# NEET MDS General Physiology and Biochemistry

## Sample Paper – 10

**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 42-year-old strict vegan has a macrocytic anaemia with hypersegmented neutrophils on the smear. He also reports tingling of both feet and has lost vibration and joint position sense in the lower limbs. Both of the classic megaloblastic deficiencies would explain the smear, but only one of them explains the cord signs. The deficient nutrient is:

(A) Folic acid

(B) Vitamin B12 (cobalamin)

(C) Iron

(D) Vitamin B6 (pyridoxine)

**Q2.** An adult on a mixed Indian diet is advised a daily calcium intake of about 600 mg. Absorption of that calcium from the small intestine is not fixed,

and the diet itself can block a large part of it. Which dietary component **hinders** calcium absorption?

(A) Calcitriol, the active form of vitamin D

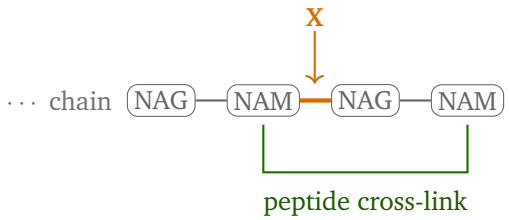
(B) Lactose

(C) Phytates and oxalates

(D) An acidic pH in the duodenum

## Enzymes and Enzyme Kinetics

**Q3.** Saliva and tears both contain lysozyme, an enzyme that lyses many bacteria on contact. The figure shows a length of the bacterial cell wall backbone. Lysozyme kills the organism by hydrolysing the bond marked **X**. Bond **X** is:




(A) The peptide cross-bridge joining adjacent chains

(B) A disulphide bond

(C) The ester bond anchoring teichoic acid

(D) The beta-1,4 glycosidic bond between N-acetylmuramic acid and N-acetylglucosamine

**Q4.** Carbonic anhydrase in the red cell and alkaline phosphatase in the osteoblast are both metalloenzymes, and both hold the same divalent metal ion at the active site. Losing that metal destroys the catalytic activity of each. The metal is:

(A) Iron

(B) Zinc

(C) Magnesium

(D) Copper

## Metabolism of Carbohydrate, Lipid and Protein

**Q5.** Whole saliva is about 99 percent water, but its inorganic fraction is what protects the tooth. Two of its ions are present at concentrations that keep saliva supersaturated with respect to enamel hydroxyapatite, which is how an early white spot lesion can remineralise. Those two ions are:

(A) Sodium and chloride

(B) Calcium and phosphate

(C) Potassium and thiocyanate

(D) Iron and iodide

**Q6.** Amino acids are split into essential and non-essential on the basis of whether the human body can build the carbon skeleton itself. Which of the following is an **essential** amino acid, that is one that must be supplied ready-made in the diet?

(A) Alanine

(B) Glutamate

(C) Lysine

(D) Serine

**Q7.** A human cell contains three major classes of RNA, each with its own job. Measured as a share of the total RNA in the cell, the most abundant class is:

(A) Ribosomal RNA (rRNA)

(B) Messenger RNA (mRNA)

(C) Transfer RNA (tRNA)

(D) Small nuclear RNA (snRNA)

## Acid Base Balance and Body Fluids

**Q8.** A 19-year-old with type 1 diabetes stopped his insulin four days ago. He is now drowsy, dehydrated, breathing deeply and rapidly, and his breath smells sweet. Capillary glucose is 480 mg/dL and urine ketones are strongly positive. The single diagnosis, together with the acid base label and the name of that breathing pattern, is:

(A) Hyperosmolar hyperglycaemic state, a metabolic alkalosis with Cheyne-Stokes breathing

(B) Diabetic ketoacidosis, a high anion gap metabolic acidosis with Kussmaul breathing

(C) Diabetic ketoacidosis, a normal anion gap metabolic acidosis with Biot breathing

(D) Hypoglycaemic coma, a respiratory acidosis with apneustic breathing

**Q9.** A labourer works a full shift in the sun without drinking. His plasma osmolality climbs from 288 to 298 mOsm/kg and he becomes intensely thirsty. The receptors that detect that rise and generate the sensation of thirst are situated in the:

(A) Hypothalamus

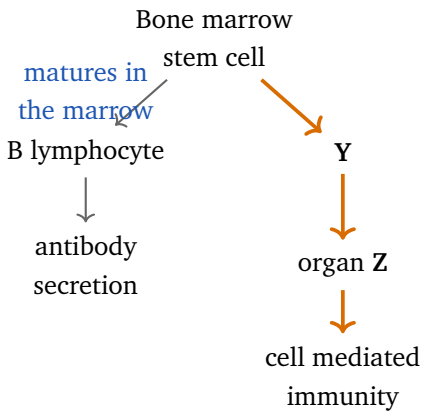
(B) Carotid body

(C) Macula densa of the kidney

(D) Wall of the right atrium

## Blood and Haemostasis

**Q10.** Both lymphocyte lines in the figure begin as one stem cell in the bone marrow. The line marked **Y** leaves the marrow while still immature, finishes its maturation in the organ marked **Z**, and then goes on to mediate cell mediated immunity instead of secreting antibody. Cell **Y** and organ **Z** are:




(A) B lymphocyte and bone marrow

(B) B lymphocyte and thymus

(C) T lymphocyte and the gut associated lymphoid tissue

(D) T lymphocyte and thymus

**Q11.** Albumin is made in the liver and accounts for roughly 60 percent of the total protein in plasma, far more than any other plasma protein. Because it is small and present in such numbers, its main physiological contribution is:

(A) Generating the colloid osmotic (oncotic) pressure of plasma

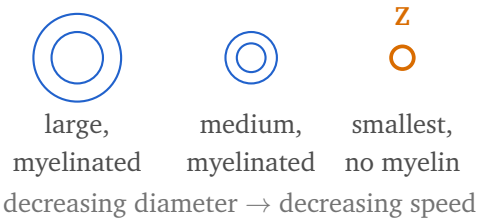
(B) Carrying oxygen from lung to tissue

(C) Forming the fibrin mesh of a clot

(D) Acting as the circulating antibody of humoral immunity

## Nerve, Muscle and Endocrine Physiology

**Q12.** Nerve fibres are classified as A, B and C by diameter, myelination and conduction velocity. The three fibres below are drawn to relative diameter, with an outer ring where myelin is present. Fibre **Z** has the smallest diameter, carries no myelin, and therefore conducts most slowly of the three. Fibre **Z** belongs to which class?



(A) C fibres

(B) A-alpha fibres

(C) B fibres

(D) A-delta fibres

**Q13.** A 35-year-old woman complains that her eyelids droop and her jaw tires while chewing a meal. The weakness is mild on waking, worsens through the day and through repeated effort, and recovers after rest. Repetitive nerve stimulation shows a decremental response. The autoantibodies in this disease are directed against:

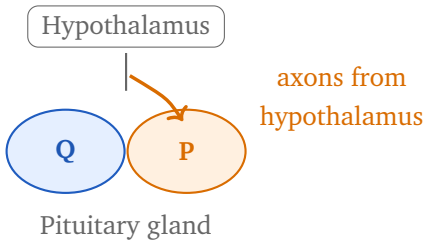
(A) Voltage-gated calcium channels on the presynaptic nerve terminal

(B) Acetylcholinesterase in the synaptic cleft

(C) Myelin of the peripheral nerve

(D) The nicotinic acetylcholine receptor on the postsynaptic membrane

**Q14.** The figure shows the pituitary hanging below the hypothalamus. Lobe **P** manufactures no hormone of its own: it merely stores and then releases peptides that were synthesised in hypothalamic neuronal cell bodies and carried down their axons. The hormones released from lobe **P** are:




(A) Growth hormone and prolactin

(B) Adrenocorticotropic hormone and thyroid stimulating hormone

(C) Oxytocin and antidiuretic hormone

(D) Follicle stimulating hormone and luteinising hormone

## Detailed Solutions

**Q1.**

### Solution

**Concept – separating vitamin B12 from folate deficiency:** The two deficiencies produce an identical blood picture, so the separation is made on the nervous system, not on the smear.

**Step 1 – explain why the smear is identical:** Both cobalamin and folate are needed to make thymidylate for DNA synthesis. When either runs short, the nucleus of the erythroid precursor cannot keep pace with the cytoplasm.

**Step 2 – name the resulting picture:** The cell grows large with an immature nucleus, giving **megaloblastic (macrocytic) anaemia** with hypersegmented neutrophils. Folate deficiency and B12 deficiency both give this.

**Step 3 – find the reaction that is unique to B12:** Cobalamin has a second role that folate does not share. As adenosylcobalamin it is the coenzyme for **methylmalonyl-CoA mutase**, which converts methylmalonyl-CoA to succinyl-CoA.

**Step 4 – follow what happens when that reaction fails:** Methylmalonyl-CoA accumulates. The excess is diverted into abnormal branched-chain fatty acids, which are laid into myelin in place of normal fatty acids, so the myelin is defective.

**Step 5 – match that to the patient:** Defective myelin in the posterior and lateral columns of the cord is **subacute combined degeneration**, which presents exactly as here: tingling of the feet with loss of vibration and joint position sense.

**Step 6 – explain the diet clue:** Vitamin B12 comes only from animal sources. A strict vegan of several years has exhausted his hepatic store, so the deficiency fits both the history and the neurology.

**Why the other options are wrong:**

- A, folic acid: gives the same megaloblastic smear, but folate has no role in myelin. Folate deficiency has **no** neurological signs, so it cannot explain the cord findings. This is the whole point of the question.
- C, iron: gives a **microcytic** hypochromic anaemia, the opposite red cell size, and no hypersegmentation.
- D, vitamin B6: deficiency gives a sideroblastic anaemia with peripheral neuropathy, not a megaloblastic smear with posterior column loss.

**Final Answer:** Vitamin B12 $\Rightarrow$ $\boxed{\text{B}}$

**Answer: (B)** Go Back to Q1

## Q2.

**Solution**

**Concept – swallowed calcium is not absorbed calcium:** Only about 30 to 40 percent of dietary calcium is normally absorbed, and the diet itself decides how much of that is available.

**Step 1 – state where absorption happens:** Calcium is absorbed mainly in the duodenum and upper jejunum, by an active carrier-mediated route and by passive paracellular diffusion.

**Step 2 – list what helps:** Calcitriol, an acid pH, lactose, and dietary protein all raise absorption, because calcium must stay in the soluble ionised form to cross the mucosa.

**Step 3 – identify what blocks it: Phytates** (from cereals, wheat bran and pulses) and **oxalates** (from spinach and similar leafy vegetables) bind calcium in the gut lumen.

**Step 4 – explain the mechanism of the block:** Calcium phytate and calcium oxalate are insoluble salts. Once the calcium is locked into an insoluble complex it can no longer be presented to the transporter, so it passes out in the stool.

**Step 5 – apply it to the 600 mg figure:** A cereal-and-pulse Indian diet is phytate-rich, which is why the intake target has to be generous even though the physiological requirement is smaller.

**Why the other options are wrong:**

- A, calcitriol: is the single most powerful **promoter**. It induces calbindin and the TRPV6 channel in the enterocyte, raising absorption. It is the opposite of what is asked.
- B, lactose: **helps** absorption by forming a soluble complex with calcium, which is one reason milk is a good calcium source.
- D, acidic pH: **helps**, because calcium salts stay ionised and soluble in acid. This is why achlorhydria and long-term proton pump inhibitors reduce calcium absorption.

**Final Answer:** Phytates and oxalates $\Rightarrow$ C

**Answer: (C)** Go Back to Q2

## Q3.

### Solution

**Concept – lysozyme is an antibacterial enzyme, not an antibody:** It is a hydrolase, and it kills by cutting one specific chemical bond in the bacterial cell wall.

**Step 1 – recall the structure of the wall:** Peptidoglycan is a long backbone of alternating sugars, **N-acetylglucosamine (NAG)** and **N-acetylmuramic acid (NAM)**, with short peptide chains hanging off the NAM units and cross-linking neighbouring backbones.

**Step 2 – read the diagram:** The chain drawn is NAG-NAM-NAG-NAM. Bond X sits on the backbone itself, between a NAM and the next NAG, and not on the green peptide cross-link.

**Step 3 – name the bond:** The backbone sugars are joined by a **beta-1,4 glycosidic bond**. Lysozyme is a muramidase and hydrolyses precisely that bond.

**Step 4 – explain how cutting it kills:** Once the backbone is cut the wall loses its tensile strength. The bacterium cannot resist its own high internal osmotic pressure, so water rushes in and the cell bursts. This is osmotic lysis.

**Step 5 – note which organisms suffer most:** Gram positive organisms have a thick, exposed peptidoglycan layer and are lysed readily. Gram negatives are partly shielded by an outer membrane.

**Step 6 – add the dental relevance:** Lysozyme is a constituent of saliva, tears, nasal secretion and neutrophil granules, so it forms part of the innate, non-specific defence of the oral cavity.

**Why the other options are wrong:**

- A, the peptide cross-bridge: is the target of **penicillin**, which blocks transpeptidase, not of lysozyme. The diagram deliberately draws this bond separately in green.
- B, disulphide bond: joins cysteine residues within proteins. Lysozyme itself contains disulphide bonds, but it does not cleave them, and peptidoglycan does not depend on them.
- C, the ester bond of teichoic acid: teichoic acid is anchored to the Gram positive wall, but it is not the backbone and it is not the lysozyme substrate.

**Final Answer:** The beta-1,4 glycosidic bond between NAM and NAG $\Rightarrow$ $\boxed{\text{D}}$

**Answer: (D)** Go Back to Q3

## Q4.

### Solution

**Concept – metalloenzymes hold their metal as part of the protein:** In a true metalloenzyme the metal is tightly bound at the active site and is essential for catalysis, so removing it abolishes activity.

**Step 1 – take the first enzyme named:** Carbonic anhydrase carries one **zinc** ion at its active site, coordinated by three histidine residues.

**Step 2 – take the second enzyme named:** Alkaline phosphatase is also a **zinc** metalloenzyme, and it additionally uses magnesium as an activator.

**Step 3 – find the common metal:** The metal shared by both, and the one whose removal kills the activity of each, is **zinc**.

**Step 4 – explain what the zinc does chemically:** Zinc is a strong Lewis acid. In carbonic anhydrase it polarises a bound water molecule and lowers its pKa, generating a hydroxide ion at neutral pH that attacks carbon dioxide. That is why the metal is indispensable rather than decorative.

**Step 5 – extend the list for the exam:** Other zinc enzymes worth remembering are carboxypeptidase, alcohol dehydrogenase, superoxide dismutase (Cu-Zn) and the matrix metalloproteinases of collagen turnover.

**Why the other options are wrong:**

- A, iron: is the metal of catalase, peroxidase, the cytochromes and haemoglobin. Neither enzyme in the stem uses it.
- C, magnesium: is required by kinases, hexokinase and enolase, and it does act as an activator of alkaline phosphatase, but it is not the metal held at the active site of carbonic anhydrase, so it is not the shared one.
- D, copper: is the metal of cytochrome c oxidase, tyrosinase, lysyl oxidase and ceruloplasmin.

**Final Answer:** Zinc $\Rightarrow$ B

**Answer: (B)** Go Back to Q4

## Q5.

### Solution

**Concept – what saliva is made of:** Saliva is roughly 99 percent water and 1 percent solids, and those solids divide into an organic fraction and an inorganic fraction.

**Step 1 – list the organic fraction:** Amylase, lysozyme, lactoferrin, peroxidase, secretory IgA, mucins and urea.

**Step 2 – list the inorganic fraction:** Sodium, potassium, chloride, bicarbonate, **calcium**, **phosphate**, thiocyanate and fluoride.

**Step 3 – pick the two that matter to enamel:** Enamel is hydroxyapatite, $Ca_{10}(PO_4)_6(OH)_2$. The only salivary ions that can build or dissolve it are **calcium** and **phosphate**, because they are its own building blocks.

**Step 4 – define supersaturation:** Saliva carries calcium and phosphate at concentrations above those needed to saturate a solution with respect to hydroxyapatite. The ion product in saliva therefore exceeds the solubility product of enamel.

**Step 5 – state the consequence:** Because the solution is supersaturated, the net movement of ions at resting pH is **into** the tooth rather than out of it. An early subsurface white spot lesion can therefore take calcium and phosphate back and remineralise, provided the surface layer is intact.

**Step 6 – note the practical use:** This is the whole rationale behind casein phosphopeptide-amorphous calcium phosphate pastes and behind advising a caries-active patient not to have a dry mouth.

**Why the other options are wrong:**

- A, sodium and chloride: are present in saliva, and their concentration rises with flow rate, but neither is a component of hydroxyapatite, so they cannot supersaturate saliva with respect to enamel.
- C, potassium and thiocyanate: both occur in saliva. Potassium is higher in saliva than in plasma, and thiocyanate feeds the antibacterial peroxidase system, but neither builds enamel.
- D, iron and iodide: are trace constituents only, and they play no part in mineral exchange with the tooth.

**Final Answer:** Calcium and phosphate $\Rightarrow$ B

**Answer: (B)** Go Back to Q5

## Q6.

### Solution

**Concept – essential means the body cannot build the carbon skeleton:** It does not mean the amino acid is more important. It means there is no human synthetic route to it, so the diet is the only source.

**Step 1 – recall the list of essential amino acids:** Methionine, arginine (semi-

essential), threonine, tryptophan, valine, isoleucine, leucine, phenylalanine, histidine (semi-essential) and **lysine**. The mnemonic MATT VIL PHLy is the usual way to hold them.

**Step 2 – test each option against that list:** Lysine appears on the list. Alanine, glutamate and serine do not.

**Step 3 – confirm lysine from first principles:** Humans have no pathway to build the lysine skeleton, and lysine is not reversibly transaminated, so once its amino group is removed it cannot be regenerated from its keto acid. That double failure is why it is strictly essential.

**Step 4 – note the nutritional consequence:** Lysine is the limiting amino acid of cereal protein, which is why a rice-only or wheat-only diet gives poor protein quality, and why a cereal plus pulse combination corrects it.

**Why the other options are wrong:**

- A, alanine: is non-essential. It is made in one step by transaminating pyruvate, which is the basis of the glucose-alanine cycle.
- B, glutamate: is non-essential. It is made by reductively aminating alpha-ketoglutarate through glutamate dehydrogenase, and it is the hub of transamination.
- D, serine: is non-essential. It is made from the glycolytic intermediate 3-phosphoglycerate.

**Final Answer:** Lysine $\Rightarrow$ C

**Answer: (C)** Go Back to Q6

## Q7.

### Solution

**Concept – the three classes of RNA and their share of the total:** The question is about abundance by mass, so match each class to how much of the cell's RNA it accounts for.

**Step 1 – name the three classes and their jobs:** Messenger RNA carries the coding message from DNA. Transfer RNA brings the amino acid. Ribosomal RNA is the structural and catalytic core of the ribosome.

**Step 2 – give their approximate shares:** Ribosomal RNA is about 80 percent of cellular RNA, transfer RNA about 15 percent, and messenger RNA only about 5 percent or less.

**Step 3 – rank them:** rRNA $>$ tRNA $>$ mRNA. The most abundant is therefore

**ribosomal RNA**.

**Step 4 – explain why rRNA dominates:** A single cell holds many thousands of ribosomes, each one built from several large rRNA molecules (28S, 18S, 5.8S and 5S in humans). rRNA is also metabolically stable and long lived, so it accumulates.

**Step 5 – explain why mRNA is the smallest share:** mRNA is transcribed on demand and is deliberately short lived, being degraded within minutes to hours so that gene expression can be switched off quickly. A molecule with a short half-life never builds up a large steady-state pool.

**Why the other options are wrong:**

- B, mRNA: is the **least** abundant of the three despite being the most diverse in sequence, because of its rapid turnover.
- C, tRNA: is the second most abundant at around 15 percent. It is the smallest RNA by size (about 75 nucleotides), which is a common source of confusion with abundance.
- D, snRNA: is a minor species confined to the nucleus, where it forms the spliceosome. It is nowhere near the major classes in quantity.

**Final Answer:** Ribosomal RNA $\Rightarrow$ A

**Answer: (A)** Go Back to Q7

## Q8.

### Solution

**Concept – recognise the diabetic emergency at the bedside:** Three facts settle it: the type of diabetes, whether ketones are present, and how the patient is breathing.

**Step 1 – use the history:** A type 1 diabetic who has omitted insulin for four days has almost no circulating insulin and a high glucagon to insulin ratio.

**Step 2 – use the ketones:** Unopposed lipolysis floods the liver with fatty acids, which are oxidised to acetoacetate and beta-hydroxybutyrate. Strongly positive urine ketones plus the sweet breath (acetone) confirm ketosis. The diagnosis is **diabetic ketoacidosis**.

**Step 3 – label the acid base disturbance:** Acetoacetate and beta-hydroxybutyrate are fixed acids. They consume bicarbonate, so bicarbonate falls and pH falls: a **metabolic acidosis**.

**Step 4 – decide the anion gap:** The ketoacid anions themselves are unmeasured anions. They take the place of bicarbonate in the electroneutrality sum while

chloride stays the same, so the anion gap **rises**. This is a **high anion gap** metabolic acidosis.

**Step 5 – name the breathing:** The acidosis stimulates the central and peripheral chemoreceptors, which drive deep, rapid, sighing respiration to blow off carbon dioxide as compensation. That pattern is **Kussmaul breathing**.

**Step 6 – assemble the answer:** Diabetic ketoacidosis, a high anion gap metabolic acidosis, with Kussmaul breathing. All three parts must be right for the option to be correct.

**Why the other options are wrong:**

- A: hyperosmolar hyperglycaemic state occurs in type 2 diabetes, has glucose usually above 600 mg/dL, and characteristically has **no significant ketosis**. It is also not an alkalosis, and Cheyne-Stokes breathing is a waxing and waning pattern of heart failure and brainstem disease.
- C: gets the diagnosis right but the chemistry wrong. Retained ketoacid anions must raise the gap, so a **normal** anion gap is impossible here. Biot breathing is irregular ataxic breathing of medullary damage.
- D: hypoglycaemia would give a **low** glucose, not 480 mg/dL, and no ketonuria of this degree. Respiratory acidosis needs a high $PaCO_2$, whereas this patient is hyperventilating and blowing $CO_2$ off.

**Final Answer:** Diabetic ketoacidosis, high anion gap metabolic acidosis, Kussmaul breathing $\Rightarrow$ B

**Answer: (B)** Go Back to Q8

## Q9.

### Solution

**Concept – thirst is a regulated reflex, not a random sensation:** Like any reflex it has a sensor, an integrating centre and an effector, and the question asks for the sensor.

**Step 1 – identify the stimulus:** Water loss without salt loss concentrates the extracellular fluid, so plasma osmolality rises from 288 to 298 mOsm/kg.

**Step 2 – name the sensor:** The sensors are **osmoreceptors**, specialised neurons that lie in the **hypothalamus**, in the circumventricular organs around the third ventricle (the organum vasculosum of the lamina terminalis and the subfornical organ).

**Step 3 – explain how they sense:** These neurons sit outside the blood brain

barrier, so they are directly exposed to plasma. When plasma osmolality rises, water leaves the osmoreceptor cell, it shrinks, and the shrinkage opens stretch-sensitive channels that make the cell fire.

**Step 4 – follow the two outputs:** The signal reaches the thirst centre in the lateral hypothalamus, producing the conscious urge to drink. In parallel it drives the supraoptic and paraventricular nuclei to release ADH, so the kidney conserves water.

**Step 5 – note the sensitivity:** A rise of only 1 to 2 percent in osmolality, that is 2 to 3 mOsm/kg, is enough to trigger thirst. The 10 mOsm/kg rise here is a powerful stimulus, which is why he is intensely thirsty.

**Step 6 – name the second, weaker stimulus:** A fall in blood volume or pressure of more than about 10 percent also causes thirst, through baroreceptors and angiotensin II. But the primary and most sensitive stimulus, and the one operating here, is osmotic.

**Why the other options are wrong:**

- B, carotid body: holds **chemoreceptors** for arterial oxygen, carbon dioxide and pH, which regulate ventilation. It does not sense osmolality.
- C, macula densa: senses **sodium chloride delivery** to the distal tubule and controls renin release and tubuloglomerular feedback. It is a renal sensor, not a thirst sensor.
- D, right atrial wall: holds **volume** (low pressure stretch) receptors, which release atrial natriuretic peptide when volume rises. It signals fullness, not concentration.

**Final Answer:** The hypothalamus $\Rightarrow$ $\boxed{\text{A}}$

**Answer: (A)** Go Back to Q9

## Q10.

### Solution

**Concept – two lymphocytes, two kinds of immunity:** Both come from one marrow stem cell, and the organ each matures in decides what it goes on to do.

**Step 1 – take the left branch of the figure:** The B lymphocyte both arises and matures in the **bone marrow** in humans (the bursa of Fabricius is the bird equivalent, which is where the letter B came from).

**Step 2 – state what the B cell does:** On antigen challenge it becomes a plasma cell and secretes antibody. That is **humoral, antibody mediated** immunity, as the

figure shows on the left.

**Step 3 – take the right branch, cell Y:** The stem cell destined to be a T lymphocyte leaves the marrow while immature and travels to the **thymus**. The thymus is organ Z.

**Step 4 – state what the thymus does to it:** In the thymus the cell rearranges its T cell receptor and undergoes positive and negative selection, so that it recognises antigen presented on MHC but does not attack self.

**Step 5 – state what the mature T cell does:** CD4 helper T cells direct the response and CD8 cytotoxic T cells kill infected and tumour cells directly. This is **cell mediated** immunity, exactly as the stem specifies for Y.

**Step 6 – read the answer off:** Cell Y is a T lymphocyte and organ Z is the thymus.

**Why the other options are wrong:**

- A, B lymphocyte and bone marrow: is a true pairing in itself, but it describes the **left** branch of the figure. Y is the branch that leaves the marrow and mediates cell mediated immunity, so it cannot be the B cell.
- B, B lymphocyte and thymus: mixes the two lines. B cells never mature in the thymus.
- C, T lymphocyte and gut associated lymphoid tissue: gets the cell right but the organ wrong. GALT is a peripheral site where mature lymphocytes meet antigen; it is secondary lymphoid tissue, not the site of T cell maturation.

**Final Answer:** T lymphocyte and thymus $\Rightarrow$ D

**Answer: (D)** Go Back to Q10

## Q11.

### Solution

**Concept – osmotic pull depends on particle number, not particle size:** Whichever plasma protein has the most molecules per litre will dominate the osmotic effect, and that protein is albumin.

**Step 1 – give the figures:** Total plasma protein is about 7 g/dL. Albumin is about 4 g/dL of that, roughly 60 percent, and the globulins plus fibrinogen make up the rest.

**Step 2 – add the size point:** Albumin is also the **smallest** major plasma protein at about 69 kDa. A given mass of a small protein contains more molecules than the same mass of a large one.

**Step 3 – combine the two:** Most protein by mass plus smallest molecule means the greatest number of dissolved particles. Albumin therefore accounts for about **75 to 80 percent** of the colloid osmotic pressure of plasma.

**Step 4 – state the value and its purpose:** Plasma oncotic pressure is about 25 mmHg. It is the inward force that pulls fluid back into the capillary and opposes the outward hydrostatic push, so it keeps water in the vascular compartment.

**Step 5 – check with a clinical test:** When albumin falls, in nephrotic syndrome, cirrhosis or kwashiorkor, oncotic pressure falls, fluid stays in the interstitium, and the patient becomes oedematous. That the deficiency produces exactly this consequence confirms the role.

**Step 6 – note albumin's second job:** Albumin also transports bilirubin, free fatty acids, calcium and many drugs. That is a real function, but it is not the major one and is not the one the numbers in the stem point to.

**Why the other options are wrong:**

- B, carrying oxygen: is the job of **haemoglobin**, which is inside the red cell and is not a plasma protein at all.
- C, forming the fibrin mesh: is the job of **fibrinogen**. Fibrinogen is a plasma protein, but it is only about 0.3 g/dL and it is large, so it contributes little oncotic pressure.
- D, acting as antibody: is the job of the **gamma globulins** (immunoglobulins), a different electrophoretic band from albumin.

**Final Answer:** Generating the colloid osmotic (oncotic) pressure of plasma $\Rightarrow$ A

**Answer: (A)** Go Back to Q11

## Q12.

### Solution

**Concept – the Erlanger and Gasser classification:** Nerve fibres are graded A, B and C, and diameter plus myelination together fix the conduction velocity.

**Step 1 – set out the classes:** Type A fibres are large and myelinated and subdivide into alpha, beta, gamma and delta in falling order of size. Type B fibres are small and myelinated. Type **C** fibres are the smallest and are **unmyelinated**.

**Step 2 – give the numbers:** A-alpha are 12 to 20 micrometres and conduct at 70 to 120 m/s. A-delta are 2 to 5 micrometres and conduct at 12 to 30 m/s. B fibres are about 3 micrometres and conduct at 3 to 15 m/s. C fibres are 0.5 to 1.5 micrometres and conduct at only 0.5 to 2 m/s.

**Step 3 – read the figure:** Z is drawn with the smallest diameter and, unlike the other two, has no outer myelin ring. Both features point to the C fibre.

**Step 4 – explain why diameter matters:** A thicker axon has a larger cross-sectional area and therefore a lower internal longitudinal resistance. Current spreads further ahead of the active region, so threshold is reached sooner downstream and the impulse moves faster.

**Step 5 – explain why myelin matters:** Myelin lets the impulse jump from node to node (saltatory conduction). A C fibre has no myelin, so the impulse must be regenerated continuously along every patch of membrane, which is slow.

**Step 6 – combine and apply:** Smallest diameter plus no myelin makes the C fibre the slowest of all. Clinically these are the fibres of slow, dull, burning second pain and of postganglionic sympathetic outflow, which is why the ache after a dental injury arrives late and lingers.

**Why the other options are wrong:**

- B, A-alpha: is the **largest** and **fastest** fibre of all, serving proprioception and skeletal motor supply. It is the exact opposite of Z.
- C, B fibres: are small but they are **myelinated** (preganglionic autonomic fibres), so at 3 to 15 m/s they are still several times faster than C fibres.
- D, A-delta: are thinly myelinated and carry fast, sharp first pain and cold at 12 to 30 m/s. Being myelinated they cannot be Z.

**Final Answer:** C fibres $\Rightarrow$ A

**Answer: (A)** Go Back to Q12

## Q13.

### Solution

**Concept – fatiguable weakness that recovers with rest is a postsynaptic disease:** The pattern of the weakness, not its distribution, tells you where the lesion sits.

**Step 1 – name the disease:** Ptosis and jaw fatigue that worsen with use and through the day, and improve after rest, are **myasthenia gravis**.

**Step 2 – name the antibody target:** The autoantibodies are IgG directed against the **nicotinic acetylcholine receptor** on the **postsynaptic** membrane of the motor end plate. About 15 percent of the remainder instead have antibodies to muscle specific kinase (MuSK).

**Step 3 – explain the three ways the antibody harms:** It blocks the acetyl-

choline binding site; it cross-links receptors and accelerates their internalisation and degradation; and it fixes complement, which destroys the folds of the end plate.

**Step 4 – explain why the weakness fatigues:** Normally each nerve impulse releases far more acetylcholine than is needed, a safety margin. With receptors depleted that margin is gone. Repeated stimulation progressively empties the readily releasable vesicle pool, so successive end plate potentials fall below threshold and more and more fibres drop out.

**Step 5 – link that to the test:** That progressive failure is precisely what a **decremental response** on repetitive nerve stimulation records, and it is why rest, which lets the vesicle pool refill, restores strength.

**Step 6 – note the dental relevance:** Jaw and tongue fatigue during a long appointment, and a real risk with muscle relaxants, sedatives and aminoglycosides, all of which worsen neuromuscular transmission in these patients.

**Why the other options are wrong:**

- A, presynaptic voltage-gated calcium channels: is the target in **Lambert-Eaton myasthenic syndrome**. Less acetylcholine is released, so strength **improves** with brief repeated effort and repetitive stimulation shows an **incremental** response. Both are the reverse of this patient.
- B, acetylcholinesterase: is not an autoimmune target. Blocking it (by neostigmine or by an organophosphate) **raises** synaptic acetylcholine. Indeed anticholinesterases are the treatment of myasthenia, not its cause.
- C, peripheral myelin: is attacked in Guillain-Barre syndrome, which gives an ascending flaccid weakness with lost reflexes, not a fluctuating fatiguable ocular and bulbar weakness.

**Final Answer:** The postsynaptic nicotinic acetylcholine receptor $\Rightarrow$ $\boxed{D}$

**Answer: (D)** Go Back to Q13

## Q14.

### Solution

**Concept – the two pituitary lobes are two different organs:** They differ in origin, in their link to the hypothalamus, and therefore in what they can do.

**Step 1 – use the clue given about lobe P:** The stem says P makes nothing itself and only stores peptides made in hypothalamic neurons and delivered down their axons. That is the defining property of the **posterior pituitary (neurohypoph-**

**ysis)**, and the orange axons in the figure mark it.

**Step 2 – explain why it can store but not synthesise:** The posterior lobe is a downgrowth of neural ectoderm from the floor of the diencephalon. It is nervous tissue, made of axon terminals and pituicytes, with no glandular cells at all.

**Step 3 – name the cell bodies:** The hormones are synthesised in the **supraoptic** and **paraventricular** nuclei of the hypothalamus, packaged with neurophysin, and transported down the hypothalamo-hypophyseal tract.

**Step 4 – name the two hormones:** Only two are stored and released from lobe P: **oxytocin** and **antidiuretic hormone (vasopressin)**. That is the answer.

**Step 5 – contrast lobe Q:** Lobe Q is the anterior pituitary (adenohypophysis), derived from Rathke's pouch, that is oral ectoderm. It is true glandular tissue and it **does** synthesise its hormones: GH, prolactin, ACTH, TSH, FSH and LH.

**Step 6 – contrast the connection:** The anterior lobe has no nerve supply from the hypothalamus. It is controlled instead by releasing and inhibiting hormones carried in the **hypothalamo-hypophyseal portal blood vessels**. The posterior lobe is controlled by direct nerve fibres. That single difference is the key to the whole question.

**Why the other options are wrong:**

- A, growth hormone and prolactin: are both synthesised by **anterior** lobe cells (somatotrophs and lactotrophs), so they belong to lobe Q.
- B, ACTH and TSH: are anterior lobe tropic hormones from corticotrophs and thyrotrophs. They are made there, which contradicts the stem's statement that P makes nothing.
- D, FSH and LH: are the anterior lobe gonadotrophins, again from lobe Q.

**Final Answer:** Oxytocin and antidiuretic hormone $\Rightarrow$ C

**Answer: (C)** Go Back to Q14

# Answer Key

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