NEET MDS General Physiology and Biochemistry

Sample Paper – 7

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 30 year old man on antitubercular therapy reports tingling, numbness and burning of both feet two months after starting isoniazid. The vitamin that is routinely co-prescribed with isoniazid to prevent this peripheral neuropathy is:

(A) Thiamine (vitamin B1)

(B) Riboflavin (vitamin B2)

(C) Niacin (vitamin B3)

(D) Pyridoxine (vitamin B6)

Q2. A 26 year old woman has a microcytic hypochromic blood picture with atrophic glossitis and koilonychia. Dietary iron is absorbed chiefly at:

(A) The duodenum and upper jejunum

(B) The stomach

(C) The terminal ileum

(D) The colon

Enzymes and Enzyme Kinetics

Q3. One International Unit (IU) of enzyme activity is the amount of enzyme that converts one micromole of substrate per minute under defined assay conditions. The **specific activity** of an enzyme preparation is:

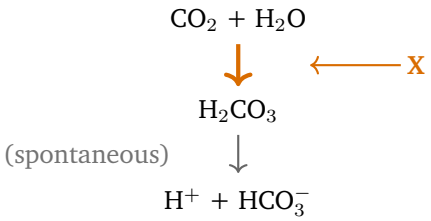
(A) The number of enzyme units per milligram of total protein

(B) The number of substrate molecules converted per enzyme molecule per second

(C) The total number of enzyme units present in the whole preparation

(D) The number of enzyme units per litre of assay buffer

Q4. The scheme below shows a reaction that proceeds far too slowly on its own to serve the body, and that is accelerated many thousand-fold by the enzyme marked **X**. This enzyme is present in high concentration in the red blood cell and in salivary duct cells. Enzyme **X** is:



(A) Salivary amylase

(B) Lactate dehydrogenase

(C) Carbonic anhydrase

(D) Catalase

Metabolism of Carbohydrate, Lipid and Protein

Q5. Before a full mouth rehabilitation, a diabetic patient is sent for a glycated haemoglobin (HbA1c) estimation. The HbA1c value tells the clinician:

(A) The average plasma glucose over the preceding 8 to 12 weeks

(B) The plasma glucose at the moment the sample was drawn

(C) The average plasma glucose over the preceding 24 hours

(D) The average plasma glucose over the preceding 7 days

Q6. A one year old infant has progressive loss of motor milestones, an exaggerated startle response to sound and a cherry red spot at the macula. In this sphingolipidosis, the ganglioside GM2 accumulates in neuronal lysosomes because of a deficiency of:

(A) Glucocerebrosidase

(B) Hexosaminidase A

(C) Sphingomyelinase

(D) Alpha-galactosidase A

Q7. Two weeks after extensive maxillofacial surgery, a patient managing on sips of clear fluid is found to be excreting distinctly more nitrogen in the urine than he is taking in as dietary protein. This state is:

(A) Positive nitrogen balance, indicating net synthesis of body protein

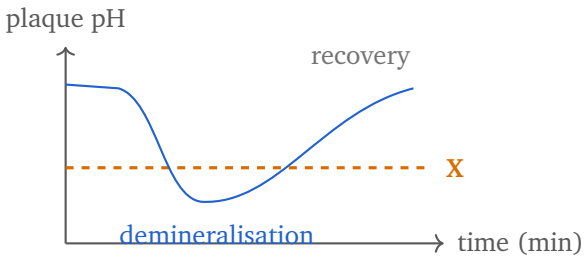
(B) Negative nitrogen balance, indicating net breakdown of body protein

(C) Nitrogen equilibrium, the expected state of a healthy adult

(D) Positive nitrogen balance, the expected state of a healthy adult

Acid Base Balance and Body Fluids

Q8. The trace below plots plaque pH after a sugary rinse. Salivary bicarbonate and phosphate buffers then return the pH to resting level. The dashed line marked **X** is the pH below which enamel hydroxyapatite starts to dissolve. That critical pH is approximately:



(A) 7.4

(B) 6.8

(C) 5.5

(D) 4.0

Q9. A patient with untreated cranial diabetes insipidus loses several litres of dilute urine, that is almost pure water, over a day. The type of dehydration produced and its effect on the fluid compartments is:

(A) Hypertonic dehydration, in which water is drawn out of the cells so the intracellular compartment shrinks as well

(B) Hypotonic dehydration, in which water is drawn out of the cells so the intracellular compartment shrinks

(C) Isotonic dehydration, in which the loss stays confined to the extracellular compartment and cell volume does not change

(D) Hypertonic dehydration, in which water moves into the cells so the intracellular compartment expands

Blood and Haemostasis

Q10. A 22 year old man attends with boggy, spongy enlargement of the gingivae that bleeds on the lightest touch, along with fever and pallor. The total leucocyte count is 92,000 per cubic millimetre and the peripheral smear is packed with blast cells. The most likely diagnosis is:

(A) Iron deficiency anaemia

(B) Acute myeloid leukaemia

(C) Aplastic anaemia

(D) A leukaemoid reaction to a periodontal abscess

Q11. A group O patient is transfused group A blood by mistake and, within minutes, develops fever, loin pain, hypotension and haemoglobinuria. The mechanism of this acute intravascular haemolysis is:

(A) Donor IgG antibodies slowly coat the recipient's red cells, which are then removed in the spleen over days

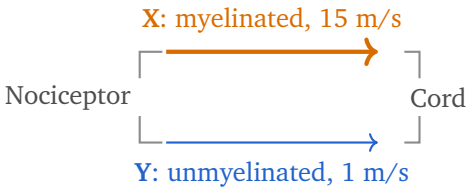
(B) The donor plasma is hypotonic and lyses the recipient's red cells osmotically

(C) Preformed IgM anti-A in the recipient binds the donor red cells and drives complement to the membrane attack complex, lysing them inside the vessels

(D) Recipient T lymphocytes are sensitised to donor red cells and destroy them over the following week

Nerve, Muscle and Endocrine Physiology

Q12. In the scheme below, two nociceptive fibres run from the pulp to the spinal cord. Fibre **X** is myelinated and conducts at about 15 m/s; fibre **Y** is unmyelinated and conducts at about 1 m/s. Fibre **X** carries:



(A) Slow, dull, burning second pain, and is a C fibre

(B) Fast, sharp, well localised first pain, and is an A-delta fibre

(C) Fast, sharp, well localised first pain, and is a C fibre

(D) Slow, dull, burning second pain, and is an A-delta fibre

Q13. Taste receptor cells respond to five basic modalities: sweet, sour, salty, bitter and umami. Taste sensation from the **posterior one third of the tongue**, including the circumvallate papillae, is carried by:

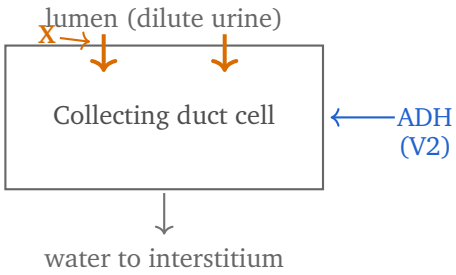
(A) The chorda tympani branch of the facial nerve

(B) The lingual branch of the mandibular nerve

(C) The vagus nerve

(D) The glossopharyngeal nerve

Q14. The figure shows a collecting duct cell responding to antidiuretic hormone (vasopressin). ADH binds the V2 receptor on the basolateral surface and, through cyclic AMP, causes the water channel marked **X** to be inserted into the apical membrane. Channel **X** is:



(A) The epithelial sodium channel (ENaC)

(B) The Na-K-2Cl cotransporter

(C) Aquaporin-1, inserted into the basolateral membrane

(D) Aquaporin-2

Detailed Solutions

Q1.

Solution

Concept – isoniazid and vitamin B6: Isoniazid does not damage nerves directly. It creates a functional deficiency of one specific vitamin.

Step 1 – name the vitamin: The vitamin involved is **pyridoxine, vitamin B6**.

Step 2 – name its coenzyme form: Pyridoxine is converted to **pyridoxal phosphate (PLP)**, the working coenzyme.

Step 3 – state the mechanism of the drug interaction: Isoniazid is a hydrazide. It condenses with pyridoxal phosphate to form isonicotinyl hydrazone, which is inactive and is excreted in the urine. Isoniazid also inhibits pyridoxine kinase, the enzyme that makes PLP.

Step 4 – link the deficiency to the nerve: PLP is needed for the synthesis of sphingolipids in myelin and for the transamination and decarboxylation reactions of amino acids. Losing PLP therefore hits peripheral nerve first, giving a symmetrical distal sensory neuropathy of tingling, numbness and burning feet.

Step 5 – state the practical point: Pyridoxine 10 to 25 mg daily is given alongside isoniazid to prevent this. It is prophylaxis, not treatment of tuberculosis.

Why the other options are wrong:

- A, thiamine: its deficiency also causes a neuropathy, but that is dry beriberi from poor diet or alcohol. Isoniazid does not chelate thiamine, and thiamine is not co-prescribed with it.
- B, riboflavin: its deficiency gives ocular and mucosal signs, not a peripheral neuropathy, and it has no interaction with isoniazid.
- C, niacin: isoniazid does mildly interfere with niacin synthesis from tryptophan, but the classical, examinable, routinely prevented adverse effect is the B6 dependent neuropathy, and it is pyridoxine that is prescribed with the drug.

Final Answer: Pyridoxine (vitamin B6) $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q1

Q2.

Solution

Concept – iron is absorbed high in the small bowel: Iron has one dedicated absorptive window, and it sits immediately beyond the stomach.

Step 1 – state the site: Iron is absorbed chiefly in the **duodenum and the upper jejunum**.

Step 2 – explain why that site: Gastric acid arriving in the duodenum keeps iron soluble and in the ferrous (Fe^{2+}) state. Further down the gut the contents turn alkaline and iron precipitates as insoluble ferric hydroxide, so it can no longer be taken up.

Step 3 – follow the transport: Duodenal cytochrome B reduces dietary ferric iron to ferrous iron. The divalent metal transporter DMT-1 on the brush border carries it into the enterocyte. Ferroportin on the basolateral side then exports it to plasma, where it rides on transferrin.

Step 4 – name the helpers and blockers: Ascorbic acid keeps iron reduced and improves uptake. Phytates, tannins in tea, antacids and calcium bind iron and reduce it. Hepcidin from the liver degrades ferroportin and shuts absorption down.

Step 5 – link to the blood picture: Without enough iron the marrow cannot fill red cells with haemoglobin, so it makes small, pale cells. That is the **microcytic hypochromic** anaemia described, with atrophic glossitis and koilonychia as the classical mucosal and nail signs.

Why the other options are wrong:

- B, stomach: it supplies the acid that makes absorption possible and reduces iron, but essentially no iron crosses the gastric mucosa itself.
- C, terminal ileum: this is the site for **vitamin B12** bound to intrinsic factor and for bile salts, not for iron.
- D, colon: absorbs water and electrolytes. Its alkaline contents keep iron insoluble.

Final Answer: The duodenum and upper jejunum $\Rightarrow$ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – activity versus specific activity: Total activity tells you how much enzyme work a tube can do. Specific activity tells you how pure the enzyme in that tube is.

Step 1 – fix the unit of activity: One IU converts 1 micromole of substrate per minute under stated conditions of pH, temperature and saturating substrate.

Step 2 – define specific activity: Specific activity = **enzyme units divided by milligrams of total protein**, that is IU per mg protein.

Step 3 – work through an example: Suppose a crude extract has 600 units in 300 mg protein. $600 \div 300 = 2$ IU per mg. After a purification step, 480 units remain in 24 mg protein. $480 \div 24 = 20$ IU per mg.

Step 4 – read the meaning: Total activity fell from 600 to 480 units, so some enzyme was lost. Yet specific activity rose from 2 to 20 IU per mg, a ten-fold purification, because far more contaminating protein was removed than enzyme. This is exactly why specific activity, and not total activity, is the index of purity.

Step 5 – note the ceiling: Specific activity reaches a plateau when the enzyme is pure, since there is then no other protein left to remove.

Why the other options are wrong:

- B, molecules converted per enzyme molecule per second: this is the **turnover number** (k_{cat}), which needs the molar concentration of pure enzyme, not milligrams of total protein.
- C, total units in the whole preparation: this is **total activity**. It ignores protein content, so it cannot report purity.
- D, units per litre: this is a **concentration** of activity, the form used for serum enzyme reports. It says nothing about protein content either.

Final Answer: Enzyme units per milligram of total protein $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – read the enzyme off its reaction: The diagram gives the substrates and the products. Only one enzyme handles that pair.

Step 1 – read the marked step: Step X hydrates carbon dioxide: $CO_2 + H_2O \rightarrow$

H_2CO_3.

Step 2 – name the enzyme: That hydration is catalysed by **carbonic anhydrase**, a zinc containing lyase.

Step 3 – explain why an enzyme is needed at all: The uncatalysed hydration is far too slow for a red cell that spends only about a second in a capillary. Carbonic anhydrase speeds it up by several orders of magnitude, which is why it is one of the fastest enzymes known.

Step 4 – follow it in the red cell: Tissue CO_2 diffuses into the red cell, carbonic anhydrase converts it to carbonic acid, which dissociates to H^+ and HCO_3^- as the second arrow shows. Bicarbonate leaves in exchange for chloride, the chloride shift, and this bicarbonate route carries roughly 70 percent of all CO_2 back to the lungs. In the lung the same enzyme runs the reaction backwards so CO_2 can be exhaled.

Step 5 – follow it in saliva: Salivary duct cells use carbonic anhydrase to generate the bicarbonate they secrete. Carbonic anhydrase VI is itself secreted into saliva and sits in the pellicle. This is the source of the bicarbonate buffer that neutralises plaque acid, which is why the enzyme matters dentally.

Why the other options are wrong:

- A, salivary amylase: it is a hydrolase that splits alpha-1,4 bonds in starch. It has nothing to do with CO_2.
- B, lactate dehydrogenase: it is an oxidoreductase interconverting pyruvate and lactate using NADH. Its substrates are not in this diagram.
- D, catalase: it breaks hydrogen peroxide down to water and oxygen. It also does not touch carbon dioxide.

Final Answer: Carbonic anhydrase $\Rightarrow$ C

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – HbA1c is a memory, not a snapshot: The value is set by how long the red cell lives, not by what the patient ate this morning.

Step 1 – state what HbA1c is: It is haemoglobin A whose N-terminal valine on the beta chain has been **non-enzymatically glycated** by glucose.

Step 2 – note that the reaction is irreversible: The initial Schiff base rearranges to a stable ketoamine (Amadori product). Once formed, it stays on that

haemoglobin molecule for the rest of the cell's life.

Step 3 – bring in the red cell: The red cell survives about 120 days. So the glycated fraction accumulates in proportion to the glucose the cell has been bathed in across its whole span.

Step 4 – weight the time period: Recent glucose contributes more than old glucose because the cell population is constantly renewing. The value therefore reflects the average plasma glucose of roughly the last **8 to 12 weeks**, weighted towards the last month.

Step 5 – read the clinical use: A value below 5.7 percent is normal, 5.7 to 6.4 percent is prediabetes, and 6.5 percent or more is diabetic. Under 7 percent is the usual control target, and it is the figure the surgeon wants before elective full mouth rehabilitation, because poor long-term control predicts infection and delayed healing.

Step 6 – note when it misleads: Anything that shortens red cell life, such as haemolysis or recent blood loss, falsely lowers HbA1c. That is a limitation of the memory, not of the assay.

Why the other options are wrong:

- B, glucose at the moment of sampling: that is a random or fasting plasma glucose. HbA1c does not change with a single meal or a single missed dose.
- C, the preceding 24 hours: no routine test reports that. Continuous glucose monitoring would, and HbA1c is far too slow for it.
- D, the preceding 7 days: this is closest to **fructosamine**, glycated serum albumin, which reflects about 2 to 3 weeks, since albumin turns over much faster than the red cell. It is not HbA1c.

Final Answer: Average plasma glucose over the preceding 8 to 12 weeks $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – sphingolipids and where they jam: Sphingolipids are built on a ceramide backbone. Each one is dismantled in the lysosome by its own enzyme, and losing that enzyme stores that lipid.

Step 1 – place the family: Ceramide plus phosphocholine gives sphingomyelin. Ceramide plus sugars gives cerebrosides, and ceramide plus a sugar chain carrying sialic acid gives gangliosides. All are heavily concentrated in neuronal membranes.

Step 2 – read the clinical picture: Loss of milestones, an exaggerated startle to sound (hyperacusis) and a **cherry red macula** in an infant is the classical triad of **Tay-Sachs disease**.

Step 3 – name the stored lipid: The lipid that accumulates is ganglioside **GM2**, exactly as the question states.

Step 4 – name the missing enzyme: GM2 is degraded by **hexosaminidase A**, which strips the terminal N-acetylgalactosamine. Its deficiency is Tay-Sachs disease. The inheritance is autosomal recessive.

Step 5 – explain the cherry red spot: Ganglioside laden ganglion cells make the retina around the macula look pale white. The fovea itself has no ganglion cells, so the normal red choroid shows through as a red dot against the pale ring. It is a contrast effect, not a deposit.

Step 6 – note the discriminator: Tay-Sachs has **no hepatosplenomegaly**. That single negative separates it from the other storage disorders below.

Why the other options are wrong:

- A, glucocerebrosidase: its deficiency is **Gaucher disease**, which stores glucocerebroside and presents with massive hepatosplenomegaly, bone crises and Gaucher cells with crumpled tissue paper cytoplasm.
- C, sphingomyelinase: its deficiency is **Niemann-Pick disease**, which stores sphingomyelin. It also shows a cherry red spot, but it comes with prominent hepatosplenomegaly and foamy histiocytes, which this infant does not have.
- D, alpha-galactosidase A: its deficiency is **Fabry disease**, X-linked, storing globotriaosylceramide. It presents in older children and adults with acroparaesthesia, angiokeratomas and renal failure, not with infantile regression.

Final Answer: Hexosaminidase A $\Rightarrow$ B

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – nitrogen balance is simple bookkeeping: Nitrogen enters only as dietary protein and leaves mainly as urea in urine, plus faecal and skin losses. Compare the two sides.

Step 1 – write the rule: Nitrogen balance = nitrogen intake − nitrogen output.

Step 2 – define the three states: Balance positive, that is intake greater than

output: nitrogen is being retained, so body protein is being **built**. Seen in growing children, pregnancy, lactation, convalescence and athletic training. Balance zero, that is intake equal to output: **nitrogen equilibrium**, the normal state of a healthy, weight-stable adult. Balance negative, that is output greater than intake: body protein is being **broken down**.

Step 3 – apply it to this patient: He excretes more nitrogen than he takes in, so output is greater than intake and the balance is **negative**.

Step 4 – explain the physiology behind it: Intake is minimal because he is on clear fluids only. At the same time, major surgery drives a catabolic response with raised cortisol, catecholamines and glucagon, so muscle protein is broken down to supply amino acids for gluconeogenesis and for acute phase protein synthesis. Those amino acids are transaminated and deaminated, the ammonia goes to urea, and the urea appears in urine.

Step 5 – note the clinical importance: Sustained negative balance means loss of lean mass, poor wound healing and a higher infection risk. It is the argument for early nutritional support after maxillofacial surgery. Negative balance also occurs in starvation, burns, sepsis, uncontrolled diabetes and when even one essential amino acid is missing from the diet.

Why the other options are wrong:

- A, positive balance with net synthesis: this needs intake to **exceed** output. Here the reverse is stated.
- C, nitrogen equilibrium: this needs intake to **equal** output. The question explicitly says output is distinctly greater.
- D, positive balance as the adult norm: doubly wrong. It contradicts the figures given, and the normal adult is in equilibrium, not in positive balance. A healthy adult who stayed in positive balance would keep gaining lean mass indefinitely.

Final Answer: Negative nitrogen balance, net breakdown of body protein $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – the critical pH of enamel: Enamel and saliva sit in a chemical equilibrium. Below one particular pH that equilibrium tips towards dissolution.

Step 1 – state the number: The critical pH for enamel hydroxyapatite is about

5.5.

Step 2 – explain what the number means: Saliva is normally supersaturated with calcium and phosphate with respect to hydroxyapatite, so the crystal is safe. As pH falls, phosphate ions are progressively protonated to HPO_4^{2-} and then $H_2PO_4^-$, which removes free PO_4^{3-} from solution. At about pH 5.5 saliva stops being saturated and enamel begins to dissolve to restore equilibrium.

Step 3 – read the trace: Resting plaque pH sits around 6.5 to 7. Sugar is fermented by plaque bacteria to lactic and other acids, the pH plunges through the marked line within about 5 minutes, stays below it while demineralisation runs, then climbs back over the next 20 to 40 minutes. This is the Stephan curve, and the area under the dashed line is the damage.

Step 4 – explain the recovery limb, that is the buffering: Saliva does the repair work with two buffers. **Bicarbonate** is the major one in stimulated saliva, with a pK_a near 6.1; it mops up H^+ as H_2CO_3, which carbonic anhydrase splits to CO_2 and water, and the CO_2 escapes, so the acid is genuinely removed. **Phosphate** is the major one in unstimulated saliva, with a pK_a near 6.8, so it is well placed to work around the resting pH. Salivary urea also releases ammonia, which adds alkali.

Step 5 – draw the clinical conclusion: Once the pH climbs back above 5.5, saliva is supersaturated again and the lesion remineralises. A dry mouth or repeated snacking keeps the curve below the line, and that is when cavitation follows.

Why the other options are wrong:

- A, 7.4: this is the pH of blood. Enamel is entirely stable here.
- B, 6.8: this is close to resting plaque and unstimulated salivary pH, and to the phosphate buffer pK_a. Enamel does not dissolve at this pH.
- D, 4.0: enamel is already dissolving well before this. A pH near 4 is relevant to erosion by dietary acids and to the critical pH of **root dentine**, which is higher than that of enamel at about 6.2, not lower. Quoting 4.0 as the enamel figure understates the risk badly.

Final Answer: About pH 5.5 $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – name dehydration by what happens to ECF tonicity: Water and sodium can be lost in any ratio. The ratio decides the tonicity of what is left behind, and tonicity decides which compartment shrinks.

Step 1 – list the three types: Isotonic: water and sodium lost in equal proportion, ECF osmolality stays about 290 mOsm/kg. **Hypertonic**: water lost in excess of sodium, ECF osmolality rises above 290. **Hypotonic**: sodium lost in excess of water, ECF osmolality falls below 290.

Step 2 – read this patient: Cranial diabetes insipidus means no ADH, so the collecting duct cannot reabsorb water. The urine is dilute, that is nearly **pure water**, and sodium is retained.

Step 3 – work out the ECF osmolality: Water leaves, sodium stays. Sodium is dissolved in less water, so plasma sodium and ECF osmolality both **rise**. This is **hypertonic dehydration**.

Step 4 – follow the water across the cell membrane: The cell membrane is freely permeable to water. Water always moves from lower osmolality to higher. The ECF is now the more concentrated side, so water is **drawn out of the cells** into the ECF.

Step 5 – state which compartments shrink: Both. The ECF shrinks from the urinary loss, and the ICF shrinks because it donates water to the ECF. That donation is also why the circulation is defended for a long time in this type, and why hypotension appears late.

Step 6 – note the clinical signature: Cell shrinkage in the brain gives the thirst, irritability and drowsiness of hypernatraemia. Correction must be slow, because rapid rehydration swells brain cells and causes cerebral oedema.

Why the other options are wrong:

- B, hypotonic dehydration: the tonicity is wrong for the mechanism described. Hypotonic dehydration follows sodium loss in excess of water, as in adrenal insufficiency or replacing salt losses with plain water, and it makes water move **into** the cells, so the ICF expands rather than shrinks. The option also contradicts itself.
- C, isotonic dehydration: this follows acute blood loss or vomiting and diarrhoea replaced proportionally, where the loss really is confined to the ECF. Pure water loss cannot be isotonic.
- D, hypertonic with the ICF expanding: the type is right but the water movement is backwards. Water cannot move into cells when the fluid outside

them is the more concentrated.

Final Answer: Hypertonic dehydration with the intracellular compartment also shrinking $\Rightarrow$ A

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – gingival enlargement can be the first sign of leukaemia: Leukaemia is a clonal proliferation of white cell precursors. The gingiva is one of the tissues those cells infiltrate, and the dentist often sees it first.

Step 1 – read the leucocyte count: Normal total leucocyte count is 4,000 to 11,000 per cubic millimetre. This patient has **92,000**, which is about eight times the upper limit.

Step 2 – read the smear: The smear is packed with **blasts**, that is immature precursors. A marrow that is releasing blasts into blood is not responding to anything, it is being replaced by a clone. That means **acute leukaemia**.

Step 3 – explain the gingival enlargement: Leukaemic cells infiltrate the gingival connective tissue directly, giving the boggy, spongy, bluish-red enlargement that starts at the interdental papilla and can bury the crowns. This is most typical of the **acute myeloid** leukaemias with a monocytic component (FAB M4 and M5), because monocytic cells have the strongest tissue homing.

Step 4 – explain the bleeding, fever and pallor: The expanding clone crowds the marrow out. Loss of megakaryocytes gives thrombocytopenia, hence gingivae that bleed on the lightest touch. Loss of erythroid precursors gives anaemia, hence pallor. Loss of functioning mature neutrophils gives infection, hence fever, even though the total count is huge.

Step 5 – assemble the diagnosis: Grossly raised count, blasts on the smear, gingival infiltration and marrow failure together give **acute myeloid leukaemia**.

Step 6 – state the dental duty: Do not scale, do not extract, do not prescribe and wait. Refer urgently for a haematology opinion. Any surgery here risks uncontrollable bleeding and sepsis.

Why the other options are wrong:

- A, iron deficiency anaemia: it explains pallor, but the total leucocyte count is normal and there are no blasts. It does not cause gingival enlargement or a count of 92,000.

- C, aplastic anaemia: the marrow is empty, so it gives **pancytopenia** with a low leucocyte count and no blasts. The count here is grossly high, which is the opposite.
- D, leukaemoid reaction: a severe infection can push the count above 50,000, but it is a reactive proliferation of **mature** neutrophils with toxic granulation, Dohle bodies and a high leucocyte alkaline phosphatase. A smear packed with blasts is never a leukaemoid reaction, and a local abscess does not infiltrate the whole gingiva.

Final Answer: Acute myeloid leukaemia $\Rightarrow$ B

Answer: (B) Go Back to Q10

Q11.

Solution

Concept – ABO antibodies are the dangerous ones because they are IgM: The class of the antibody decides whether the red cells burst in the vessel or are quietly eaten in the spleen.

Step 1 – identify the antibody present: A group O recipient has **no A or B antigen** on his cells, so he carries **preformed anti-A and anti-B** in plasma from infancy. These are natural antibodies raised against gut bacterial antigens, and they are present without any prior transfusion.

Step 2 – note their class: Natural ABO isoagglutinins are **IgM**, which is a pentamer.

Step 3 – explain why IgM matters: Complement C1q needs two Fc regions held close together to be activated. One pentameric IgM molecule provides that on its own, so IgM is an extremely efficient complement activator. IgG needs two molecules to land side by side, which is far less likely.

Step 4 – follow the complement cascade: Anti-A binds the transfused group A cells. C1 is activated, the classical pathway runs through C4, C2 and C3 to C5, and C5b through C9 assemble the **membrane attack complex**. This punches pores through the red cell membrane.

Step 5 – state the consequence: The cells lyse **inside the blood vessels** within minutes. Free haemoglobin appears in plasma, saturates haptoglobin, spills into urine as haemoglobinuria, and precipitates in tubules to cause acute kidney injury. Anaphylatoxins C3a and C5a release histamine and cause hypotension, while cytokine release causes the fever and loin pain. Tissue factor exposure can set off disseminated intravascular coagulation.

Step 6 – read the timing: Minutes, not days. That immediacy is the fingerprint of preformed IgM plus complement, and it is why an ABO mismatch is the transfusion error that kills.

Why the other options are wrong:

- A, IgG coating with splenic removal: this describes a **delayed extravascular** haemolytic reaction, typical of Rh or Kidd antibodies. It appears days to weeks later with a falling haemoglobin and jaundice, not with hypotension within minutes.
- B, osmotic lysis by hypotonic donor plasma: donor plasma is isotonic. Osmotic lysis is a laboratory phenomenon of putting cells in hypotonic saline, and it has nothing to do with blood groups. If it were true, every transfusion would haemolyse.
- D, T cell mediated destruction over a week: T cells do not lyse red cells, which carry no HLA class I peptides to present in this way. The timescale is also wrong by orders of magnitude. Donor T lymphocytes do matter, but in transfusion associated graft versus host disease, an entirely different entity.

Final Answer: Preformed IgM anti-A activating complement to the membrane attack complex $\Rightarrow$ C

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – nociceptors feed two fibres of very different speed: Pain arrives in two waves because the two fibre types that carry it conduct at very different rates.

Step 1 – read the diagram: X is **myelinated** and runs at about 15 m/s. Y is **unmyelinated** and runs at about 1 m/s.

Step 2 – name the fibre from those two facts: A myelinated nociceptive fibre in the 5 to 30 m/s range is an **A-delta** fibre, 1 to 5 micrometres in diameter. An unmyelinated nociceptive fibre in the 0.5 to 2 m/s range is a **C** fibre. So X is A-delta and Y is C.

Step 3 – explain why myelin makes it fast: Myelin insulates the axon so current jumps from node to node, that is saltatory conduction. Conduction speed also rises with axon diameter. X is both myelinated and thicker, so it wins on both counts.

Step 4 – assign the pain quality to X: A-delta fibres carry **first pain**: sharp, pricking, well localised, and it stops when the stimulus stops. They are well lo-

calised because each fibre serves a small receptive field and they project through the neospinothalamic tract to the ventral posterolateral nucleus and then to the somatosensory cortex, which has a precise map.

Step 5 – assign the pain quality to Y for contrast: C fibres carry **second pain**: dull, burning, aching, poorly localised and persistent. They have large overlapping receptive fields and project through the paleospinothalamic tract to the reticular formation and limbic areas, which is why this pain is diffuse and emotionally unpleasant.

Step 6 – put a clinical face on it: This is exactly the two-stage pain of dentistry. Cold on exposed dentine gives the instant sharp jab of A-delta hypersensitivity. Irreversible pulpitis gives the throbbing, poorly localised, sleep-robbing ache of C fibres. Local anaesthetic blocks the small unmyelinated C fibres first and the A-delta fibres slightly later, which is why sharp pain can outlast the dull ache as a block sets in.

Why the other options are wrong:

- A, slow burning second pain in a C fibre: this correctly describes fibre **Y**, not X. The question asks about X.
- C, fast sharp pain in a C fibre: the pain quality is right for X but the fibre name is wrong. C fibres are unmyelinated and are the slow ones, so they cannot conduct at 15 m/s.
- D, slow dull pain in an A-delta fibre: the fibre name is right for X but the pain quality is wrong. A myelinated fibre conducting at 15 m/s delivers the first, fast pain.

Final Answer: Fast, sharp, well localised first pain in an A-delta fibre $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – taste follows the tongue's developmental origin, not its surface: The tongue is built from two different embryological parts, and each keeps its own nerve for taste.

Step 1 – divide the tongue: The sulcus terminalis separates the anterior two thirds, from the first and second branchial arches, from the posterior one third, from the third arch.

Step 2 – assign the nerves: Anterior two thirds, taste: **facial nerve** through the

chorda tympani. Posterior one third including the circumvallate papillae, taste: **glossopharyngeal nerve**. Epiglottis and posterior pharynx, taste: **vagus** through the internal laryngeal nerve.

Step 3 – place the circumvallate papillae: The circumvallate papillae sit in a V immediately **in front of** the sulcus terminalis, so anatomically they are in the anterior two thirds. Yet their taste is carried by the **glossopharyngeal** nerve, because the third arch mucosa grows forward over them during development. This is the classical exception, and it is what the question is testing.

Step 4 – answer the question: Taste from the posterior one third and from the circumvallate papillae is carried by the glossopharyngeal nerve.

Step 5 – trace the central pathway: All taste fibres, whether VII, IX or X, run to the **nucleus of the solitary tract** in the medulla, then to the ventral posteromedial nucleus of the thalamus, then to the taste cortex in the insula and the frontal operculum.

Step 6 – note the five modalities: Sweet, umami and bitter are detected by G protein coupled receptors, T1R2/T1R3, T1R1/T1R3 and the T2R family, working through gustducin. Sour and salty use ion channels directly, H^+ and Na^+ respectively. All five can be sensed anywhere there are taste buds, so the old tongue map of zones is wrong.

Why the other options are wrong:

- A, chorda tympani of the facial nerve: it carries taste from the **anterior two thirds**, not the posterior one third. Its damage in middle ear surgery causes loss of taste at the tongue tip.
- B, lingual branch of the mandibular nerve: it carries **general sensation**, that is touch, pain and temperature, from the anterior two thirds. It carries no taste of its own; it simply gives the chorda tympani a ride.
- C, vagus nerve: it carries taste from the **epiglottis and pharynx**, a small territory behind the posterior third of the tongue.

Final Answer: The glossopharyngeal nerve $\Rightarrow$ D

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – ADH works by moving water channels, not by pumping water: The collecting duct is waterproof by default. ADH makes it leaky by installing pores.

Step 1 – name the hormone and its origin: Antidiuretic hormone, also called vasopressin, is a nonapeptide made in the supraoptic and paraventricular nuclei of the hypothalamus, carried down axons and stored in the **posterior pituitary**, which releases it.

Step 2 – name the trigger: Hypothalamic osmoreceptors fire when plasma osmolality rises above about 285 mOsm/kg. A fall in blood volume or pressure, sensed by baroreceptors, also releases it.

Step 3 – follow the signal inside the cell: ADH binds the **V2 receptor** on the basolateral membrane. This is a Gs coupled receptor, so adenylyl cyclase raises cyclic AMP, which activates protein kinase A.

Step 4 – identify the channel marked X: Protein kinase A phosphorylates **aquaporin-2**, which sits waiting in cytoplasmic vesicles. The vesicles fuse with the **apical** membrane, inserting AQP-2 into the surface facing the urine. That is channel X.

Step 5 – complete the water's path: Water enters the cell through AQP-2 down the osmotic gradient created by the hypertonic medullary interstitium, then leaves across the basolateral membrane through **aquaporin-3 and aquaporin-4**, which are permanently there. The urine left behind is concentrated, and the volume falls. Withdraw ADH and AQP-2 is endocytosed straight back, so the duct is waterproof again within minutes.

Step 6 – read the disease: No ADH, or no response to it, means AQP-2 is never inserted. Water runs straight through, giving **diabetes insipidus**: several litres a day of dilute urine with an osmolality under 300 mOsm/kg, matching thirst and polydipsia, and a rising plasma sodium. **Cranial** diabetes insipidus is a failure of secretion and responds to desmopressin. **Nephrogenic** diabetes insipidus is a failure of the V2 receptor or of AQP-2 itself, often from lithium, and desmopressin does not help. The excess state, SIADH, is the mirror image with concentrated urine and hyponatraemia.

Why the other options are wrong:

- A, ENaC: it is an apical **sodium** channel of the principal cell and it is upregulated by **aldosterone**, not by ADH. It moves sodium, not water.
- B, Na-K-2Cl cotransporter: it sits in the **thick ascending limb** of the loop of Henle, is the target of furosemide, and builds the medullary gradient. It is

not in the collecting duct and it does not respond to ADH.

- C, aquaporin-1 in the basolateral membrane: AQP-1 is in the **proximal tubule and the descending limb**, where it is always on and is not hormone regulated. The channel ADH inserts is apical, and it is AQP-2.

Final Answer: Aquaporin-2 $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q14

Answer Key

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