NEET MDS General Physiology and Biochemistry

Sample Paper – 6

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 24-year-old presents with painful fissuring at both angles of the mouth, a smooth magenta tongue and scaling around the nasolabial folds. The deficient vitamin is the one whose coenzyme forms are FMN and FAD. That vitamin is:

(A) Riboflavin (vitamin B2)

(B) Pyridoxine (vitamin B6)

(C) Cyanocobalamin (vitamin B12)

(D) Ascorbic acid (vitamin C)

Q2. A young adult taking a high dose retinoid preparation for months develops a set of symptoms attributed to hypervitaminosis A. A recognised feature of chronic vitamin A toxicity is:

(A) Night blindness with Bitot spots on the conjunctiva

(B) Raised intracranial pressure with headache, blurred vision and papilloedema

(C) Megaloblastic anaemia with hypersegmented neutrophils

(D) A prolonged prothrombin time from failed gamma-carboxylation of clotting factors

Enzymes and Enzyme Kinetics

Q3. Enzymes are highly specific for their substrates. The *induced fit* model, which replaced the older lock and key model, states that:

(A) The active site is a rigid pre-formed cavity into which only a complementary substrate slots, like a key into a lock

(B) Substrate binding makes the active site change shape, so the site moulds itself around the substrate and lines up the catalytic groups

(C) The substrate must first be chemically altered before it is able to bind the active site

(D) The enzyme accepts any molecule of matching molecular weight, irrespective of its shape or charge

Q4. Salivary amylase (ptyalin) starts carbohydrate digestion in the mouth. Its substrate, its optimum pH and its fate once the bolus reaches the stomach are, respectively:

(A) Cellulose; pH 2.0; it works better in gastric acid

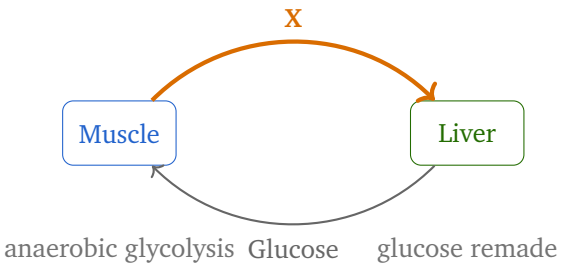
(B) Starch; pH 2.0; it is switched on by gastric acid

(C) Starch; pH 6.8; it hydrolyses the alpha-1,6 branch points and is unaffected by gastric acid

(D) Starch; pH 6.8; it is inactivated once gastric acid drops the pH below about 4

Metabolism of Carbohydrate, Lipid and Protein

Q5. The diagram below outlines the Cori cycle running between exercising skeletal muscle and the liver. The substance carried in the blood from muscle to liver at the step marked **X** is:



(A) Pyruvate

(B) Glycerol

(C) Lactate

(D) Acetyl-CoA

Q6. Which statement correctly describes the digestion and absorption of dietary protein?

(A) Pepsin starts digestion in the acid stomach, pancreatic trypsin and chymotrypsin continue it in the alkaline duodenum, and the products cross the intestinal mucosa mainly as free amino acids and small peptides

(B) Trypsin starts digestion in the acid stomach and pepsin finishes it in the duodenum, and the products are absorbed as free amino acids

(C) Digestion is entirely gastric, and dietary protein is absorbed intact into the portal blood as whole polypeptides

(D) No luminal digestion occurs, and whole protein molecules pass into the lymphatics inside chylomicrons

Q7. Basal metabolic rate is measured at complete physical and mental rest, in a comfortable room, at least 12 hours after the last meal. Which of the following *raises* basal metabolic rate?

(A) Prolonged starvation

(B) Hypothyroidism

(C) Fever, which lifts basal metabolic rate by roughly 10 to 13 percent for every 1 degree Celsius rise in body temperature

(D) Deep sleep

Acid Base Balance and Body Fluids

Q8. A patient in septic shock with severe pneumonia and tiring respiratory muscles has these arterial values: pH 7.08, $PaCO_2$ 55 mmHg, bicarbonate 15 mEq/L. The correct interpretation is:

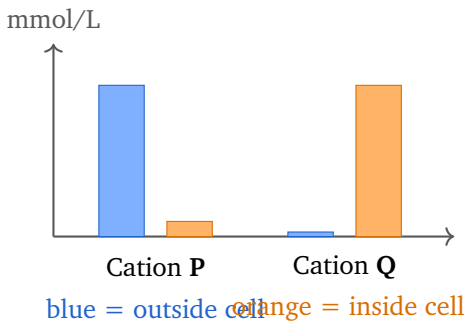
(A) A mixed disorder, because a bicarbonate of 15 mEq/L should have driven the $PaCO_2$ down to about 30 mmHg, and instead it is high at 55 mmHg

(B) A simple metabolic acidosis with fully appropriate respiratory compensation

(C) A simple respiratory acidosis with appropriate renal compensation

(D) A metabolic acidosis with respiratory overcompensation, since compensation routinely pushes the pH past 7.40

Q9. The chart below compares the concentrations of two cations, **P** and **Q**, outside and inside a typical cell. **P** is the principal cation of the extracellular fluid and **Q** the principal cation of the intracellular fluid. **P** and **Q** are, respectively:



(A) Potassium and sodium

(B) Sodium and calcium

(C) Calcium and potassium

(D) Sodium and potassium

Blood and Haemostasis

Q10. The clotting factors carry Roman numerals as well as names. Which factor and name pairing is correct?

(A) Factor I is prothrombin

(B) Factor III is tissue factor

(C) Factor II is fibrinogen

(D) Factor IV is vitamin K

Q11. Blood drawn for a full blood count is collected into a tube containing EDTA, and blood for a coagulation screen into a tube containing sodium citrate. Both additives keep the sample fluid by:

(A) Enhancing the action of antithrombin on thrombin and factor Xa

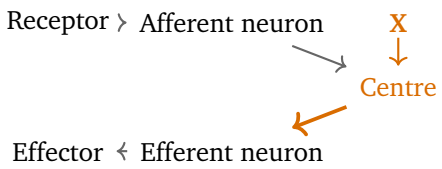
(B) Chelating calcium ions, so the calcium-dependent steps of the cascade cannot proceed

(C) Blocking vitamin K epoxide reductase in the hepatocyte

(D) Converting plasminogen to plasmin and dissolving fibrin as it forms

Nerve, Muscle and Endocrine Physiology

Q12. The diagram shows the anatomical pathway of a reflex, with the integrating centre in the spinal cord marked **X**. The correct order of the five components of a reflex arc, from stimulus to response, is:



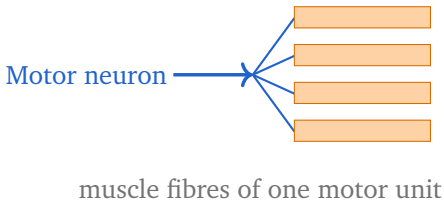
(A) Receptor, afferent neuron, centre, efferent neuron, effector

(B) Receptor, efferent neuron, centre, afferent neuron, effector

(C) Effector, afferent neuron, centre, efferent neuron, receptor

(D) Afferent neuron, receptor, centre, effector, efferent neuron

Q13. The figure shows one motor unit: a single alpha motor neuron together with every muscle fibre it supplies. When a muscle is asked to produce steadily greater force, the nervous system chiefly:



(A) Increases the diameter of each individual muscle fibre from moment to moment

(B) Increases the amplitude of the action potential travelling along each fibre

(C) Switches off the small motor units and fires only the largest one

(D) Recruits progressively more motor units, small ones first and larger ones as the demand rises

Q14. Which statement about calcitonin is correct?

(A) It is secreted by the chief cells of the parathyroid gland and raises plasma calcium

(B) It is secreted by the follicular cells of the thyroid and raises plasma calcium

(C) It is secreted by the parafollicular C cells of the thyroid and lowers plasma calcium

(D) It is secreted by the parafollicular C cells of the thyroid and raises plasma calcium by stimulating osteoclasts

Detailed Solutions

Q1.

Solution

Concept – riboflavin deficiency is read off the mouth: Vitamin B2 deficiency, or ariboflavinosis, announces itself with oral and perioral signs that a dentist sees first.

Step 1 – use the biochemical clue: The question says the vitamin's coenzymes are **FMN** (flavin mononucleotide) and **FAD** (flavin adenine dinucleotide). Those two are the coenzyme forms of **riboflavin**, so the vitamin is named by that clue alone.

Step 2 – state what those coenzymes do: FMN and FAD are the prosthetic groups of the flavoproteins, which carry hydrogen in oxidation reduction reactions such as succinate dehydrogenase and the respiratory chain.

Step 3 – explain why the mouth suffers first: Oral mucosa and lip epithelium turn over very fast, so tissues with a high replication rate fail first when the cell's energy transfer flavoproteins run short.

Step 4 – match each clinical sign: Fissuring at the angles of the mouth is **angular cheilitis (angular stomatitis)**. The smooth magenta tongue is **glossitis** from atrophy of the filiform papillae. Scaling around the nasolabial folds is the seborrhoeic dermatitis of riboflavin deficiency. Corneal vascularisation completes the classic picture.

Step 5 – confirm: Oral signs plus FMN and FAD gives riboflavin, vitamin B2.

Why the other options are wrong:

- B, pyridoxine: its coenzyme is pyridoxal phosphate, not FMN or FAD. It can also give cheilosis, but the coenzyme clue excludes it outright.
- C, cyanocobalamin: works as methylcobalamin and adenosylcobalamin. Its deficiency gives megaloblastic anaemia and subacute combined degeneration, not an FMN or FAD defect.
- D, ascorbic acid: is not a flavin coenzyme at all. It acts as a reducing agent in hydroxylation reactions, and its deficiency gives bleeding gums rather than angular cheilitis with a magenta tongue.

Final Answer: Riboflavin (vitamin B2) $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – vitamin A is fat soluble, so it accumulates: Fat soluble vitamins are stored rather than excreted in urine, so a sustained high intake builds up to toxic levels. Vitamin A is the classic example.

Step 1 – note where it is stored: Retinol is stored in the stellate (Ito) cells of the liver. Excess intake loads the liver, which is why toxicity gives hepatomegaly and eventually fibrosis.

Step 2 – recall the hallmark neurological effect: Chronic hypervitaminosis A raises cerebrospinal fluid pressure, producing **pseudotumour cerebri (benign intracranial hypertension)**: headache, nausea, blurred vision and papilloedema on fundoscopy.

Step 3 – add the rest of the picture: Dry peeling skin, cracked lips, alopecia, bone and joint pain, cortical hyperostosis, and in pregnancy a serious risk of teratogenicity.

Step 4 – check the option against that list: Raised intracranial pressure with headache, blurred vision and papilloedema is exactly the recognised feature, so option B is correct.

Step 5 – keep toxicity and deficiency apart: Toxicity is not simply more of the deficiency picture. They are different syndromes, and the exam tests whether you can separate them.

Why the other options are wrong:

- A, night blindness with Bitot spots: is vitamin A **deficiency**, the exact opposite state. Toxicity does not cause it.
- C, megaloblastic anaemia with hypersegmented neutrophils: is folate or vitamin B12 deficiency, and has nothing to do with retinol excess.
- D, prolonged prothrombin time from failed gamma-carboxylation: is vitamin K deficiency. Vitamin A plays no part in carboxylating clotting factors.

Final Answer: Raised intracranial pressure with papilloedema $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – two models of how a substrate meets an active site: Enzyme specificity means one enzyme handles one substrate or one class of substrates. The two models differ over whether the active site is rigid or flexible.

Step 1 – state the older lock and key model: Emil Fischer proposed that the active site is a **rigid** cavity of fixed shape, and only a substrate exactly complementary to it can enter, like a key into a lock.

Step 2 – state the problem with it: A rigid site explains specificity, but it cannot explain why enzymes bind transition states more tightly than substrates, nor why some enzymes accept several related substrates, nor how regulatory conformational changes occur.

Step 3 – state the induced fit model: Koshland proposed that the active site is **flexible**. When the substrate approaches, it induces a conformational change so the site closes and moulds around the substrate.

Step 4 – state what that achieves: Only after this change do the catalytic groups line up correctly with the bonds to be broken. Catalysis therefore begins only when the right substrate binds, which sharpens specificity rather than loosening it.

Step 5 – give a worked example: Hexokinase is the standard illustration. Its two lobes swing shut around glucose, excluding water. Without induced fit the enzyme would waste ATP by phosphorylating water instead.

Step 6 – pick the option: Option B describes substrate binding causing a conformational change that moulds the site around the substrate, which is induced fit.

Why the other options are wrong:

- A: is the **lock and key** model, the older one the question asks you to move beyond.
- C: is invented. The substrate is altered **by** catalysis after binding, not before it, and no model requires prior alteration.
- D: contradicts specificity itself. Enzymes select on shape, charge and hydrogen bonding, never on molecular weight.

Final Answer: Substrate binding moulds the active site around it $\Rightarrow$ B

Answer: (B) Go Back to Q3

Q4.

Solution

Concept – every enzyme has an optimum pH, and amylase's is near neutral: Salivary amylase is secreted into a mouth held near neutral pH, and it stops the moment its environment turns acid.

Step 1 – name the substrate: Salivary amylase is an **alpha-amylase**. It hydrolyses **starch** (and glycogen), splitting the internal alpha-1,4 glycosidic bonds to give maltose, maltotriose and alpha-limit dextrins.

Step 2 – state the optimum pH: Its optimum is about **pH 6.8**, close to the neutral pH of resting saliva, which is roughly 6.5 to 7.4.

Step 3 – see what the stomach does to that: Gastric juice has a pH of about 1.5 to 3.5. As the bolus mixes with acid, the pH of the food mass falls.

Step 4 – state the consequence for the enzyme: Once the pH falls below about **4**, salivary amylase is denatured and irreversibly inactivated. Acid protonates the ionisable groups of the active site, the tertiary structure unfolds, and catalysis stops.

Step 5 – note the timing detail: Amylase keeps working for a short while inside the bolus, because the centre of a swallowed bolus is not acidified immediately. Up to about 30 to 40 percent of starch may be broken down before the acid finally penetrates.

Step 6 – state what takes over: Pancreatic amylase then completes starch digestion in the duodenum, where bicarbonate has raised the pH back to about 7 to 8, suiting an alpha-amylase again.

Step 7 – pick the option: Starch, pH 6.8, inactivated below pH 4. That is option D.

Why the other options are wrong:

- A, cellulose at pH 2.0: humans secrete no cellulase at all, and pH 2.0 destroys amylase rather than suiting it.
- B, starch at pH 2.0 switched on by acid: the pH is wrong and the direction is wrong. Acid inactivates amylase, it does not activate it.
- C, alpha-1,6 bonds and unaffected by acid: two errors. Alpha-amylase cannot cut the alpha-1,6 branch points, which is precisely why alpha-limit dextrins remain and need isomaltase later, and amylase is very much affected by gastric acid.

Final Answer: Starch; pH 6.8; inactivated below pH 4 $\Rightarrow$ D

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – the Cori cycle moves a metabolic burden from muscle to liver: Hard-working muscle runs short of oxygen. The Cori cycle lets it keep making ATP by exporting the problem to the liver.

Step 1 – see what happens in the muscle: During vigorous exercise the oxygen supply cannot keep pace, so muscle runs **anaerobic glycolysis**. Glucose becomes pyruvate, and the cell nets 2 ATP per glucose.

Step 2 – see why muscle cannot stop at pyruvate: Glycolysis needs a steady supply of NAD^+ at the glyceraldehyde-3-phosphate dehydrogenase step. Without oxygen the mitochondria cannot regenerate NAD^+, so glycolysis would grind to a halt within seconds.

Step 3 – see how muscle solves it: Lactate dehydrogenase reduces pyruvate to **lactate**, and in doing so it oxidises NADH back to NAD^+. Glycolysis can therefore continue.

Step 4 – identify what is exported: Lactate cannot be used further in the anaerobic muscle, so it diffuses into the blood. **Lactate** is therefore the substance travelling from muscle to liver at step X.

Step 5 – follow it into the liver: In the hepatocyte, lactate dehydrogenase runs the reaction the other way, back to pyruvate. The liver then rebuilds glucose from that pyruvate.

Step 6 – close the loop: Glucose is released into the blood and returns to the muscle, which is the lower arrow in the diagram. The cycle is complete: lactate out, glucose back.

Step 7 – note the cost: Muscle gains 2 ATP per glucose, while the liver spends 6 ATP to rebuild it. The cycle is not free. It buys the muscle time and shifts the energy cost to a well-oxygenated organ.

Why the other options are wrong:

- A, pyruvate: is the precursor **inside** the muscle, but it is reduced to lactate before export precisely so that NAD^+ is regenerated. Exporting pyruvate would leave NADH stranded and glycolysis stalled.
- B, glycerol: reaches the liver from adipose tissue when triglyceride is broken down. It is not a product of muscle glycolysis and is not part of the Cori cycle.
- D, acetyl-CoA: cannot leave the cell, because there is no plasma membrane transporter for it, and in any case it is not formed when oxygen is short.

Final Answer: Lactate $\Rightarrow$ C

Answer: (C) Go Back to Q5

Q6.

Solution

Concept – protein digestion happens in two places with two different pH values: Follow the protein down the gut and name the enzyme working at each station.

Step 1 – the mouth: There is no protein digestion in the mouth. Saliva contains no proteolytic enzyme of any consequence.

Step 2 – the stomach: Gastric chief cells secrete pepsin, which works at the acid pH of 1.5 to 2.5 supplied by the parietal cells. Pepsin is an endopeptidase that cuts internal bonds beside aromatic amino acids, breaking protein into large peptides.

Step 3 – the duodenum: Pancreatic juice brings **trypsin**, chymotrypsin and elastase, all endopeptidases, plus carboxypeptidases A and B, which are exopeptidases that trim amino acids off the carboxyl end. Bicarbonate raises the pH to about 8, which suits them.

Step 4 – the brush border: Aminopeptidases and dipeptidases on the enterocyte microvilli finish the job, leaving free amino acids, dipeptides and tripeptides.

Step 5 – absorption: Free amino acids enter the enterocyte by sodium-dependent co-transporters, while di- and tripeptides enter through the PepT1 transporter and are hydrolysed to amino acids inside the cell.

Step 6 – the destination: Amino acids leave the enterocyte and enter the **portal vein**, going to the liver. They do not enter the lymph. That route belongs to fats.

Step 7 – pick the option: Pepsin in the acid stomach, trypsin and chymotrypsin in the alkaline duodenum, absorption mainly as amino acids and small peptides. That is option A.

Why the other options are wrong:

- B: swaps the two enzymes. Trypsin is pancreatic and would be destroyed by gastric acid; pepsin is gastric and is inactivated by the alkaline duodenum.
- C: says digestion is entirely gastric and protein is absorbed whole. Removing the stomach does not stop protein digestion, which proves pancreatic enzymes do the bulk of the work, and intact protein is not absorbed in adults.
- D: puts protein into chylomicrons. Chylomicrons carry **fat** into the lymph. Amino acids are water soluble and go to the portal blood.

Final Answer: Pepsin, then trypsin and chymotrypsin, absorbed as amino acids $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – basal metabolic rate is the resting cost of staying alive: BMR is the energy an awake but completely resting body spends, roughly 1600 to 1800 kcal per day in an adult male, or about 35 to 40 kcal per square metre per hour.

Step 1 – list what raises it: Fever, hyperthyroidism, exercise and the period after it, cold exposure, pregnancy, adrenaline and noradrenaline, growth in children, and the specific dynamic action of a protein meal.

Step 2 – list what lowers it: Hypothyroidism, prolonged starvation, sleep, ageing and loss of lean body mass.

Step 3 – take the fever figure: Every 1 degree Celsius rise in body temperature lifts BMR by about **10 to 13 percent**. This is van't Hoff's effect: heat speeds every chemical reaction in the cell, and the body is a bag of chemical reactions.

Step 4 – do the arithmetic on it: Take a baseline BMR of 1600 kcal per day.

A 2 degree Celsius fever raises it by $2 \times 12 = 24$ percent.

$1600 \times 0.24 = 384$ kcal.

$1600 + 384 = 1984$ kcal per day.

That is why a febrile patient wastes and needs extra calories.

Step 5 – pick the option: Fever is the only listed factor that raises BMR, so option C is correct.

Why the other options are wrong:

- A, prolonged starvation: **lowers** BMR by up to 20 to 30 percent. The body downregulates thyroid conversion and burns less to protect its remaining fuel stores.
- B, hypothyroidism: **lowers** BMR, sometimes by 30 to 40 percent, because thyroid hormone is the main long-term setter of metabolic rate. Hyperthyroidism would have been the correct answer.
- D, deep sleep: **lowers** energy expenditure by about 10 to 15 percent below the basal figure, mainly through reduced muscle tone. BMR is measured awake for exactly this reason.

Final Answer: Fever $\Rightarrow$ C

Answer: (C) Go Back to Q7

Q8.

Solution

Concept – compensation follows rules, and breaking a rule reveals a second disorder: In a simple disorder, the compensating system moves in a **predictable** direction by a **predictable** amount. If it has not, there is a second primary disorder sitting on top.

Step 1 – read the pH: pH 7.08 is well below 7.35, so this is a severe **acidaemia**.

Step 2 – read the bicarbonate: Bicarbonate is 15 mEq/L against a normal of 22 to 26. A low bicarbonate is acidifying, so there is a **metabolic acidosis**, which fits the lactic acidosis of septic shock.

Step 3 – read the carbon dioxide: $PaCO_2$ is 55 mmHg against a normal of 35 to 45. A high $PaCO_2$ is also acidifying, so there is a **respiratory acidosis** as well.

Step 4 – state the rule that settles it: Both values are pushing the pH the **same way**. In a simple disorder with compensation they must push **opposite** ways. Two acidifying values therefore mean two acidoses, not one with compensation.

Step 5 – prove it with Winter's formula: Expected $PaCO_2 = (1.5 \times HCO_3^-) + 8 \pm 2$

$= (1.5 \times 15) + 8$

$= 22.5 + 8$

$= 30.5$ mmHg, so about 28 to 33 mmHg.

Step 6 – compare with the measured value: Measured $PaCO_2$ is 55 mmHg, far above the expected 30.5.

$55 - 30.5 = 24.5$ mmHg higher than compensation predicts.

The patient's tiring respiratory muscles cannot deliver the hyperventilation the kidneys are demanding, so a respiratory acidosis is superimposed. This is a **mixed metabolic and respiratory acidosis**.

Step 7 – state the two limits of compensation: First, compensation is never complete. It pulls the pH back **towards** 7.40 but never all the way there, so an abnormal pH always still points to the primary disorder. Second, compensation never **overshoots**. It cannot drive the pH past normal to the opposite side, because the stimulus for compensating is the abnormal pH itself, and that stimulus disappears as the pH normalises. A pH on the wrong side of 7.40 for the supposed primary disorder always means a second disorder.

Why the other options are wrong:

- B, appropriate respiratory compensation: would need a $PaCO_2$ near 30 mmHg. At 55 mmHg the lungs are making the acidosis worse, not compensating for it.
- C, respiratory acidosis with renal compensation: renal compensation **raises** bicarbonate to buffer a high CO_2. Here the bicarbonate is low at 15, the opposite of compensation, so a metabolic acidosis must also be present.
- D, respiratory overcompensation: is impossible. Compensation never drives the pH past normal, which is exactly the limit this question tests. In any case the pH is 7.08, deeply acidic, so nothing has been overcorrected.

Final Answer: A mixed disorder, $PaCO_2$ far above the expected 30 mmHg $\Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – the two cations sit on opposite sides of the membrane: Water moves freely between compartments, but ions do not. That is what gives extracellular and intracellular fluid completely different ionic profiles despite identical osmolarity.

Step 1 – read the chart for cation P: P is very high outside the cell and very low inside. That is the profile of **sodium**: about 140 mmol/L in plasma against roughly 10 to 15 mmol/L inside the cell.

Step 2 – read the chart for cation Q: Q is very low outside and very high inside. That is the profile of **potassium**: about 4 to 5 mmol/L in plasma against roughly 140 to 150 mmol/L inside the cell.

Step 3 – name the pump that maintains the difference: The **sodium potassium ATPase** in every plasma membrane pushes 3 sodium out and pulls 2 potassium in for each ATP hydrolysed. It runs continuously and consumes a substantial share of the body's resting energy.

Step 4 – complete the anion picture: Chloride and bicarbonate are the main extracellular anions. Phosphates, sulphates and negatively charged proteins are the main intracellular anions. Magnesium is the second intracellular cation, calcium the second extracellular one.

Step 5 – note why osmolarity still matches: Both compartments sit at about 290 mOsm/L. The ionic **species** differ completely, but the total particle count does not, so no net water shift occurs.

Step 6 – state the clinical consequence: Because potassium is almost entirely intracellular, plasma potassium is a poor guide to total body potassium, and small

plasma shifts have large effects on the resting membrane potential of cardiac muscle.

Step 7 – pick the option: P is sodium, Q is potassium, in that order. That is option D.

Why the other options are wrong:

- A, potassium and sodium: has the right pair but in the **reversed** order. Sodium is extracellular, not intracellular.
- B, sodium and calcium: sodium is right, but calcium is not the main intracellular cation. Free cytosolic calcium is around 0.0001 mmol/L, one hundred thousand times lower than the value the chart shows for Q, because calcium is a signalling ion and must be kept almost absent from the cytosol.
- C, calcium and potassium: potassium is right, but plasma calcium is only about 2.5 mmol/L, far too low to be the principal extracellular cation.

Final Answer: Sodium and potassium $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – learn the first four factors by heart: The Roman numerals were assigned in the order the factors were discovered, not the order they act. The first four are the ones examiners return to.

Step 1 – factor I: Factor I is **fibrinogen**, the soluble plasma protein that thrombin converts into the fibrin mesh.

Step 2 – factor II: Factor II is **prothrombin**, made in the liver, vitamin K dependent, and converted to thrombin by factor Xa.

Step 3 – factor III: Factor III is **tissue factor**, also called thromboplastin. It is not in plasma. It sits in the subendothelial tissue and is exposed only when a vessel is torn, whereupon it starts the extrinsic limb with factor VII.

Step 4 – factor IV: Factor IV is **calcium**. It is the only factor that is not a protein, and it is needed to anchor the vitamin K dependent factors to phospholipid surfaces.

Step 5 – check each option against that list: Option B says factor III is tissue factor. That is correct, so B is the answer.

Step 6 – note two more that are worth knowing: There is no factor VI; the number was retired when it turned out to be activated factor V. Factor XIII is fibrin

stabilising factor, which cross-links the fibrin strands into a firm clot.

Why the other options are wrong:

- A, factor I is prothrombin: wrong. Factor I is fibrinogen. Prothrombin is factor II.
- C, factor II is fibrinogen: wrong, and it is simply the mirror image of the same error. Factor II is prothrombin.
- D, factor IV is vitamin K: wrong. Factor IV is calcium. Vitamin K is a vitamin, not a clotting factor, and it carries no Roman numeral of its own.

Final Answer: Factor III is tissue factor $\Rightarrow$ B

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – take away the calcium and the cascade cannot run: Calcium is factor IV. Several steps of coagulation are absolutely calcium dependent, so removing calcium from a tube stops clotting immediately.

Step 1 – state where calcium is needed: The gamma-carboxyglutamate residues on factors II, VII, IX and X bind calcium ions, and those calcium ions bridge the factors onto the negatively charged phospholipid surface of activated platelets. Without that bridge the tenase and prothrombinase complexes cannot assemble.

Step 2 – state how EDTA works: EDTA (ethylenediaminetetraacetic acid) is a **chelating agent**. It grips calcium ions in a tight, essentially irreversible complex, driving ionised calcium to near zero.

Step 3 – state how citrate works: Sodium citrate binds calcium too, but more **loosely** and reversibly.

Step 4 – explain why the two are used for different tests: EDTA removes calcium so thoroughly that clotting can never be restarted, which is fine for a full blood count where you only want intact cells. Citrate's binding is reversible, so adding calcium back to the plasma restarts the cascade on demand. That is exactly what a PT or aPTT assay does, which is why coagulation tubes use citrate and not EDTA.

Step 5 – note the in vivo point: Neither works inside the body at a useful dose, because stripping the plasma calcium enough to stop clotting would cause fatal tetany first. Both are **in vitro** anticoagulants only.

Step 6 – contrast heparin: Heparin is the anticoagulant that works both in vitro

and in vivo, and its mechanism is completely different. It binds **antithrombin** and increases its activity roughly a thousandfold, so antithrombin then inactivates thrombin and factor Xa. Heparin does not touch calcium at all.

Step 7 – pick the option: EDTA and citrate both act by chelating calcium, so option B is correct.

Why the other options are wrong:

- A, enhancing antithrombin: is the mechanism of **heparin**, not of EDTA or citrate. This is the commonest trap in the question.
- C, blocking vitamin K epoxide reductase: is an **in vivo** hepatic mechanism that stops the liver making new functional factors over days. It cannot work in a collection tube, which contains no hepatocytes.
- D, converting plasminogen to plasmin: describes a **thrombolytic** such as streptokinase or alteplase. Those dissolve a clot that has already formed; they do not prevent clotting.

Final Answer: Chelating calcium ions $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – a reflex is a fixed, involuntary response over a fixed anatomical path: The reflex arc is that path, and it always has five components in one order.

Step 1 – the receptor: The stimulus is detected first. In the knee jerk this is the **muscle spindle** in quadriceps, which senses the stretch produced by the tendon tap.

Step 2 – the afferent neuron: The signal travels centrally along the **afferent (sensory) neuron**, a fast Ia fibre whose cell body sits in the dorsal root ganglion.

Step 3 – the centre: The fibre enters the spinal cord and reaches the **centre**, marked X in the diagram, where the decision is made. In the knee jerk the Ia fibre synapses directly on the alpha motor neuron in the ventral horn.

Step 4 – define monosynaptic from that: Because there is exactly **one** synapse at the centre and no interneuron in between, the stretch reflex is called **monosynaptic**. It is the only monosynaptic reflex in the body, and its single synapse is why its latency is so short, around 20 milliseconds. Every other reflex, such as the withdrawal reflex, has one or more interneurons and is polysynaptic.

Step 5 – the efferent neuron: The **efferent (motor) neuron** carries the command

out of the ventral horn to the periphery.

Step 6 – the effector: The **effector** is the organ that acts. Here it is the quadriceps, which contracts and extends the knee. An effector may equally be smooth muscle or a gland.

Step 7 – assemble the order: Receptor, afferent neuron, centre, efferent neuron, effector. Stimulus in at the receptor, response out at the effector. That is option A.

Why the other options are wrong:

- B: swaps the afferent and efferent neurons. That would carry the sensory signal outwards and the motor command inwards, which is anatomically impossible.
- C: swaps the receptor and the effector, so the stimulus would be detected by the muscle and the response delivered to the spindle. The arc would run backwards.
- D: puts the afferent neuron before the receptor, meaning a signal would travel before anything had been detected. It also separates the effector from the end of the path.

Final Answer: Receptor, afferent, centre, efferent, effector $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – the motor unit is the smallest amount of muscle the brain can switch on: One alpha motor neuron plus every muscle fibre it supplies is one motor unit. Nothing smaller can be activated, because when the neuron fires, every fibre in its unit contracts.

Step 1 – note the all-or-none problem: A muscle fibre obeys the all-or-none law. It either contracts fully or not at all, and a bigger stimulus does not make it contract harder. So force cannot be graded by contracting each fibre a little more.

Step 2 – state the innervation ratio: The number of fibres per neuron varies with the job. Extraocular muscles have about 3 to 10 fibres per neuron, giving very fine control. Gastrocnemius has 1000 to 2000, giving power rather than precision.

Step 3 – state the first mechanism the body uses: Recruitment. To generate more force, the central nervous system simply activates more motor units at once. Since each unit adds its own fixed quantum of tension, the total climbs in steps.

Step 4 – state the order of recruitment: Recruitment follows the **size principle**

of Henneman. Small motor neurons have a higher input resistance, so a given synaptic current depolarises them more and they reach threshold first. Small units, which are slow, fatigue resistant and weak, are therefore recruited first. Large units, which are fast, fatigable and powerful, join only as demand rises.

Step 5 – explain why that order is sensible: It means a light everyday task uses only the fatigue resistant units, so the muscle can hold posture all day. The powerful units are kept in reserve for a heavy effort, and they are the ones that tire quickly.

Step 6 – state the second mechanism: Rate coding. Firing each recruited neuron faster fuses its individual twitches into a sustained tetanic contraction, which produces more tension than single twitches do. Recruitment and rate coding work together, but recruitment is the chief mechanism over most of the force range.

Step 7 – pick the option: Progressive recruitment of more motor units, small ones first and larger ones later. That is option D.

Why the other options are wrong:

- A, increasing fibre diameter: is hypertrophy, which takes weeks of training. It cannot grade force from one second to the next, which is what the question asks.
- B, increasing action potential amplitude: violates the all-or-none law. The amplitude of the muscle action potential is fixed by the ion gradients and does not encode force.
- C, switching off small units and firing only the largest: reverses the size principle and would waste the fatigue resistant units. Large units are **added** to the small ones, never substituted for them.

Final Answer: Recruits progressively more motor units, small first $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – one gland, two hormones, two different cell types: The thyroid gland contains two quite separate endocrine populations, and calcitonin comes from the smaller one.

Step 1 – identify the cell of origin: Calcitonin is a 32 amino acid polypeptide secreted by the **parafollicular cells**, also called **C cells**. They lie between the thyroid follicles rather than lining them, and they are of neural crest origin.

Step 2 – separate them from the follicular cells: The follicular cells are the ones that trap iodine and make thyroxine. They have nothing to do with calcitonin. Two hormones, one gland, two independent cell lines.

Step 3 – state the stimulus for secretion: A **rise** in plasma calcium stimulates the C cells through their calcium sensing receptor. Gastrin and other gut hormones also stimulate release after a meal.

Step 4 – state its action on bone: Calcitonin **inhibits** osteoclasts directly. Osteoclasts carry calcitonin receptors, and within minutes of exposure they retract their ruffled borders and stop resorbing. Bone therefore stops releasing calcium into the blood.

Step 5 – state its action on the kidney: Calcitonin increases urinary excretion of calcium and phosphate by reducing their tubular reabsorption. More is lost in urine.

Step 6 – add the two effects: Less calcium out of bone plus more calcium out in urine gives a **fall** in plasma calcium. So calcitonin lowers plasma calcium, the opposite direction to parathyroid hormone.

Step 7 – note the clinical points: Serum calcitonin is the tumour marker for **medullary carcinoma of the thyroid**, which arises from the C cells. Yet total thyroidectomy does not cause hypocalcaemia from calcitonin loss, which tells you calcitonin is not essential for day-to-day calcium control in adults.

Step 8 – pick the option: Parafollicular C cells of the thyroid, lowers plasma calcium. That is option C.

Why the other options are wrong:

- A, chief cells of the parathyroid raising calcium: both halves are wrong for calcitonin. That description belongs to parathyroid hormone.
- B, follicular cells raising calcium: wrong cell and wrong direction. Follicular cells make thyroxine and triiodothyronine, not calcitonin.
- D, C cells raising calcium via osteoclasts: the cell of origin is right but the action is exactly backwards. Calcitonin **inhibits** osteoclasts, which lowers plasma calcium. This option is the trap for anyone who remembers the cell but not the direction.

Final Answer: Parafollicular C cells of the thyroid; lowers plasma calcium $\Rightarrow$ $\boxed{C}$

Answer: (C) Go Back to Q14

Answer Key

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