NEET MDS General Physiology and Biochemistry

Sample Paper – 3

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 55-year-old woman has a smooth beefy red tongue, tingling of both feet and a macrocytic anaemia. Her gastric parietal cells have been destroyed by autoantibodies, so intrinsic factor is absent and the vitamin cannot be absorbed at the terminal ileum. The deficient vitamin is:

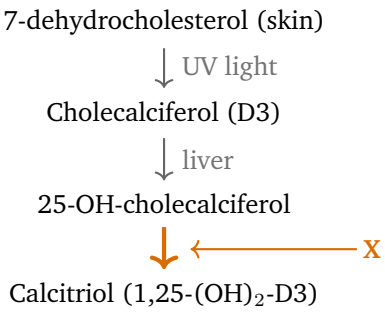
(A) Folic acid

(B) Vitamin B6 (pyridoxine)

(C) Vitamin B12 (cobalamin)

(D) Vitamin B1 (thiamine)

Q2. The chain below shows how vitamin D becomes active. The step marked **X** converts 25-hydroxycholecalciferol into the fully active hormone calcitriol. The enzyme catalysing **X**, and the organ in which it sits, are:



(A) 25-hydroxylase, in the liver

(B) 7-dehydrocholesterol reductase, in the skin

(C) 1-alpha-hydroxylase, in the kidney

(D) 24-hydroxylase, in the kidney

Enzymes and Enzyme Kinetics

Q3. A 58-year-old man reaches casualty 4 hours after crushing chest pain. The laboratory is asked for the creatine kinase isoenzyme that is most specific for cardiac muscle and that rises early after myocardial infarction. That isoenzyme is:

(A) LDH-5

(B) CK-BB (CK-1)

(C) CK-MM (CK-3)

(D) CK-MB (CK-2)

Q4. Hexokinase catalyses: glucose + ATP $\rightarrow$ glucose-6-phosphate + ADP. Under the six IUBMB classes, this reaction belongs to the class:

(A) Oxidoreductase

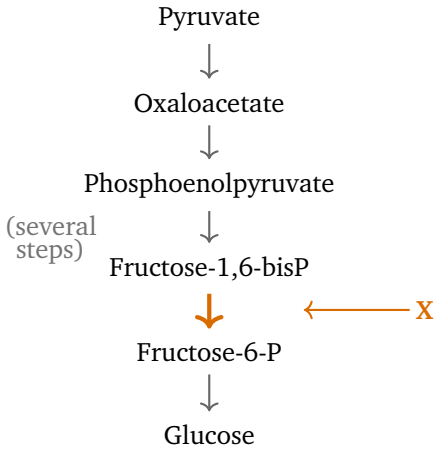
(B) Transferase

(C) Hydrolase

(D) Ligase

Metabolism of Carbohydrate, Lipid and Protein

Q5. The outline below traces gluconeogenesis from pyruvate to glucose. The step marked **X** bypasses an irreversible glycolytic step and is the rate limiting step of gluconeogenesis. The enzyme catalysing **X** is:



(A) Fructose-1,6-bisphosphatase

(B) Pyruvate carboxylase

(C) Aldolase

(D) Enolase

Q6. A 7-month-old infant has a doll-like face, a massively enlarged liver, severe hypoglycaemia within a few hours of a missed feed, lactic acidosis, hyperuricaemia and hyperlipidaemia. Glycogen structure is normal but the liver cannot release free glucose into the blood. The deficient enzyme is:

(A) Glucose-6-phosphatase

(B) Lysosomal acid alpha-glucosidase

(C) Muscle glycogen phosphorylase

(D) Debranching enzyme (amylo-1,6-glucosidase)

Q7. Statins lower plasma LDL cholesterol by competitively inhibiting the rate limiting enzyme of cholesterol biosynthesis, which converts HMG-CoA into mevalonate. That enzyme is:

(A) HMG-CoA synthase

(B) HMG-CoA lyase

(C) HMG-CoA reductase

(D) Squalene epoxidase

Acid Base Balance and Body Fluids

Q8. A 22-year-old patient panics in the dental chair, breathes rapidly and deeply, and complains of tingling fingers and circumoral numbness. Arterial blood gas shows pH 7.52, $PaCO_2$ 26 mmHg, bicarbonate 22 mEq/L. The correct interpretation is:

(A) Metabolic alkalosis

(B) Respiratory acidosis

(C) Metabolic acidosis

(D) Respiratory alkalosis

Q9. For the bicarbonate buffer system the pKa is 6.1. In a healthy person the plasma bicarbonate is 24 mmol/L and the dissolved carbon dioxide is 1.2 mmol/L. Using the Henderson-Hasselbalch equation, the plasma pH is (take $\log_{10} 20 = 1.3$):

(A) 6.1

(B) 6.8

(C) 7.1

(D) 7.4

Blood and Haemostasis

Q10. An Rh negative mother, already sensitised during her first delivery, is carrying a second Rh positive fetus. The fetus develops erythroblastosis fetalis. The immediate cause of the fetal haemolysis is:

(A) Maternal anti-D IgG crossing the placenta and coating fetal red cells

(B) Maternal anti-D IgM crossing the placenta and coating fetal red cells

(C) Fetal anti-D IgG crossing into the maternal circulation

(D) Maternal anti-A IgM crossing the placenta and lysing fetal red cells

Q11. The normal platelet count in an adult is 150,000 to 400,000 per microlitre. Below which count does spontaneous bleeding, not provoked by trauma or surgery, usually begin?

(A) 100,000 per microlitre

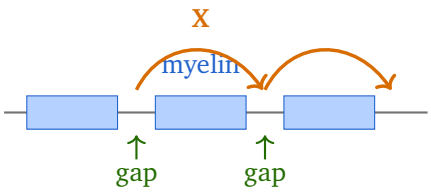
(B) 20,000 per microlitre

(C) 150,000 per microlitre

(D) 400,000 per microlitre

Nerve, Muscle and Endocrine Physiology

Q12. The figure shows a myelinated axon. The curved arrows marked **X** show the impulse skipping the internodes and reappearing only at the gaps. This mode of conduction, and the reason for it, are:



(A) Saltatory conduction, because voltage-gated sodium channels are concentrated at the nodes of Ranvier while myelin insulates the internodes

(B) Continuous conduction, because the myelin sheath itself conducts the current along the axon

(C) Saltatory conduction, because the myelin sheath carries the highest density of voltage-gated sodium channels

(D) Continuous conduction, because the internodal membrane has a very low electrical resistance

Q13. Compared with white (fast glycolytic, type II) fibres, red (slow oxidative, type I) skeletal muscle fibres are best described as:

(A) Poor in myoglobin and mitochondria, dependent on anaerobic glycolysis, and quick to fatigue

(B) Rich in myoglobin and mitochondria, dependent on oxidative phosphorylation, and resistant to fatigue

(C) Rich in myoglobin but poor in capillaries, contracting rapidly and fatiguing rapidly

(D) Poor in myoglobin but rich in mitochondria, contracting rapidly and resistant to fatigue

Q14. Thyroid hormones are made by iodinating tyrosine residues on thyroglobulin inside the follicle. Regarding T3 and T4, the correct statement is:

(A) T3 is the main hormone secreted by the gland, and T4 is the more biologically active form

(B) T4 is the main hormone secreted by the gland, and T3, formed largely by peripheral 5'-deiodination of T4, is the more biologically active form

(C) T3 and T4 are secreted in equal amounts and are equally active at the nuclear receptor

(D) T4 is the active hormone at the nuclear receptor, and T3 is an inactive degradation product

Detailed Solutions

Q1.

Solution

Concept – vitamin B12 needs a carrier protein to be absorbed: B12 is the only vitamin that cannot be absorbed unless the stomach supplies a specific transport protein.

Step 1 – name the carrier: Gastric parietal cells secrete **intrinsic factor**, and it binds vitamin B12 in the small intestine.

Step 2 – follow the absorption route: The B12-intrinsic factor complex travels down the gut and is taken up by cubilin receptors in the **terminal ileum**. No intrinsic factor means no uptake, however much B12 the diet contains.

Step 3 – explain the autoimmune lesion: Autoantibodies against parietal cells and against intrinsic factor destroy the source of the carrier. This is **pernicious anaemia**, a chronic atrophic gastritis.

Step 4 – explain the macrocytic anaemia: B12 is needed by methionine synthase, which regenerates tetrahydrofolate. Without it folate is trapped as methyltetrahydrofolate, DNA synthesis stalls, and the marrow releases large immature megaloblastic red cells.

Step 5 – explain the neurology: B12 is also the coenzyme of methylmalonyl-CoA mutase. Its loss lets methylmalonyl-CoA build up and damage myelin, giving the tingling feet of subacute combined degeneration.

Step 6 – read the oral sign: The smooth beefy red tongue is atrophic glossitis, a classic B12 finding a dentist sees first.

Why the other options are wrong:

- A, folic acid: also causes a macrocytic anaemia and glossitis, but folate absorption in the jejunum does not need intrinsic factor, and folate deficiency does **not** cause the neurological signs described.
- B, vitamin B6: deficiency gives a **microcytic** sideroblastic anaemia with seborrhoeic dermatitis and convulsions, and its absorption is intrinsic factor independent.
- D, vitamin B1: deficiency causes beriberi and Wernicke encephalopathy, not a macrocytic anaemia, and it needs no carrier protein.

Final Answer: Vitamin B12 $\Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – vitamin D is a prohormone activated in two hydroxylation steps: The vitamin made in skin or eaten in food is inert. Two separate organs must each add one hydroxyl group before it works.

Step 1 – the skin step: Ultraviolet B light converts 7-dehydrocholesterol in the skin into **cholecalciferol (vitamin D3)**. This is a photochemical ring opening, not a hydroxylation.

Step 2 – the first hydroxylation: In the **liver**, 25-hydroxylase adds a hydroxyl at carbon 25, giving **25-hydroxycholecalciferol (calcidiol)**. This is the main circulating form and the one measured to assess vitamin D status, but it is still only weakly active.

Step 3 – the second hydroxylation, which is step X: In the **kidney**, **1-alpha-hydroxylase** in the proximal tubule adds a hydroxyl at carbon 1, giving **1,25-dihydroxycholecalciferol (calcitriol)**, the fully active hormone.

Step 4 – match X to the diagram: The arrow marked X runs from 25-OH-cholecalciferol to calcitriol, so X is the renal 1-alpha-hydroxylation. The enzyme is 1-alpha-hydroxylase and the organ is the kidney.

Step 5 – note the control: 1-alpha-hydroxylase is the regulated step. Parathyroid hormone and a low plasma phosphate switch it on; calcitriol itself switches it off. This is why chronic kidney disease causes calcitriol deficiency and renal bone disease even when sunlight exposure is normal.

Why the other options are wrong:

- A, 25-hydroxylase in the liver: is the **earlier** step, the one that makes the substrate of X, not X itself.
- B, 7-dehydrocholesterol reductase in the skin: acts in cholesterol synthesis and is not part of this activation chain at all; the skin step here is driven by UV light.
- D, 24-hydroxylase in the kidney: is the **inactivating** enzyme. It makes 24,25-dihydroxy-D3, which is the route of disposal, not of activation.

Final Answer: 1-alpha-hydroxylase, in the kidney $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – isoenzymes catalyse the same reaction but differ in tissue of origin: Because each tissue carries its own mixture, the isoenzyme found in blood tells you which organ was damaged.

Step 1 – recall the structure of creatine kinase: CK is a dimer built from two subunit types, M (muscle) and B (brain). Three combinations exist: CK-BB, CK-MB and CK-MM.

Step 2 – assign each to its tissue: CK-BB is from brain, CK-MM is from skeletal muscle, and **CK-MB** is the form enriched in cardiac muscle.

Step 3 – fit the time course: After a myocardial infarction CK-MB starts rising at about 4 to 6 hours, peaks at about 24 hours and returns to normal by 48 to 72 hours. At 4 hours it is the isoenzyme the lab can already use.

Step 4 – confirm against the question: The question asks for the **creatine kinase** isoenzyme that is most cardiac specific and rises early. Only CK-MB satisfies both.

Step 5 – add the clinical footnote: Troponin I and T are more specific still and are the modern first-line markers, but among the CK isoenzymes offered here CK-MB is the answer, and its rapid fall makes it useful for detecting reinfarction.

Why the other options are wrong:

- A, LDH-5: is the **liver and skeletal muscle** isoenzyme. The cardiac LDH isoenzyme is LDH-1, and even that is a **late** marker peaking at 2 to 3 days, so it fails both parts of the question.
- B, CK-BB: comes from **brain** and smooth muscle, so it is not cardiac specific.
- C, CK-MM: is the dominant form in **skeletal** muscle. It rises after any intramuscular injection, trauma or heavy exercise, so it cannot localise damage to the heart.

Final Answer: CK-MB $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q3

Q4.

Solution

Concept – the six IUBMB classes are named after what the reaction does: Read the equation, ask what moved, and the class follows.

Step 1 – list the six classes: 1 oxidoreductases (transfer electrons), 2 transferases

(transfer a group), 3 hydrolases (split a bond using water), 4 lyases (split a bond without water, often making a double bond), 5 isomerases (rearrange within one molecule), 6 ligases (join two molecules using ATP).

Step 2 – read the given equation: glucose + ATP → glucose-6-phosphate + ADP.

Step 3 – ask what actually moved: A **phosphate group** moved from ATP onto glucose. Nothing was oxidised, nothing was hydrolysed, and no new molecule was joined together.

Step 4 – assign the class: Group transfer from a donor to an acceptor is the definition of a **transferase**. Hexokinase is a phosphotransferase, and every kinase belongs here.

Step 5 – generalise the rule: The suffix helps. Kinase means transferase, dehydrogenase and oxidase mean oxidoreductase, -ase on a substrate name with water means hydrolase, synthase means lyase, and synthetase with ATP means ligase.

Why the other options are wrong:

- A, oxidoreductase: needs electrons or hydrogen to move, as with lactate dehydrogenase. Glucose is neither oxidised nor reduced here.
- C, hydrolase: needs water as the attacking molecule, as with amylase or trypsin. No water appears in this equation.
- D, ligase: joins two separate molecules into one new covalent bond using ATP, as with DNA ligase or pyruvate carboxylase. Here ATP is only a phosphate donor, and glucose and ATP are not being fused together.

Final Answer: Transferase ⇒ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – gluconeogenesis is not simply glycolysis run backwards: Three glycolytic steps are irreversible, so gluconeogenesis must go around each of them with its own enzyme.

Step 1 – list the bypasses: Pyruvate to phosphoenolpyruvate uses pyruvate carboxylase then PEP carboxykinase; fructose-1,6-bisphosphate to fructose-6-phosphate uses **fructose-1,6-bisphosphatase**; glucose-6-phosphate to glucose uses glucose-6-phosphatase.

Step 2 – read the marked step off the diagram: X runs from fructose-1,6-bisphosphate to fructose-6-phosphate.

Step 3 – name its enzyme: That is the **fructose-1,6-bisphosphatase** bypass, which simply removes the phosphate at carbon 1.

Step 4 – confirm it is rate limiting: Fructose-1,6-bisphosphatase is the enzyme that sets the pace of gluconeogenesis. It is inhibited by AMP and by fructose-2,6-bisphosphate, and it is activated by citrate.

Step 5 – see why that regulation makes sense: When the cell is short of energy, AMP is high and fructose-2,6-bisphosphate is high, so the bisphosphatase is switched off and glucose is burned rather than built. When glucagon rises in fasting, fructose-2,6-bisphosphate falls, the brake is released, and the liver exports glucose. The same signal that switches this enzyme off switches its glycolytic counterpart on, which stops the two pathways running at once as a futile cycle.

Why the other options are wrong:

- B, pyruvate carboxylase: is a genuine gluconeogenic bypass enzyme, but it works at the **first** arrow of the diagram, pyruvate to oxaloacetate, not at X.
- C, aldolase: is a shared reversible enzyme that splits or joins fructose-1,6-bisphosphate and the triose phosphates. It sits above X in the diagram and is not regulated.
- D, enolase: is another shared reversible enzyme, interconverting 2-phosphoglycerate and phosphoenolpyruvate. It is not a bypass step and is not rate limiting.

Final Answer: Fructose-1,6-bisphosphatase $\Rightarrow$ A

Answer: (A) Go Back to Q5

Q6.

Solution

Concept – glycogen storage diseases are named by the enzyme that is missing: Work out which reaction has failed from the pattern of hypoglycaemia, organ enlargement and lactate.

Step 1 – read the key clue: Glycogen structure is normal but the liver **cannot release free glucose**. So glycogen is being broken down to glucose-6-phosphate perfectly well, and the block is at the very last step.

Step 2 – name that last step: Glucose-6-phosphate is dephosphorylated to free glucose by **glucose-6-phosphatase** in the endoplasmic reticulum of liver and kidney.

Step 3 – name the disease: Its deficiency is **von Gierke disease, glycogen stor-**

age disease type I, an autosomal recessive defect.

Step 4 – explain the hypoglycaemia: Glucose-6-phosphate is charged and cannot cross the membrane. Since this same enzyme is the final step of **both** glycogenolysis and gluconeogenesis, neither pathway can deliver glucose to the blood, so the infant crashes within hours of a missed feed.

Step 5 – explain the lactic acidosis: Glucose-6-phosphate that cannot leave is pushed back through glycolysis to pyruvate and then to lactate, which floods the blood.

Step 6 – explain the uric acid and the lipids: Trapped glucose-6-phosphate is diverted into the HMP shunt, raising ribose-5-phosphate and purine synthesis, so uric acid rises. Lactate also competes with urate for renal excretion, worsening it. Excess acetyl-CoA and NADPH drive triglyceride and cholesterol synthesis, giving the hyperlipidaemia.

Step 7 – explain the hepatomegaly: Glycogen that can never be exported keeps accumulating, giving the enormous liver and the doll-like face of a fat-cheeked, thin-limbed infant.

Why the other options are wrong:

- B, lysosomal acid alpha-glucosidase: is Pompe disease, type II. Its main problem is **cardiomegaly** with a floppy infant, and blood glucose is normal because cytosolic glycogenolysis is untouched.
- C, muscle glycogen phosphorylase: is McArdle disease, type V. It causes exercise intolerance and muscle cramps with myoglobinuria, but the liver and the blood glucose are **normal**.
- D, debranching enzyme: is Cori disease, type III. It does cause hepatomegaly and hypoglycaemia, but the stored glycogen is **abnormal**, with short outer branches (limit dextrin), and lactate and urate are typically normal. The question states the glycogen structure is normal, which excludes it.

Final Answer: Glucose-6-phosphatase $\Rightarrow$ A

Answer: (A) Go Back to Q6

Q7.

Solution

Concept – the committed step of cholesterol synthesis is where the drug acts: A rate limiting enzyme is always the useful drug target, because inhibiting it throttles the whole pathway.

Step 1 – trace the early pathway: Two acetyl-CoA condense to acetoacetyl-CoA. A third acetyl-CoA is added by HMG-CoA synthase to give HMG-CoA. All of this happens in the cytosol.

Step 2 – take the next reaction: HMG-CoA is reduced to **mevalonate** by **HMG-CoA reductase**, using 2 NADPH. The enzyme is embedded in the endoplasmic reticulum membrane.

Step 3 – confirm it is rate limiting: This is the committed and rate limiting step of cholesterol synthesis. Mevalonate has no other fate, so once it is made the cell is committed.

Step 4 – match the substrate clue: The question says the enzyme converts HMG-CoA into mevalonate. That is HMG-CoA reductase by definition.

Step 5 – link to the statins: Statins resemble HMG-CoA and bind the active site **competitively**. Less cholesterol is made in hepatocytes, so the liver upregulates its LDL receptors, pulls more LDL out of the plasma, and plasma LDL falls.

Step 6 – note the natural regulation: The same enzyme is inhibited by cholesterol itself and by glucagon (through phosphorylation), and it is activated by insulin. Its transcription is controlled by SREBP-2, which is why the drug effect and the feedback act on the same target.

Why the other options are wrong:

- A, HMG-CoA synthase: makes HMG-CoA, the **substrate** of the reductase. It is one step too early, and it is not the regulated step of this pathway.
- B, HMG-CoA lyase: is a **mitochondrial** enzyme of ketogenesis. It splits HMG-CoA into acetoacetate and acetyl-CoA, taking the molecule away from cholesterol synthesis entirely.
- D, squalene epoxidase: acts far **downstream**, oxidising squalene on the way to lanosterol. It is not rate limiting and it is not the statin target.

Final Answer: HMG-CoA reductase $\Rightarrow$ C

Answer: (C) Go Back to Q7

Q8.

Solution

Concept – read the ABG in a fixed order: pH first, then decide which value is driving it, then check the other for compensation.

Step 1 – read the pH: pH 7.52 is above 7.45, so this is an **alkalosis**.

Step 2 – decide which system is the culprit: $PaCO_2$ is 26 mmHg, which is low (normal 35 to 45). Carbon dioxide is an acid, so blowing it off raises the pH. A low CO_2 therefore **explains** the alkalosis, and the primary problem is **respiratory**.

Step 3 – check the other value against that: Bicarbonate is 22 mEq/L, at the low end of the 22 to 26 range. A low bicarbonate would push the pH *down*, so it opposes the alkalosis rather than causing it. That is early renal compensation, which takes days to develop fully.

Step 4 – name the diagnosis: High pH plus low $PaCO_2$ is **respiratory alkalosis**.

Step 5 – fit the clinical story: Anxiety-driven hyperventilation is the commonest cause seen in a dental chair. Others are fever, pain, high altitude, salicylate poisoning and pulmonary embolism.

Step 6 – explain the tingling: Alkalosis makes plasma proteins more negatively charged, so more calcium binds to albumin and the **ionised** calcium falls. That is why the patient gets circumoral numbness, tingling fingers and, if it continues, carpopedal spasm, even though total calcium is normal. Rebreathing raises the $PaCO_2$ and settles it.

Why the other options are wrong:

- A, metabolic alkalosis: also raises the pH, but it needs a **high** bicarbonate, above 26, as after vomiting. Here bicarbonate is 22, which is not high.
- B, respiratory acidosis: requires a **high** $PaCO_2$ and a low pH. Both values here are the opposite.
- C, metabolic acidosis: requires a **low** pH with a low bicarbonate. The pH here is 7.52, which is alkalaemic, so it is excluded at Step 1.

Final Answer: Respiratory alkalosis $\Rightarrow$ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – the bicarbonate buffer sets plasma pH through a ratio, not through absolute amounts: The Henderson-Hasselbalch equation converts that ratio into a pH.

Step 1 – write the equation: $\mathrm{pH} = \mathrm{p}K_\mathrm{a} + \log_{10} \dfrac{[\mathrm{HCO_3^-}]}{[\mathrm{H_2CO_3}]}$

Step 2 – put in the numbers given: $\mathrm{pH} = 6.1 + \log_{10} \dfrac{24}{1.2}$

Step 3 – work out the ratio: $\dfrac{24}{1.2} = 20$

Step 4 – take the logarithm: $\log_{10} 20 = 1.3$

Step 5 – add: $\mathrm{pH} = 6.1 + 1.3$

Step 6 – state the result: $\mathrm{pH} = 7.4$

Step 7 – read the physiology back: The healthy 20:1 ratio is what holds plasma at 7.4. The numerator, bicarbonate, is controlled by the **kidney**; the denominator, dissolved carbon dioxide, is controlled by the **lung**. Because pH depends on the ratio, a fall in bicarbonate can be offset by a matching fall in carbon dioxide, which is exactly how compensation works.

Step 8 – note why this buffer matters despite a poor pKa: A pKa of 6.1 is 1.3 units away from 7.4, so on paper it is a weak buffer at blood pH. It is nonetheless the most important extracellular buffer because both of its components are open systems, continuously adjusted by lung and kidney.

Why the other options are wrong:

- A, 6.1: is the **pKa** itself. It is the pH you would get only if the ratio were 1:1, since $\log_{10} 1 = 0$.
- B, 6.8: would need a ratio of about 5:1, and it is the pKa of the phosphate buffer, not this calculation.
- C, 7.1: would need a ratio of 10:1, that is $6.1 + 1.0$. The given ratio is 20:1, not 10:1.

Final Answer: 7.4 $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – only IgG crosses the placenta: This one fact decides most Rh incompatibility questions.

Step 1 – set up the sensitisation: An Rh negative mother has no D antigen. At the first delivery, fetal Rh positive red cells leak into her circulation and she mounts a primary immune response.

Step 2 – follow the antibody class switch: The primary response makes **IgM**, which is too large to cross the placenta, so the first baby escapes. Memory B cells then switch to producing **anti-D IgG**.

Step 3 – apply it to the second pregnancy: In the next Rh positive pregnancy, a small antigen exposure triggers a brisk secondary response. Maternal **anti-D IgG** crosses the placenta by FcRn-mediated active transport.

Step 4 – explain the haemolysis: The IgG coats fetal red cells, which are then destroyed extravascularly by splenic macrophages. This is **erythroblastosis fetalis**, or haemolytic disease of the newborn.

Step 5 – explain the name: The anaemic fetal marrow responds by pushing out immature nucleated red cells, erythroblasts, into the blood. Severe cases give hydrops fetalis, and after birth the unconjugated bilirubin load can cross into the basal ganglia and cause kernicterus.

Step 6 – note the prevention: Anti-D immunoglobulin given to the mother within 72 hours of delivery mops up the fetal cells before she can be sensitised, which is why the disease is now uncommon.

Why the other options are wrong:

- B, maternal anti-D IgM: IgM is a pentamer of about 900 kDa and **cannot** cross the placenta. This is precisely why the first Rh positive baby is spared.
- C, fetal anti-D IgG: the fetus is Rh **positive**, so it has the D antigen and cannot make antibodies against its own red cells. The direction of transfer is also wrong.
- D, maternal anti-A IgM: ABO incompatibility is a real cause of neonatal jaundice, but naturally occurring anti-A and anti-B are IgM and do not cross the placenta. The IgG anti-A of group O mothers can cross, yet it gives a mild disease, and in any case the question describes Rh sensitisation, not ABO.

Final Answer: Maternal anti-D IgG crossing the placenta $\Rightarrow$ A

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – bleeding risk falls in steps as the platelet count falls: Thrombocytopenia is not one threshold but a ladder, and the question asks for the rung at which bleeding starts without any injury.

Step 1 – fix the normal range: 150,000 to 400,000 per microlitre, as the question states.

Step 2 – take the first rung: Between 50,000 and 150,000 the patient is thrombocytopenic on paper but bleeds normally in daily life. Minor surgery is generally safe above 50,000.

Step 3 – take the second rung: Between 20,000 and 50,000 the patient bleeds **after** trauma, extraction or surgery, but not on their own.

Step 4 – take the third rung, which the question asks for: Below about **20,000 per microlitre** the patient starts bleeding **spontaneously**: petechiae, purpura, gingival oozing and epistaxis with no provocation. Below 10,000 the risk of serious internal or intracranial bleeding rises sharply and prophylactic platelet transfusion is considered.

Step 5 – confirm against the wording: The question specifies bleeding that is **not** provoked by trauma or surgery, which is the 20,000 rung.

Step 6 – note the dental relevance: Spontaneous gingival bleeding in a patient with no periodontal cause should prompt a platelet count, because it may be the first sign of leukaemia, aplastic anaemia or immune thrombocytopenia.

Why the other options are wrong:

- A, 100,000: is well within the safe band. These patients bleed normally, and the count is only a caution before major surgery.
- C, 150,000: is the **lower limit of normal**, not a bleeding threshold at all.
- D, 400,000: is the **upper limit of normal**. Counts above it are thrombocytosis, which carries a thrombotic rather than a bleeding risk.

Final Answer: 20,000 per microlitre $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – myelin makes conduction fast by forcing the impulse to skip: An impulse is regenerated only where the membrane is exposed, so insulating most of the axon speeds it up.

Step 1 – name what the gaps are: The bare gaps between adjacent myelin segments are the **nodes of Ranvier**, about 1 micrometre long and spaced roughly 1 to 2 millimetres apart.

Step 2 – locate the sodium channels: Voltage-gated sodium channels are packed at very high density **at the nodes** and are almost absent under the myelin. An action potential can therefore only be regenerated at a node.

Step 3 – state what the myelin does: Myelin is a stack of Schwann cell or oligodendrocyte membranes. It raises the membrane resistance and lowers the membrane capacitance, so current leaks out far less along the internode.

Step 4 – follow the current: Sodium entering at one node spreads passively inside the axon as local current. Because the internode leaks so little, that current still reaches the next node above threshold.

Step 5 – name the mode: The impulse therefore appears to jump from node to node. This is **saltatory conduction**, from the Latin *saltare*, to leap, and it is exactly what the arrows marked X show.

Step 6 – state the payoff: Regenerating the potential at a few thousand nodes instead of continuously along every micrometre is both faster (up to about 120 m/s in a large myelinated fibre against about 1 m/s in an unmyelinated one) and cheaper, since far less sodium enters and the Na-K pump has less to undo.

Step 7 – link to disease: Demyelination, as in multiple sclerosis or Guillain-Barre syndrome, lets current leak across the exposed internode. It cannot reach the next node, so conduction slows or blocks completely.

Why the other options are wrong:

- B, continuous conduction because myelin conducts: myelin is a lipid **insulator**, not a conductor. Continuous conduction is what *unmyelinated* fibres do, and it is the slow option.
- C, saltatory but channels in the myelin: the mode is right but the reason is wrong. Sodium channels are concentrated at the **nodes**; the myelinated internode is nearly channel free, which is why no impulse is generated there.
- D, continuous with low internodal resistance: this reverses the physics. Myelin makes internodal resistance **high**, which is what preserves the local current, and the figure plainly shows skipping rather than continuous

spread.

Final Answer: Saltatory conduction, sodium channels clustered at the nodes of Ranvier $\Rightarrow$ A

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – fibre colour follows fibre fuel: Red fibres are red because they are built to use oxygen, and everything else about them follows from that.

Step 1 – explain the colour: Red fibres are rich in **myoglobin**, the oxygen-binding pigment, and are surrounded by a dense capillary network. That pigment is what makes them look red.

Step 2 – link colour to machinery: Myoglobin is only useful if the fibre can burn the oxygen it holds, so red fibres also carry **many mitochondria** and high oxidative enzyme activity.

Step 3 – link machinery to fuel: They generate ATP by **oxidative phosphorylation**, largely from fatty acids. Oxidative ATP supply is steady and does not build up lactate.

Step 4 – link fuel to fatigue: Because ATP is resupplied as fast as it is used and no lactate accumulates, red fibres are **resistant to fatigue**. They also contract slowly, because they carry the slow myosin ATPase isoform.

Step 5 – contrast the white fibre: White (type II, fast glycolytic) fibres have little myoglobin, few mitochondria, few capillaries, plenty of glycogen and fast myosin ATPase. They rely on anaerobic glycolysis, contract rapidly and powerfully, and fatigue quickly as lactate builds up.

Step 6 – put it to use: Red fibres dominate postural muscles that must hold a load all day, such as the soleus. White fibres dominate muscles built for short bursts, such as the extraocular muscles. The masseter is mixed, which lets it both maintain postural tone and deliver a powerful bite.

Why the other options are wrong:

- A: describes the **white** fibre exactly. Poor myoglobin, anaerobic glycolysis and rapid fatigue are the type II profile, the opposite of what is asked.
- C: is internally inconsistent. A fibre rich in myoglobin is by definition built for oxygen, so it would not be poorly capillarised, and it would not fatigue rapidly.

- D: is also inconsistent. A fibre rich in mitochondria and fatigue resistant is oxidative, and an oxidative fibre is myoglobin **rich**, not myoglobin poor.

Final Answer: Rich in myoglobin and mitochondria, oxidative, fatigue resistant $\Rightarrow$ B

Answer: (B) Go Back to Q13

Q14.

Solution

Concept – the thyroid secretes mostly the weaker hormone and lets the tissues finish the job: Separate what the gland *releases* from what actually *acts* at the receptor.

Step 1 – get iodide into the follicle: The basal Na-I symporter traps iodide from the blood against a steep gradient, concentrating it 30 to 40 times inside the follicular cell.

Step 2 – oxidise and attach it: Thyroid peroxidase oxidises iodide and iodinates tyrosine residues on thyroglobulin, making monoiodotyrosine (MIT) and diiodotyrosine (DIT). No dietary iodine means no hormone, which is why iodine deficiency causes goitre and cretinism.

Step 3 – couple them: DIT + DIT gives **T4** (four iodines). MIT + DIT gives **T3** (three iodines). Coupling favours T4 heavily.

Step 4 – read the secretion ratio: The gland releases roughly 90 percent T4 and only about 10 percent T3. So **T4 is the main secreted hormone**.

Step 5 – follow T4 in the periphery: 5'-deiodinase in liver, kidney and other tissues removes one iodine from the outer ring of T4 to make **T3**. Most circulating T3 is made this way, not by the gland.

Step 6 – compare potency: T3 binds the nuclear thyroid hormone receptor with roughly 10 times the affinity of T4. So **T3 is the active hormone** and T4 behaves as a long-lived circulating prohormone and reservoir (half-life about 7 days, against about 1 day for T3).

Step 7 – combine: T4 is the main secreted form, T3 is the main active form, and peripheral deiodination is the bridge. That is option B.

Why the other options are wrong:

- A: has both halves reversed. It makes T3 the secreted form and T4 the potent one, whereas the gland secretes mostly T4 and T3 is the more potent.
- C: equal amounts is wrong (the ratio is about 9:1 in favour of T4) and equal

activity is wrong (T3 is roughly ten times more potent at the receptor).

- D: inverts the pharmacology. T3 is not a degradation product but the active hormone. The genuinely inactive product of deiodination is **reverse T3**, made when the inner ring is deiodinated instead, as happens in sick euthyroid syndrome.

Final Answer: T4 mainly secreted, T3 from peripheral deiodination is more active $\Rightarrow$ B

Answer: (B) Go Back to Q14

Answer Key

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