NEET MDS General Physiology and Biochemistry

Sample Paper – 4

Duration: 11 Minutes **Maximum Marks: 56**

Instructions

- This paper contains **14** Multiple Choice Questions (Single Best Response), modelled on the General Physiology and Biochemistry content of **NEET MDS** conducted by NBEMS.
- The **14-question** length matches the number of Physiology and Biochemistry items you actually meet in the real 240-question paper.
- Each correct answer carries **+4 marks**. There is **negative marking of 1 mark** for every incorrect answer; unattempted questions carry no penalty.
- Every question has **exactly one best answer**. Choose the single most appropriate option.
- Attempt this practice paper in one timed sitting of about **11 minutes**, the pace the real computer-based test demands.

Vitamins and Minerals

Q1. A woman planning pregnancy is advised a supplement periconceptionally to reduce the risk of an open neural tube defect in the baby. The same vitamin, when deficient, produces a megaloblastic anaemia with a normal serum B12 level. That vitamin is:

(A) Folic acid

(B) Niacin

(C) Biotin

(D) Pantothenic acid

Q2. Which fat-soluble vitamin is the principal chain-breaking antioxidant of cell membranes, protecting membrane polyunsaturated fatty acids

from lipid peroxidation, so that its deficiency in the newborn causes haemolytic anaemia?

(A) Vitamin A

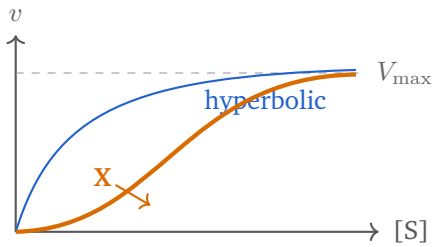
(B) Vitamin E

(C) Vitamin K

(D) Vitamin D

Enzymes and Enzyme Kinetics

Q3. Two enzymes are assayed over a range of substrate concentrations. The curve marked **X** is sigmoid (S-shaped), while the other curve is a simple hyperbola. The enzyme giving curve **X** is also switched off by the end product of its own pathway. Enzyme **X** is best described as:



(A) An allosteric enzyme showing feedback inhibition

(B) An enzyme obeying simple Michaelis-Menten kinetics

(C) A zymogen awaiting proteolytic activation

(D) A ribozyme made of RNA

Q4. Trypsinogen secreted by the pancreas is inactive until enteropeptidase in the duodenum converts it to trypsin, and pepsinogen is likewise converted to pepsin. The molecular event that activates such a proenzyme is:

(A) Reversible phosphorylation of a serine residue by a kinase

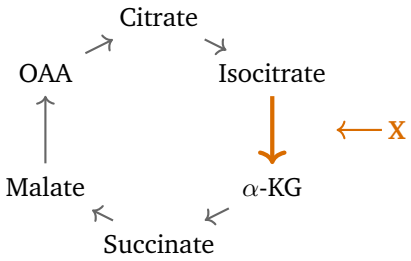
(B) Irreversible cleavage of a specific peptide bond with loss of a fragment

(C) Reversible binding of an allosteric activator to a regulatory site

(D) Increased transcription of the gene coding for the enzyme

Metabolism of Carbohydrate, Lipid and Protein

Q5. In the outline of the TCA cycle below, the step marked **X** is the rate limiting step of the cycle and is allosterically activated by ADP and inhibited by ATP and NADH. The enzyme catalysing **X** is:



(α-KG = alpha-ketoglutarate, OAA = oxaloacetate)

(A) Citrate synthase

(B) Isocitrate dehydrogenase

(C) Succinate dehydrogenase

(D) Malate dehydrogenase

Q6. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) transfer an amino group from an amino acid to alpha-ketoglutarate. Both enzymes are inactive without a coenzyme derived from vitamin B6. That coenzyme is:

(A) Biotin

(B) Tetrahydrofolate

(C) Pyridoxal phosphate

(D) Coenzyme A

Q7. Among the plasma lipoproteins, the particle that picks up excess cholesterol from peripheral tissues and carries it back to the liver for disposal, a process called reverse cholesterol transport, is:

(A) Chylomicron

(B) Very low density lipoprotein (VLDL)

(C) Low density lipoprotein (LDL)

(D) High density lipoprotein (HDL)

Acid Base Balance and Body Fluids

Q8. A patient with pyloric obstruction has been vomiting repeatedly for four days and is losing large volumes of gastric juice. The acid base disturbance expected is:

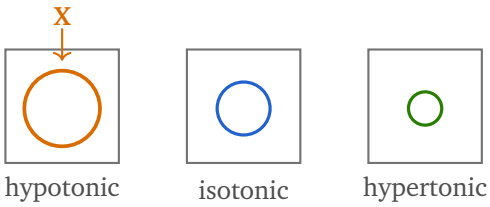
(A) Metabolic alkalosis

(B) Metabolic acidosis

(C) Respiratory alkalosis

(D) Respiratory acidosis

Q9. The figure shows red cells placed in three solutions. The cell marked **X** sits in a solution whose effective osmolarity is well below that of plasma. What happens to cell **X**?



(A) Water enters the cell, which swells and may burst

(B) Water leaves the cell, which shrinks and becomes crenated

(C) There is no net movement of water and the cell keeps its normal volume

(D) Sodium is actively pumped in and the cell loses water

Blood and Haemostasis

Q10. A patient with a chronic dental infection has a markedly raised erythrocyte sedimentation rate (ESR). The change in the blood that most directly speeds up the fall of red cells in the Westergren tube is:

(A) A rise in the packed cell volume, as in polycythaemia

(B) Sickling of red cells, as in sickle cell disease

(C) A rise in plasma fibrinogen and globulins, which promotes rouleaux formation

(D) A fall in plasma fibrinogen, as in afibrinogenaemia

Q11. In a healthy adult, the normal total leucocyte count and the commonest cell in the differential count are:

(A) 4,000 to 11,000 per cubic mm, with lymphocytes the commonest

(B) 40,000 to 110,000 per cubic mm, with neutrophils the commonest

(C) 150,000 to 400,000 per cubic mm, with neutrophils the commonest

(D) 4,000 to 11,000 per cubic mm, with neutrophils the commonest

Nerve, Muscle and Endocrine Physiology

Q12. Two muscle relaxants are used during a general anaesthetic for a maxillofacial procedure. Suxamethonium (succinylcholine) produces visible fasciculations before paralysis, while vecuronium does not. The correct account of how these two drugs act is:

(A) Both block the release of the transmitter from the motor nerve terminal

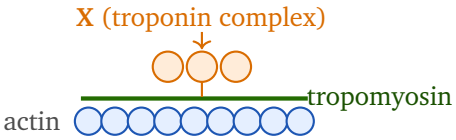
(B) Suxamethonium is a competitive antagonist at the end plate and vecuronium is an agonist

(C) Both are competitive antagonists, and neostigmine reverses each of them

(D) Suxamethonium is an agonist causing persistent depolarisation of the end plate, while vecuronium is a competitive antagonist reversed by neostigmine

Q13. The figure shows a thin filament at rest. When the muscle fibre is excited, calcium released from the sarcoplasmic reticulum binds one component

of the complex marked **X**, which then drags tropomyosin aside and uncovers the binding site on actin. The component that binds the calcium is:



(A) Troponin I

(B) Troponin C

(C) Troponin T

(D) Tropomyosin itself

Q14. Growth hormone drives longitudinal bone growth largely through insulin-like growth factor 1 (IGF-1) made in the liver. A pituitary adenoma secreting excess growth hormone produces:

(A) Acromegaly if it appears before the epiphyses fuse and gigantism if it appears after fusion

(B) Dwarfism at any age, because excess hormone closes the epiphyses early

(C) Gigantism if it appears before the epiphyses fuse and acromegaly if it appears after fusion

(D) Cretinism in the child and myxoedema in the adult

Detailed Solutions

Q1.

Solution

Concept – folate is needed wherever cells divide fast: Folate carries one-carbon units, and the tissues that suffer first are the ones dividing most quickly.

Step 1 – name the active form: Dietary folic acid is reduced to **tetrahydrofolate (THF)**, the working coenzyme.

Step 2 – state its key job: THF donates a one-carbon unit for the conversion of deoxyuridylate (dUMP) to thymidylate (dTMP), the step that supplies thymine for DNA.

Step 3 – explain the anaemia: Without thymidylate, DNA synthesis stalls while cytoplasmic RNA and protein synthesis carry on. The marrow precursors grow big but cannot divide, giving **megaloblastic anaemia** with macro-ovalocytes and hypersegmented neutrophils.

Step 4 – separate it from vitamin B12: Vitamin B12 deficiency gives an identical blood picture, so the question deliberately tells you the serum B12 is normal. That leaves folate as the deficient vitamin.

Step 5 – explain the neural tube link: The neural tube closes by about day 28 of gestation, before most women know they are pregnant. Folate supports the rapid cell division of neural tube closure, so a periconceptional supplement, started before conception, cuts the risk of spina bifida and anencephaly.

Step 6 – note the oral relevance: Folate deficiency also shows in the mouth as glossitis, angular cheilitis and recurrent aphthous ulcers, which is why the dental surgeon often spots it first.

Why the other options are wrong:

- B, niacin: deficiency causes pellagra, that is dermatitis, diarrhoea and dementia, with a beefy red tongue. It does not cause megaloblastic anaemia or neural tube defects.
- C, biotin: is the carboxylation coenzyme. Its deficiency is rare and causes dermatitis and alopecia, not megaloblastosis.
- D, pantothenic acid: forms coenzyme A. Its deficiency is almost never seen clinically and presents as burning feet, not anaemia.

Final Answer: Folic acid $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – an antioxidant that must live inside the membrane: Lipid peroxidation happens in the fatty core of the membrane, so the vitamin that stops it has to be fat-soluble and sit there.

Step 1 – identify the vitamin: Vitamin E (alpha-tocopherol) is the main lipid-soluble antioxidant of cell membranes.

Step 2 – describe the chemistry: Free radicals attack the double bonds of membrane polyunsaturated fatty acids and start a chain reaction of peroxidation.

Step 3 – explain how vitamin E stops it: Vitamin E donates a hydrogen atom to the lipid peroxyl radical, becoming a stable, poorly reactive tocopheryl radical. The chain therefore breaks, which is why it is called a chain-breaking antioxidant.

Step 4 – note how it is regenerated: Vitamin C in the aqueous phase reduces the tocopheryl radical back to vitamin E, so the two vitamins work as a team across the membrane.

Step 5 – link to the clinical clue: The red cell membrane is rich in polyunsaturated fatty acids and is under constant oxygen stress. Without vitamin E its membrane peroxidises and the cell lyses, giving the **haemolytic anaemia of the premature newborn**, the exact picture in the stem.

Why the other options are wrong:

- A, vitamin A: is fat-soluble and its carotenoid precursors do quench some radicals, but its defined role is in vision as retinal and in epithelial differentiation as retinoic acid. It is not the principal membrane antioxidant.
- C, vitamin K: is fat-soluble but works as the cofactor for gamma-carboxylation of clotting factors. It has no antioxidant role and its deficiency causes bleeding, not haemolysis.
- D, vitamin D: is a steroid hormone precursor acting through a nuclear receptor to control calcium handling. Its deficiency causes rickets and osteomalacia.

Final Answer: Vitamin E $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – the shape of the curve tells you the kind of enzyme: A hyperbola means one substrate site acting alone; an S-shaped curve means several sites talking to each other.

Step 1 – read curve X off the graph: Curve X starts almost flat, then rises steeply, then plateaus. That is a **sigmoid** curve.

Step 2 – ask what makes a curve sigmoid: Sigmoid kinetics arise when the enzyme has **several subunits** and the binding of substrate to one subunit makes the next subunit bind more easily. This is positive cooperativity.

Step 3 – explain the flat foot and the steep rise: At low substrate the first molecule binds a low-affinity, tense form, so velocity barely moves. Once it binds, the whole molecule flips to a high-affinity relaxed form and the remaining sites fill quickly, which is the steep part.

Step 4 – use the second clue: The stem says the end product of the pathway switches enzyme X off. An end product does not resemble the substrate, so it cannot be sitting in the active site. It must bind a separate **allosteric** site, and this is **feedback inhibition**.

Step 5 – put the two clues together: Sigmoid kinetics plus feedback inhibition by the end product is the definition of an **allosteric enzyme**. Aspartate transcarbamoylase and phosphofructokinase-1 are the standard examples, and haemoglobin shows the same sigmoid binding for oxygen.

Why the other options are wrong:

- B, Michaelis-Menten enzyme: gives the **hyperbolic** curve, that is the other curve on the graph. Such an enzyme has no cooperativity, and K_m is a valid constant for it, whereas an allosteric enzyme is described by $K_{0.5}$ instead.
- C, zymogen: is an inactive precursor. It would show no activity at all until cleaved, so it could not generate either curve.
- D, ribozyme: is a catalytic RNA such as the peptidyl transferase of the ribosome. Nothing in the graph or the stem points to RNA, and ribozymes are not the classic example of sigmoid feedback control.

Final Answer: An allosteric enzyme showing feedback inhibition $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – dangerous enzymes are made switched off: A protease made in an active form would digest the cell that made it, so it is secreted as an inactive proenzyme and switched on only where it is needed.

Step 1 – name the class: An inactive precursor of an enzyme is a **zymogen** or proenzyme, and it is named with the suffix -ogen or the prefix pro-.

Step 2 – follow trypsinogen: The pancreas secretes trypsinogen. In the duodenum, **enteropeptidase (enterokinase)** cuts a single peptide bond and removes a short hexapeptide from the N-terminal end.

Step 3 – explain why cutting activates it: Removing that fragment lets the rest of the chain refold, which brings the catalytic serine, histidine and aspartate into line and completes the substrate binding pocket. The active site is created by the cut.

Step 4 – follow pepsinogen: Pepsinogen is secreted by gastric chief cells. Gastric acid, and then pepsin itself, cleave off the blocking peptide to give **pepsin**. Both examples in the stem therefore work the same way.

Step 5 – state the key property: Proteolytic cleavage is **irreversible**. A cut peptide bond cannot be put back, so the cell cannot switch the enzyme off again; it must instead inhibit it, for example with pancreatic trypsin inhibitor.

Step 6 – note the clinical point: If trypsinogen is activated inside the pancreas rather than the gut, it activates the other zymogens as well and the gland digests itself. That is acute pancreatitis.

Why the other options are wrong:

- A, phosphorylation: is a genuine control mechanism, used for glycogen phosphorylase and glycogen synthase, but it is **reversible** and a phosphatase can undo it. It is not how a zymogen is activated.
- C, allosteric activator binding: is also reversible and non-covalent. It tunes the rate of an already active enzyme rather than converting a dead precursor into a live one.
- D, increased transcription: makes **more** enzyme molecules, and it takes hours. The zymogen protein is already present in the gut lumen and is activated within seconds, so transcription cannot be the answer.

Final Answer: Irreversible cleavage of a specific peptide bond $\Rightarrow$ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – the TCA cycle is paced by the cell's energy charge: The cycle has three regulated enzymes, and the one that is rate limiting responds directly to how much ATP the cell already has.

Step 1 – read the marked step off the ring: Step X converts **isocitrate to alpha-ketoglutarate**.

Step 2 – name the enzyme: That conversion is catalysed by **isocitrate dehydrogenase**, which oxidises and decarboxylates isocitrate, releasing the first carbon dioxide of the cycle and producing the first NADH.

Step 3 – match the regulation given in the stem: Isocitrate dehydrogenase is allosterically **activated by ADP** and **inhibited by ATP and NADH**, exactly as the question states.

Step 4 – explain why that makes it the pacemaker: A high ADP level means the cell is short of energy, so the cycle is switched on. A high ATP and NADH level means the cell is already rich in energy, so the cycle slows. That is precisely the behaviour a rate limiting enzyme must show.

Step 5 – list the other regulated points for contrast: Citrate synthase and the alpha-ketoglutarate dehydrogenase complex are also regulated, but isocitrate dehydrogenase is the accepted **rate limiting** enzyme of the cycle.

Why the other options are wrong:

- A, citrate synthase: catalyses the entry step, condensing acetyl-CoA with oxaloacetate to give citrate. It is regulated, but the diagram marks the step after citrate, not the step that makes citrate.
- C, succinate dehydrogenase: converts succinate to fumarate, is the only cycle enzyme bound to the inner mitochondrial membrane, and is complex II of the electron transport chain. It produces $FADH_2$ and is not a control point.
- D, malate dehydrogenase: converts malate to oxaloacetate in the last step. That reaction is freely reversible and is pulled forward by removal of oxaloacetate, so it is not rate limiting.

Final Answer: Isocitrate dehydrogenase $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – transamination moves an amino group, it does not destroy it: Before nitrogen can be excreted it has to be collected onto one carrier molecule, and transamination is how that collection happens.

Step 1 – write the general reaction: Amino acid + alpha-ketoglutarate → keto acid + glutamate.

Step 2 – write the two named examples: ALT: alanine + alpha-ketoglutarate → pyruvate + glutamate.

AST: aspartate + alpha-ketoglutarate → oxaloacetate + glutamate.

Step 3 – identify the coenzyme: Both aminotransferases require **pyridoxal phosphate (PLP)**, the active coenzyme derived from vitamin B6.

Step 4 – explain what PLP does: The aldehyde group of PLP forms a Schiff base with the amino group of the amino acid. The nitrogen is handed to PLP, making pyridoxamine phosphate, and is then handed on to alpha-ketoglutarate. PLP is the shuttle that carries the amino group across.

Step 5 – note the fate of the nitrogen: Glutamate carries the collected nitrogen to the liver mitochondria, where glutamate dehydrogenase releases free ammonia for the urea cycle.

Step 6 – note the clinical use: Because ALT is largely hepatic and AST is also in heart and muscle, their serum levels are the standard liver function tests, and a raised AST to ALT ratio suggests alcoholic liver disease.

Why the other options are wrong:

- A, biotin: is the coenzyme for **carboxylation** reactions such as pyruvate carboxylase and acetyl-CoA carboxylase. It carries carbon dioxide, not amino groups.
- B, tetrahydrofolate: carries **one-carbon** units for purine and thymidylate synthesis. It is not involved in transamination.
- D, coenzyme A: carries **acyl** groups, as in acetyl-CoA and succinyl-CoA, and is derived from pantothenic acid rather than vitamin B6.

Final Answer: Pyridoxal phosphate ⇒ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – each lipoprotein has one job and one direction: Sort the four particles by where they are made and which way they move lipid, and the answer follows.

Step 1 – chylomicron: Made in the intestine, it carries **dietary** triglyceride from gut to peripheral tissues.

Step 2 – VLDL: Made in the liver, it carries **endogenous** triglyceride from liver out to the tissues.

Step 3 – LDL: Formed as VLDL is stripped of its triglyceride, it delivers **cholesterol from the liver out to the tissues**. It is the atherogenic particle, the so-called bad cholesterol.

Step 4 – HDL: Made in the liver and intestine as a discoid particle, it picks up free cholesterol from peripheral cells and takes it **back to the liver**. That direction, tissue to liver, is **reverse cholesterol transport**, which is what the question asks for.

Step 5 – name the machinery: Cholesterol leaves the cell through the ABCA1 transporter, is esterified inside HDL by **LCAT** (activated by apo A-I), and the cholesteryl ester is delivered to the liver via the SR-B1 receptor.

Step 6 – state the consequence: Because HDL removes cholesterol from vessel walls, a high HDL is protective against atherosclerosis, which is why it is called the good cholesterol.

Why the other options are wrong:

- A, chylomicron: is the largest and least dense particle and is triglyceride-rich. It moves lipid **from the gut outwards**, the opposite direction, and its marker is apo B-48.
- B, VLDL: also carries triglyceride **away** from the liver, not cholesterol back to it.
- C, LDL: carries cholesterol **to** the tissues through the apo B-100 receptor. Its excess deposits in the arterial wall rather than clearing it.

Final Answer: High density lipoprotein $\Rightarrow$ D

Answer: (D) Go Back to Q7

Q8.

Solution

Concept – name the fluid that is being lost: The acid base result of vomiting depends entirely on what the vomitus contains, and gastric juice is acid.

Step 1 – state what gastric juice holds: Gastric juice is rich in **hydrochloric acid**, secreted by the parietal cells, along with sodium, potassium and chloride.

Step 2 – follow the hydrogen ion: Each hydrogen ion that the parietal cell pumps into the lumen leaves one **bicarbonate** ion behind in the blood. This is the alkaline tide.

Step 3 – see what vomiting does to that balance: Normally the acid reaches the duodenum and is neutralised, so nothing is gained. In pyloric obstruction the acid is vomited out instead, and the matching bicarbonate stays in the plasma unopposed.

Step 4 – name the disturbance: Loss of hydrogen ions with retention of bicarbonate raises the pH. This is a **metabolic alkalosis**, since the primary change is a rise in bicarbonate rather than a change in carbon dioxide.

Step 5 – add the volume effect: Vomiting also loses chloride, sodium and water. Volume depletion raises aldosterone, which makes the kidney retain sodium in exchange for further loss of hydrogen and potassium. The alkalosis is therefore maintained, giving the classic **hypokalaemic hypochloraemic metabolic alkalosis** of pyloric obstruction.

Step 6 – name the compensation: The lungs hypoventilate to retain carbon dioxide and pull the pH back down, so the $PaCO_2$ rises. The rise in carbon dioxide is compensatory and does not change the primary diagnosis.

Why the other options are wrong:

- B, metabolic acidosis: would follow the loss of **alkaline** intestinal fluid, as in severe diarrhoea or a high output ileostomy, where bicarbonate is lost. Vomiting loses acid, so the pH moves the other way.
- C, respiratory alkalosis: needs hyperventilation blowing off carbon dioxide. There is nothing in the stem to make this patient hyperventilate, and the primary change here is in bicarbonate.
- D, respiratory acidosis: needs hypoventilation with carbon dioxide retention. The rise in carbon dioxide here is a compensatory response, not the primary problem, and the pH is alkaline rather than acid.

Final Answer: Metabolic alkalosis $\Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – water follows the effective osmolarity: Osmolarity counts every solute; tonicity counts only the solutes that cannot cross the membrane and can therefore hold water on their own side.

Step 1 – fix the reference value: Plasma osmolarity is about **285 to 295 mOsm/L**, and the red cell interior sits at the same value, which is why a cell in plasma keeps its shape.

Step 2 – read the condition given: The solution around cell X has an effective osmolarity well **below** that of plasma, so it is **hypotonic**.

Step 3 – decide the direction of water movement: Water always moves from the compartment with less solute to the one with more. Outside is dilute and inside is concentrated, so water moves **into** the cell.

Step 4 – state the effect on the cell: The cell takes up water and swells. The red cell first rounds up into a sphere, and if water entry continues the membrane gives way and the cell **haemolyses**, releasing haemoglobin.

Step 5 – check the other two cells for consistency: The isotonic cell is unchanged, since there is no net gradient. The hypertonic cell loses water, shrinks and crenates. The figure shows exactly this size gradient, largest to smallest.

Step 6 – note the clinical relevance: This is why intravenous fluid must be isotonic, and why plain water can never be infused into a vein.

Why the other options are wrong:

- B, water leaves and the cell crenates: is what happens in a **hypertonic** solution such as 3 percent saline. It is the third cell in the figure, not cell X.
- C, no net movement: describes an **isotonic** solution such as 0.9 percent saline, which is the middle cell.
- D, sodium pumped in: is wrong on two counts. The sodium-potassium ATPase pumps sodium **out** of the cell, not in, and osmotic swelling is passive water movement that needs no pump at all.

Final Answer: Water enters and the cell swells and may burst $\Rightarrow$ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – ESR measures how fast red cells clump, not how sick the patient is: The rate of fall depends on the size of the falling unit, and single red cells fall slowly because they repel one another.

Step 1 – state why cells normally stay apart: Red cells carry a negative surface charge from sialic acid, the zeta potential. Like charges repel, so the cells stay separate and sink slowly.

Step 2 – state what an infection does to plasma: Chronic infection raises the acute phase proteins, above all **fibrinogen**, and also the immunoglobulins.

Step 3 – link that to the cells: These large asymmetrical proteins are positively charged relative to the cell surface. They neutralise the zeta potential, so the repulsion is lost.

Step 4 – explain rouleaux: The cells now stack together like a pile of coins. This is **rouleaux formation**.

Step 5 – apply the physics: A rouleau is a much bigger unit than a single cell, and its mass rises faster than its surface drag. The stack therefore sinks quickly and the ESR is high.

Step 6 – keep the perspective: ESR is a non-specific marker. It is raised in infection, tuberculosis, malignancy, rheumatoid arthritis and pregnancy, so it supports a diagnosis rather than making one.

Why the other options are wrong:

- A, polycythaemia: **lowers** the ESR. Packing more cells into the column increases the friction between them, so they fall more slowly. Anaemia is what raises the ESR.
- B, sickling: also **lowers** the ESR. Sickle cells are rigid and misshapen and cannot stack into rouleaux at all.
- D, a fall in fibrinogen: works the opposite way. With less fibrinogen the zeta potential is preserved, rouleaux do not form and the ESR is low. This is also why the ESR is low in disseminated intravascular coagulation, where fibrinogen is consumed.

Final Answer: A rise in plasma fibrinogen and globulins $\Rightarrow$ C

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – know the three counts and do not mix them up: Red cells, white cells and platelets each have their own order of magnitude, and the distractors are built out of those numbers.

Step 1 – state the normal total leucocyte count: The normal TLC in an adult is **4,000 to 11,000 per cubic mm**.

Step 2 – write out the differential count: Neutrophils 40 to 75 percent, lymphocytes 20 to 45 percent, monocytes 2 to 10 percent, eosinophils 1 to 6 percent, basophils 0 to 1 percent.

Step 3 – pick the commonest: The largest share belongs to the **neutrophil**, so it is the commonest white cell in adult blood.

Step 4 – combine the two facts: 4,000 to 11,000 per cubic mm with neutrophils commonest is option D.

Step 5 – note the age exception: Between about two years and puberty the differential is reversed and lymphocytes predominate. The question specifies an adult, so neutrophils stand.

Step 6 – note the clinical use: A count above 11,000 is leucocytosis, and an acute dental abscess raises it with a neutrophil predominance and a shift to the left. A count below 4,000 is leucopenia, which matters before any surgical procedure.

Why the other options are wrong:

- A: has the right count but the wrong cell. Lymphocytes are the commonest in a child, not in an adult.
- B: has the right cell but a count ten times too high. A TLC of 40,000 to 110,000 is a leukaemoid reaction or leukaemia, not a normal value.
- C: quotes 150,000 to 400,000 per cubic mm, which is the normal **platelet** count, not the leucocyte count.

Final Answer: 4,000 to 11,000 per cubic mm with neutrophils commonest $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – two ways to paralyse the same receptor: A neuromuscular blocker can either sit on the nicotinic receptor and keep it switched on, or sit on it and keep the transmitter out. The two mechanisms behave differently at the bedside.

Step 1 – take suxamethonium first: Suxamethonium is two acetylcholine molecules joined together. It is an **agonist** at the nicotinic receptor of the motor end plate.

Step 2 – explain the fasciculations: Because it is an agonist, it opens the channels and depolarises the end plate, and that initial burst of disorganised muscle activity is seen as **fasciculations**.

Step 3 – explain why depolarising causes paralysis: Unlike acetylcholine, suxamethonium is not broken down by acetylcholinesterase in the cleft. It stays bound, so the end plate remains depolarised. A permanently depolarised membrane keeps the neighbouring voltage-gated sodium channels inactivated, so no further muscle action potential can be launched and the fibre goes flaccid.

Step 4 – take vecuronium: Vecuronium is a non-depolarising blocker. It is a **competitive antagonist** that occupies the nicotinic receptor without opening it, so acetylcholine cannot act. There is no depolarisation, and therefore no fasciculation.

Step 5 – reverse each one: Neostigmine inhibits acetylcholinesterase and raises the acetylcholine level in the cleft. That extra acetylcholine outcompetes vecuronium and **reverses** it. The same drug given with suxamethonium raises acetylcholine at an already depolarised end plate and makes the block **worse**, so it must never be used for that purpose.

Step 6 – match to the options: Suxamethonium as a depolarising agonist and vecuronium as a competitive antagonist reversed by neostigmine is option D.

Why the other options are wrong:

- A, both block transmitter release: describes the mechanism of botulinum toxin and of aminoglycosides, which act on the **presynaptic** terminal. Both drugs in the question act on the postsynaptic receptor.
- B: has the two drugs exactly swapped. Vecuronium is the antagonist and suxamethonium is the agonist, and a swapped account cannot explain the fasciculations described in the stem.
- C: says both are competitive and both reverse with neostigmine. If suxamethonium were competitive it would not depolarise, so it could not fasciculate, and giving neostigmine with it would deepen rather than lift the block.

Final Answer: Suxamethonium depolarises as an agonist, vecuronium is a competitive antagonist $\Rightarrow$ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – the thin filament is a switch, and calcium throws it: At rest the actin site is physically covered. Contraction begins only when something moves the cover, and that something is calcium.

Step 1 – read the resting state from the figure: Tropomyosin lies along the actin filament and blocks the myosin binding site. The troponin complex, marked X, is anchored to it.

Step 2 – break the complex into its three subunits: Troponin **T** binds tropomyosin and holds the complex in place. Troponin **I** inhibits, that is it holds tropomyosin over the actin site. Troponin **C** is the calcium binder.

Step 3 – release the calcium: The muscle action potential runs down the T tubule, the dihydropyridine receptor senses the voltage and opens the ryanodine receptor of the sarcoplasmic reticulum, and stored calcium floods into the cytosol.

Step 4 – bind the calcium: Calcium binds **troponin C**, which is the answer the question asks for. The C stands conveniently for calcium.

Step 5 – follow the conformational change: Calcium binding to troponin C changes the shape of the whole complex. Troponin I loses its grip, troponin T drags tropomyosin deeper into the actin groove, and the myosin binding site on actin is **uncovered**.

Step 6 – close the loop: Myosin heads can now attach, and cross-bridge cycling follows. When the calcium is pumped back into the sarcoplasmic reticulum by SERCA, it leaves troponin C, tropomyosin slides back over the site and the muscle relaxes. Relaxation is therefore an active, ATP-consuming step, which is why rigor mortis sets in when ATP runs out.

Why the other options are wrong:

- A, troponin I: is part of the complex but it is the **inhibitory** subunit. It holds tropomyosin over the actin site and it does not bind calcium.
- C, troponin T: is the subunit that **attaches** the complex to tropomyosin. It transmits the change but has no calcium binding site of its own. Note that cardiac troponins T and I, not C, are the enzymes measured after a myocardial infarction.

- D, tropomyosin: is the rope that covers the site. It is moved by the troponin complex, and it has no calcium binding site.

Final Answer: Troponin C $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – the epiphysis decides the shape of the disease: Growth hormone excess produces two different pictures, and the only thing that separates them is whether the growth plates are still open.

Step 1 – state how growth hormone works: Growth hormone is released from the anterior pituitary somatotrophs. It acts on the liver to produce **IGF-1 (somatomedin C)**, and IGF-1 does most of the growth-promoting work on cartilage and bone.

Step 2 – take the child: Before puberty the epiphyseal growth plates are still open cartilage. Excess growth hormone drives chondrocyte proliferation there, so the long bones keep lengthening and the child becomes abnormally tall. That is **gigantism**.

Step 3 – take the adult: After puberty the sex steroids fuse the epiphyses to the shafts. There is no growth plate left, so the bone cannot get longer.

Step 4 – say what happens instead: Growth now goes into appositional and periosteal thickening and into soft tissue. The hands and feet broaden, the jaw grows forward, the brow ridges thicken and the nose and lips enlarge. That is **acromegaly**.

Step 5 – fix the order: Before fusion gives gigantism; after fusion gives acromegaly. That order is option C.

Step 6 – note the dental relevance: Acromegaly matters directly to the dental surgeon. Mandibular prognathism, spacing of the teeth, an anterior open bite and macroglossia are common presenting features, and the dentist may be the first to notice that dentures no longer fit.

Why the other options are wrong:

- A: has the two conditions reversed. The long bones can only lengthen while the plates are open, so the tall child cannot be the acromegalic one.
- B, dwarfism: is caused by growth hormone **deficiency**, or by receptor insensitivity with a failure to make IGF-1 as in Laron dwarfism. Excess hormone

does not close the epiphyses early.

- D, cretinism and myxoedema: are the childhood and adult pictures of **hypothyroidism**, not of growth hormone excess. They belong to a different hormone axis altogether.

Final Answer: Gigantism before fusion and acromegaly after fusion $\Rightarrow$ C

Answer: (C) Go Back to Q14

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	A	2	B	3	A	4	B	5	B
6	C	7	D	8	A	9	A	10	C
11	D	12	D	13	B	14	C		