and is mimicked pathologically by *central diabetes insipidus* (no ADH) and *nephrogenic diabetes insipidus* (no response to ADH).

Solution

Q7. (D) Inner medullary collecting duct

The **medullary osmotic gradient** is established by the countercurrent multiplier (loop of Henle) and maintained by the countercurrent exchanger (vasa recta). Interstitial osmolality increases from ≈300 mOsm/kg in the cortex to ≈1200 mOsm/kg in the inner medulla. As tubular fluid travels along the **collecting duct** (when ADH is present):

- *Cortical CD*: fluid equilibrates to ≈300 mOsm/kg
- *Outer medullary CD*: fluid concentrates to ≈600 mOsm/kg
- *Inner medullary CD*: water reabsorption continues into the 1200 mOsm/kg interstitium; urea recycling (via UT-A1/UT-A3 transporters) further raises inner medullary osmolality → urine is maximally concentrated to ≈**1200 mOsm/kg** here.

Maximum urine concentration = inner medullary osmolality. Reduced medullary gradient (e.g., loop diuretics, chronic renal disease) limits maximum urinary concentration.

Solution

Q8. (C) Steroidogenic cells and hepatocytes

The **smooth endoplasmic reticulum (SER)** is richly developed in cells with high lipid metabolism activity:

- **Adrenal cortex, Leydig cells (testes), granulosa cells (ovaries)**: SER contains steroidogenic enzymes (CYP11A1 — cholesterol side-chain cleavage; CYP17A1; CYP21A2; HSD enzymes) for synthesis of glucocorticoids, mineralocorticoids, and sex hormones from cholesterol.
- **Hepatocytes**: SER houses cytochrome P450 enzymes (CYP3A4, CYP2D6, etc.) for phase I drug metabolism and detoxification; also synthesises cholesterol, triglycerides, and VLDL.
- **Muscle cells**: The SER is specialised as the *sarcoplasmic reticulum (SR)*, which sequesters and releases Ca^{2+} to regulate contraction.

The rough ER (with ribosomes) is abundant in secretory cells (pancreatic acini, plasma cells). The SER is absent or minimal in mature erythrocytes (anucleate) and neurons primarily use RER for protein synthesis.

Solution

Q9. (B) Type I (immediate / IgE-mediated) hypersensitivity

Anaphylaxis is the prototypical **Type I hypersensitivity** reaction. Mechanism:

1. *Sensitisation*: First exposure to antigen (e.g., penicillin) $\rightarrow$ Th2-driven IgE production $\rightarrow$ IgE binds FcεRI receptors on mast cells and basophils (no symptoms yet).
2. *Elicitation*: Re-exposure cross-links IgE on mast cells $\rightarrow$ within seconds, degranulation releases preformed mediators: **histamine, tryptase, heparin** (immediate phase); de novo synthesis of **leukotrienes C4/D4, prostaglandins, platelet-activating factor** (late phase).
3. *Effects*: Vasodilation + increased permeability (hypotension, urticaria, angioedema), bronchospasm (wheeze, dyspnoea), pruritus.

Treatment: **adrenaline (epinephrine) IM** is first-line. Compare: Type II (cytotoxic-IgG/IgM), Type III (immune complex), Type IV (delayed-T cell mediated, $>$24 h).

Solution

Q10. (A) Approximately 180 mg/dL (10 mmol/L)

Glucose is freely filtered at the glomerulus and **completely reabsorbed** in the proximal convoluted tubule under normal blood glucose levels, via SGLT2 (apical, 90%) and SGLT1 (apical, 10%) co-transporters with GLUT2 (basolateral). The tubular maximum (T_m) for glucose $\approx$ 375 mg/min. When plasma glucose exceeds $\approx$**180 mg/dL**, filtered load exceeds T_m and glucose "spills" into urine (*glycosuria*). In Type 1 and Type 2 diabetes mellitus, sustained hyperglycaemia above this threshold causes osmotic diuresis (glucose in tubular fluid retains water) $\rightarrow$ polyuria, polydipsia. SGLT2 inhibitors (e.g., empagliflozin) lower the renal threshold intentionally to treat diabetes. The renal threshold is slightly variable (lower in pregnancy, higher in chronic hyperglycaemia due to increased T_m).

Solution

Q11. (D) Right heart failure

Jugular venous pressure (JVP) reflects right atrial pressure. In **right heart failure** (cor pulmonale, RV infarct, left heart failure with RV involvement), the failing right ventricle cannot sustain forward flow, causing venous backpressure into the right atrium and great veins $\rightarrow$ elevated JVP (visible as jugular vein distension). Clinical signs: elevated JVP ($>$3 cm above sternal angle), peripheral oedema, hepatomegaly, ascites. Other causes of elevated JVP:

- Cardiac tamponade — pericardial fluid compresses the heart; *Kussmaul's sign* (JVP rises on inspiration) may be absent.
- Constrictive pericarditis — calcified pericardium restricts filling; Kussmaul's sign present.
- Superior vena cava (SVC) obstruction — non-pulsatile JVP elevation; caused by lung cancer, lymphoma.

Hypovolaemia causes a *decreased* JVP; anaemia causes compensatory tachycardia but normal or slightly decreased JVP.

Solution

Q12. (A) HCN channels — mixed Na^+/K^+ influx

SA node pacemaker cells **spontaneously depolarise** during diastole — the pacemaker potential — through the following ionic sequence:

1. **I_f (funny current)**: At the end of repolarisation (≈ -60 mV), **HCN (Hyperpolarisation-activated Cyclic Nucleotide-gated) channels** open, allowing inward mixed Na^+/K^+ current (net inward $\rightarrow$ slow depolarisation). Called "funny" because it activates on hyperpolarisation (opposite to most channels). cAMP from β1-adrenoceptor activation increases HCN channel opening speed (faster rate); ACh from M2 receptors decreases cAMP and slows I_f.
2. **T-type Ca^{2+} channels** (threshold ≈ -50 mV): Contribute to late phase of slow depolarisation.
3. **L-type Ca^{2+} channels**: Drive the upstroke (phase 0) of the SA node action potential (unlike ventricular cells which use fast Na^+ channels).

Ivabradine selectively blocks HCN channels $\rightarrow$ slows heart rate without affecting contractility.

Solution

Q13. (B) Lactate, glycerol, and glucogenic amino acids

Gluconeogenesis synthesises glucose from non-carbohydrate precursors during fasting, prolonged exercise, or stress. Principal substrates:

- **Lactate**: Generated by anaerobic glycolysis in muscles and RBCs; transported to liver and converted back to glucose via the *Cori cycle* (lactate $\rightarrow$ pyruvate $\rightarrow$ oxaloacetate $\rightarrow$ phosphoenolpyruvate $\rightarrow$ glucose).
- **Glycerol**: Released by lipolysis from adipose TAG; enters gluconeogenesis at glycerol-3-phosphate $\rightarrow$ DHAP.
- **Glucogenic amino acids**: Most amino acids (all except leucine and lysine, which are purely ketogenic) are converted to TCA intermediates (pyruvate, OAA, α-ketoglutarate, succinyl-CoA, fumarate) that feed gluconeogenesis; alanine and glutamine are most important quantitatively (glucose-alanine cycle).

Long-chain even-chain fatty acids cannot contribute net carbon to gluconeogenesis (acetyl-CoA cannot be converted to OAA in mammals); they do provide ATP to drive gluconeogenesis.

Solution

Q14. (B) The power stroke — Pi release causes myosin head to pivot

The **crossbridge cycle** (Lymn-Taylor cycle) has four key steps:

1. *ATP hydrolysis*: Myosin head binds ATP → ATPase activity → ADP + Pi remain bound → head "cocks" into high-energy (90°) conformation. (Step 1)
2. **Power stroke**: Cocked myosin head binds actin → **Pi release** → myosin head pivots to 45° low-energy conformation, pulling the actin filament toward the M-line by ≈10 nm → force is generated. (Step 2)
3. *ADP release*: ADP dissociates; head is in rigor (tightly bound to actin). (Step 3) — *This is the state of rigor mortis when ATP is depleted post-mortem.*
4. *Detachment*: New ATP binds myosin → reduces myosin-actin affinity → head detaches, ready for a new cycle. (Step 4)

Contraction requires Ca^{2+} (releases troponin inhibition of tropomyosin) and ATP (for both power stroke cycling and SERCA pump to re-sequester Ca^{2+}).

Solution

Q15. (C) Cobalt (Co) — the only metal-containing vitamin

Vitamin B_{12} (cobalamin) is the only vitamin with a *metal ion* at its core — a **cobalt (Co)** atom co-ordinated within a *corrin ring* (a tetrapyrrole ring system similar to porphyrin but more reduced and with one direct C–C bond). The cobalt can be in +1, +2, or +3 oxidation states. Two active forms:

- **Methylcobalamin**: Cofactor for *methionine synthase* (converts homocysteine → methionine; also regenerates tetrahydrofolate for DNA synthesis → deficiency causes megaloblastic anaemia).
- **Adenosylcobalamin**: Cofactor for *methylmalonyl-CoA mutase* (converts methylmalonyl-CoA → succinyl-CoA; deficiency → methylmalonic acidaemia → demyelination → *subacute combined degeneration of the spinal cord*).

B_{12} absorption requires *intrinsic factor* (from gastric parietal cells); deficiency causes *pernicious anaemia*.

Solution

Q16. (B) Water and electrolyte absorption

The **large intestine (colon)** receives approximately 1.5 L of fluid daily from the ileocaecal valve and reduces this to ≈100–200 mL of faeces — absorbing approximately 1.3–1.4 L of water. The primary colonic functions are:

- **Water absorption**: Osmotic reabsorption following active Na^+ absorption (via ENaC channels on colonocytes, aldosterone-regulated) and Cl^-/HCO_3^- exchange.
- **Electrolyte absorption**: Na^+, Cl^-; K^+ is secreted (regulated by aldosterone).
- **Fermentation by gut microbiome**: Anaerobic bacteria ferment dietary fibre → short-chain fatty acids (SCFA: butyrate, propionate, acetate — fuel colonocytes); also produce vitamins K and certain B vitamins.
- **Storage and defecation**: Faeces stored in sigmoid colon/rectum; defecation coordinated by rectoanal reflexes.

Most protein, fat, carbohydrate, and vitamin absorption is completed in the small intestine.

Solution

Q17. (B) Prevention of polyspermy via the zona reaction

After a single sperm penetrates the zona pellucida and fuses with the oocyte plasma membrane, two rapid mechanisms prevent **polyspermy** (fertilisation by more than one sperm, which would cause triploidy and embryo non-viability):

1. **Fast block** (seconds): Membrane depolarisation immediately after sperm-egg fusion makes the zona and membrane temporarily resistant to further sperm fusion.
2. **Zona reaction** (1–5 min): Cortical granules (beneath the egg plasma membrane) undergo *exocytosis* → release of proteases (ovastacin, VERL) and other enzymes into the perivitelline space → harden the zona pellucida and inactivate/cleave ZP2 and ZP3 sperm receptors → permanently prevents additional sperm penetration.

The zona pellucida is retained until the blastocyst stage (≈day 5–6), when it undergoes *hatching* to allow implantation into the endometrium. IVF techniques often require partial zona drilling (assisted hatching) to facilitate implantation.

Solution

Q18. (D) Diastole — ventricular filling

The **cardiac cycle** (duration ≈800 ms at 75 bpm) consists of:

- **Diastole (≈500 ms)**: Ventricular filling phase:
  1. *Isovolumetric relaxation* (≈60 ms): Aortic valve closes, mitral valve still closed; pressure falls but volume unchanged.
  2. *Rapid ventricular filling* (≈100 ms): AV valves open as ventricular pressure drops below atrial; ≈70% of SV enters; produces the third heart sound (S3) if abnormal.
  3. *Diastasis* (slow filling): Pressures nearly equalise; ≈10% of filling.
  4. *Atrial systole (atrial kick)*: ≈20% of final ventricular filling; lost in atrial fibrillation.
- **Systole (≈300 ms)**: Isovolumetric contraction + ejection phase.

In tachycardia, diastole is disproportionately shortened (systole shortens less), reducing filling time and stroke volume — explaining why very fast heart rates reduce cardiac output.

Solution

Q19. (B) L1–L2 (conus medullaris)

The spinal cord terminates as the **conus medullaris** at the level of **L1–L2** in adults (it is relatively higher in neonates, at L3). Below L2, the vertebral canal contains the **cauda equina** ("horse's tail") — a bundle of lumbar, sacral, and coccygeal nerve roots that descend to their respective intervertebral foramina. Clinical implications:

- **Lumbar puncture**: Needle inserted at **L3–L4 or L4–L5** (below the cord); the cauda equina roots are displaced by the needle rather than injured (they float in CSF). The *Tuffier's line* (line between iliac crests) marks the L4 level.
- **Conus medullaris syndrome**: Lesion at L1–L2 causes mixed UMN and LMN signs (bladder/bowel dysfunction, saddle anaesthesia, less severe leg weakness).
- **Cauda equina syndrome**: Lesion below L2 causes pure LMN signs, severe radicular pain, bowel/bladder dysfunction — a surgical emergency.

Solution

Q20. (A) Trophic hormones stimulate other endocrine glands

Trophic (tropic) hormones are defined by their primary action: stimulating a *target endocrine gland* to grow and produce its own hormones. All classic trophic hormones are secreted by the **anterior pituitary** under control of hypothalamic releasing/inhibiting hormones:

- **TSH** (thyroid-stimulating hormone) ← TRH → stimulates thyroid to produce T3/T4.
- **ACTH** (adrenocorticotropic hormone) ← CRH → stimulates adrenal cortex to produce cortisol.
- **FSH** (follicle-stimulating hormone) ← GnRH → stimulates ovarian follicle development / testicular spermatogenesis.
- **LH** (luteinising hormone) ← GnRH → stimulates ovulation and corpus luteum / Leydig cell testosterone production.

Each operates via a *hypothalamo-pituitary-target organ axis* with negative feedback. *Growth hormone* (GH) acts on non-endocrine targets (bone, liver) — it is somatotrophic, not strictly "trophic" in this classification.

Solution

Q21. (D) Approximately 0.1 seconds (100 ms)

The **AV node** (located in the Koch triangle at the interatrial septum) has several unique electrophysiological properties that produce a **deliberate conduction delay of ≈0.1 s (100 ms)**:

- *Slow conduction velocity* (≈0.05 m/s vs. 4 m/s in Purkinje fibres) due to small cell diameter, fewer gap junctions, and Ca^{2+}-dependent (rather than fast Na^{+}-dependent) action potential upstroke.
- This delay **allows atrial systole to complete ventricular filling** (the "atrial kick") before ventricular contraction, optimising cardiac output.
- On the ECG, the **PR interval** (0.12–0.20 s) includes atrial depolarisation (P wave) + AV nodal delay (PR segment). A PR >0.20 s indicates *first-degree AV block*.

The AV node is the *only normal electrical connection* between atria and ventricles (fibrous annulus is otherwise insulating). It also acts as a *backup pacemaker* (40–60 bpm) if the SA node fails (junctional rhythm).

Solution

Q22. (A) CN V, VII, IX, X, XII

Swallowing (deglutition) is a complex reflex coordinated by the *swallowing centre* in the medulla oblongata, involving five cranial nerves:

- **CN V (Trigeminal)**: Motor — muscles of mastication (temporalis, masseter, medial/lateral pterygoids), mylohyoid, anterior belly of digastric, tensor veli palatini; sensory — oral cavity.
- **CN VII (Facial)**: Motor — buccinator (compresses cheeks), orbicularis oris; also controls labial seal.
- **CN IX (Glossopharyngeal)**: Sensory — posterior tongue, pharynx (triggers swallow reflex); Motor — stylopharyngeus muscle (elevates pharynx/larynx).
- **CN X (Vagus)**: Motor — pharyngeal constrictors (via pharyngeal plexus), laryngeal muscles (via RLN — closes glottis to prevent aspiration), oesophagus (peristalsis).
- **CN XII (Hypoglossal)**: Motor — intrinsic and extrinsic tongue muscles (propels food bolus posteriorly).

Dysphagia from cranial nerve palsies or brainstem strokes can cause aspiration pneumonia — a major clinical complication.

Solution

Q23. (C) Charge barrier (negative glycocalyx) repels anionic albumin

The **glomerular filtration barrier** is a three-layer structure:

1. *Fenestrated glomerular capillary endothelium*: Pores ≈70–100 nm diameter prevent passage of blood cells and platelets but allow plasma proteins.
2. *Glomerular basement membrane (GBM)*: Type IV collagen + laminin + heparan sulfate proteoglycans; provides both **size** (>8 nm diameter excluded) and **charge** (negative heparan sulfate glycocalyx) filtration barriers. Albumin (MW ≈69 kDa, diameter ≈7.2 nm, net negative charge at physiological pH) is repelled primarily by the **electrostatic charge barrier**.
3. *Podocyte slit diaphragms*: Nephrin and podocin proteins; final size-selective barrier (≈40 nm slits).

Nephrotic syndrome (proteinuria >3.5 g/day, hypoalbuminaemia, oedema) results from *loss of the charge barrier* (minimal change disease — effacement of foot processes but intact GBM structurally) or *structural GBM damage* (membranous nephropathy, FSGS). Normal urine albumin <30 mg/day.

Solution

Q24. (A) ICF ≈ 28 L (67% of TBW)

Body fluid compartments in a 70 kg adult male (TBW ≈ 42 L = 60% of body weight):

- **Intracellular fluid (ICF) ≈ 28 L (2/3 of TBW)**: The **largest** compartment; high K^+, Mg^{2+}, phosphate, proteins; separated from ECF by cell membranes maintaining composition via Na^+/K^+-ATPase.
- **Extracellular fluid (ECF) ≈ 14 L (1/3 of TBW)**: High Na^+, Cl^-, HCO_3^-:
  - Interstitial fluid ≈ 10.5 L (3/4 of ECF)
  - Plasma ≈ 3.5 L (1/4 of ECF) — distinguished from ISF by plasma proteins (higher oncotic pressure)
  - Transcellular fluid ≈ 1 L (CSF, synovial, pleural, peritoneal, aqueous humour)

Key clinical application: Isotonic saline (0.9% NaCl) distributes only in ECF; D5W (becomes free water) distributes across all compartments (2/3 ICF, 1/3 ECF). This guides IV fluid selection in hyponatraemia, hypovolaemia, and shock.

Solution

Q25. (A) Mast cells and basophils store and release histamine

Histamine is synthesised from the amino acid l-histidine by histidine decarboxylase and stored preformed in secretory granules:

- **Mast cells**: Tissue-resident (especially in skin, respiratory mucosa, gut wall, around blood vessels); express FcεRI receptors for IgE; degranulate in response to IgE cross-linking, complement (C3a, C5a — anaphylatoxins), physical stimuli (cold, trauma), neuropeptides (substance P).
- **Basophils**: Circulating granulocytes (<1% of WBCs); similar IgE-mediated degranulation; also release IL-4 and IL-13 (Th2 cytokines).
- **Gastric ECL (enterochromaffin-like) cells**: Store histamine separately; release it locally in response to gastrin and ACh → stimulates H2 receptors on parietal cells → HCl secretion (blocked by H2 antagonists: ranitidine, famotidine).

Histamine receptors: H1 (vascular permeability, bronchospasm, itch — blocked by antihistamines: cetirizine, loratadine); H2 (gastric acid — blocked by H2 blockers); H3 (presynaptic CNS); H4 (immune cells).