NEET MDS General Physiology and Biochemistry

Sample Paper – 9

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 vegetarian patient on iron tablets is advised to swallow them with a glass of lemon water rather than with tea. Vitamin C improves the absorption of non-haem iron mainly because it:

(A) Reduces dietary ferric (Fe^{3+}) iron to the ferrous (Fe^{2+}) form, which is the form the duodenal transporter DMT1 carries into the enterocyte

(B) Carries iron across the basolateral membrane of the enterocyte in place of ferroportin

(C) Raises the pH of the duodenal lumen so that ferric iron remains soluble

(D) Stimulates hepcidin release from the liver, which in turn opens ferroportin channels

Q2. A 2-year-old child with bowed legs and widened wrists and a 45-year-old woman with diffuse bone pain, proximal muscle weakness and pseudofractures are both found to be deficient in vitamin D. The child is labelled rickets and the woman osteomalacia. The best explanation for the two different pictures is:

(A) The child lacks a functioning vitamin D receptor whereas the adult has one

(B) Rickets is caused by calcium deficiency alone and osteomalacia by phosphate deficiency alone

(C) In the child the epiphyseal growth plates are still open, so unmineralised cartilage and osteoid pile up at the plates and give bowing and widened metaphyses, while in the adult the plates have fused and only newly remodelled osteoid fails to mineralise, giving bone pain and pseudofractures

(D) The adult absorbs no calcium at all from the gut whereas the child absorbs a normal amount

Enzymes and Enzyme Kinetics

Q3. The enzyme that works best at an acid pH, whose tartrate-resistant isoform is used as a marker of osteoclast activity in bone, and whose tartrate-inhibited isoform arises from the prostate, is:

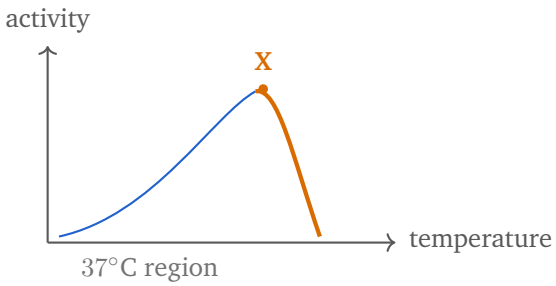
(A) Acid phosphatase

(B) Alkaline phosphatase

(C) Lactate dehydrogenase

(D) Gamma-glutamyl transferase

Q4. The curve below plots the activity of a human enzyme against temperature. Activity climbs to the peak marked **X** and then collapses steeply. The steep fall beyond **X** is because:



(A) The substrate is used up faster than it can be replaced at high temperature

(B) Heat above the optimum breaks the weak bonds holding the tertiary structure, so the enzyme denatures irreversibly and the shape of the active site is lost

(C) The enzyme molecules move too fast to collide with substrate above the optimum

(D) The K_m of the enzyme falls to zero above the optimum

Metabolism of Carbohydrate, Lipid and Protein

Q5. The pentose phosphate pathway runs in the cytosol alongside glycolysis, consumes no ATP and generates no ATP. The two things this pathway supplies to the cell are:

(A) ATP and NADH for oxidative phosphorylation

(B) Ribose-5-phosphate for nucleotide synthesis and NADPH for reductive biosynthesis

(C) Acetyl-CoA and $FADH_2$ for the TCA cycle

(D) Glycerol-3-phosphate and NADH for triacylglycerol synthesis

Q6. During translation on the ribosome, the molecule that reads a codon on the messenger RNA and delivers the matching amino acid to the growing chain is:

(A) Messenger RNA itself, which carries the anticodon

(B) The ribosomal RNA of the small subunit, which pairs its own anticodon with the codon

(C) Transfer RNA, which carries an anticodon complementary to the codon and the matching amino acid on its 3′ CCA end

(D) Aminoacyl-tRNA synthetase, which sits in the A site and base pairs with the codon

Q7. Amino acids are classified as glucogenic, ketogenic or both according to the fate of their carbon skeletons. The pair that is **purely ketogenic** is:

(A) Leucine and lysine

(B) Alanine and glutamine

(C) Isoleucine and phenylalanine

(D) Glycine and serine

Acid Base Balance and Body Fluids

Q8. A young adult has a persistent metabolic acidosis with a low plasma bicarbonate, a raised plasma chloride and a **normal** anion gap. Renal function is otherwise preserved, and the defect lies in the tubular handling of hydrogen ion and bicarbonate. The diagnosis is:

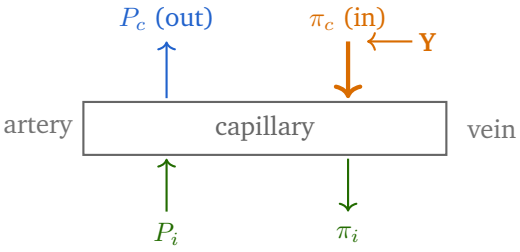
(A) Renal tubular acidosis

(B) Diabetic ketoacidosis

(C) Lactic acidosis

(D) Salicylate poisoning

Q9. The diagram shows a capillary with the four Starling forces arrowed. A child with nephrotic syndrome loses albumin in the urine and develops generalised oedema. In Starling terms the oedema arises because:



(A) The capillary hydrostatic pressure P_c rises because albumin loss raises the blood pressure inside the capillary

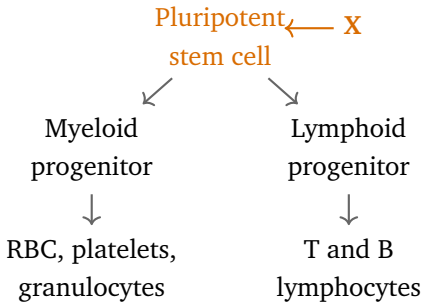
(B) Force **Y**, the plasma colloid osmotic pressure, falls with the albumin, so the inward pull opposing P_c is weakened and net filtration into the interstitium outstrips lymphatic drainage

(C) The interstitial colloid osmotic pressure π_i falls, which sucks fluid back out of the tissues

(D) The lymphatics are physically blocked by albumin casts

Blood and Haemostasis

Q10. In the scheme below, the cell marked **X** is the pluripotent haematopoietic stem cell from which every blood cell line descends. The order in which the **sites** of blood formation take over during fetal and post-natal life is:



(A) Bone marrow alone, from the early embryo right through adult life

(B) Liver first, then the yolk sac, then the spleen

(C) Thymus first, then the bone marrow, then the liver

(D) Yolk sac in early embryonic life, then the liver and spleen in mid fetal life, then the bone marrow from late fetal life onwards

Q11. The organ whose macrophages strip aged and abnormally shaped red cells out of the circulation as they squeeze through its cords and sinusoids, making it the chief site of extravascular haemolysis, is:

(A) Liver

(B) Spleen

(C) Bone marrow

(D) Thymus

Nerve, Muscle and Endocrine Physiology

Q12. A bolus in the mouth reflexly inhibits the muscles of mastication, the jaw drops, the drop stretches the jaw closing muscles, and the stretch reflex snaps the jaw shut again onto the bolus, restarting the cycle. This chewing reflex is organised in:

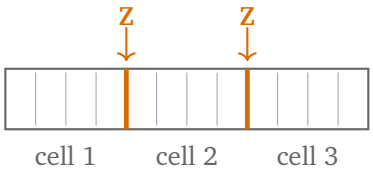
(A) The cerebellum

(B) The hypothalamus

(C) The cervical spinal cord

(D) The brainstem, in the reticular formation near the trigeminal motor nucleus

Q13. The diagram shows cardiac muscle fibres joined end to end at the structures marked **Z**. Which statement about cardiac muscle is correct?



(A) Its cells are electrically insulated from one another, so each fibre contracts on its own

(B) Its refractory period is very short, so it can be tetanised as readily as skeletal muscle

(C) Gap junctions in the intercalated discs let current pass from cell to cell so the muscle behaves as a functional syncytium, its long refractory period prevents tetanus, and its pacemaker cells give it automaticity

(D) It depends entirely on its nerve supply for rhythm and stops beating the moment it is denervated

Q14. The adrenal medulla develops from neural crest, its chromaffin cells are in effect modified postganglionic sympathetic neurons without axons,

and they are driven directly by preganglionic sympathetic fibres in the splanchnic nerves. The hormone that forms the bulk (about 80 percent) of its secretion is:

(A) Cortisol

(B) Aldosterone

(C) Acetylcholine

(D) Adrenaline

Detailed Solutions

Q1.

Solution

Concept – iron is only absorbed in one oxidation state: Dietary non-haem iron and the duodenal transporter that carries it do not accept the same form of iron, and vitamin C bridges that gap.

Step 1 – state the form iron arrives in: Most non-haem iron in food, especially plant food, is in the **ferric (Fe^{3+})** state.

Step 2 – state the form that can be absorbed: The divalent metal transporter **DMT1** on the brush border of the duodenal enterocyte carries only **ferrous (Fe^{2+})** iron.

Step 3 – identify the mismatch: So ferric iron in the lumen cannot be taken up as it stands. It must first be reduced by one electron.

Step 4 – bring in vitamin C: Ascorbic acid is a **reducing agent**. It donates the electron that converts Fe^{3+} to Fe^{2+}, working alongside the brush border ferrireductase.

Step 5 – add the second benefit: Ascorbate also chelates the ferrous iron and keeps it soluble in the alkaline duodenum, so it does not precipitate as insoluble hydroxide before it reaches DMT1.

Step 6 – close the clinical loop: That is exactly why iron tablets are given with citrus juice and not with tea, since the tannates in tea do the opposite and bind iron into an unabsorbable complex.

Why the other options are wrong:

- B, basolateral transport: iron leaves the enterocyte through **ferroportin**, and vitamin C plays no transporter role there. Vitamin C acts in the lumen and at the brush border.
- C, raising luminal pH: an **acid** environment favours iron absorption, which is why gastric acid helps and why proton pump inhibitors hinder it. Raising the pH would make things worse, and ascorbic acid is an acid in any case.
- D, hepcidin: hepcidin is the negative regulator of iron. It **degrades** ferroportin and reduces iron entry into blood. Stimulating hepcidin would block absorption, not enhance it, and ferroportin is a transporter and not a channel opened by hepcidin.

Final Answer: It reduces ferric iron to the absorbable ferrous form $\Rightarrow$ A

Answer: (A) Go Back to Q1

Q2.

Solution

Concept – one vitamin, one biochemical defect, two skeletons: Rickets and osteomalacia are the same disease of defective mineralisation. The age of the skeleton decides how it looks.

Step 1 – state the shared defect: Vitamin D deficiency lowers calcium and phosphate absorption from the gut, so the calcium-phosphate product falls and newly laid osteoid cannot be mineralised.

Step 2 – look at the growing skeleton: In a child the **epiphyseal growth plates are still open** and are actively laying down cartilage that must be calcified before it is replaced by bone.

Step 3 – state what fails there: That cartilage is not calcified, so it piles up. The plate widens, the metaphysis flares, and the soft under-mineralised shaft bends under weight.

Step 4 – name those signs: Bowed legs, widened wrists and ankles, the rachitic rosary at the costochondral junctions, frontal bossing and delayed fontanelle closure. Delayed tooth eruption and enamel hypoplasia belong here too, which is the dental relevance.

Step 5 – look at the adult skeleton: In an adult the growth plates have **fused**, so there is no growing cartilage to affect. Only the osteoid laid down during normal remodelling can go unmineralised.

Step 6 – state what that produces: Wide unmineralised osteoid seams on otherwise normal bone give diffuse bone pain, tenderness, proximal myopathy with a waddling gait, and Looser zones or pseudofractures.

Step 7 – put the two together: Same deficiency, same failure to mineralise. Open plates give rickets, fused plates give osteomalacia, which is option C.

Why the other options are wrong:

- A, receptor defect: hereditary vitamin D resistant rickets from a faulty receptor is a rare separate disease. It does not explain ordinary nutritional deficiency, which is what both patients have.
- B, calcium versus phosphate: both conditions come from the **same** vitamin D deficiency and both involve low calcium and low phosphate. Splitting them by mineral is simply wrong.
- D, no absorption in the adult: the adult absorbs less calcium, not none, and the amount absorbed is not what separates the two pictures. The state of the growth plate is.

Final Answer: Open growth plates in the child, fused plates in the adult $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – name the enzyme from its optimum pH and its isoforms: The question gives three clues: an acid optimum, an osteoclast isoform resistant to tartrate, and a prostatic isoform inhibited by tartrate. All three point to one enzyme.

Step 1 – use the pH clue: An enzyme that hydrolyses phosphate esters best at pH around 5 is **acid phosphatase**.

Step 2 – use the osteoclast clue: Osteoclasts secrete **tartrate-resistant acid phosphatase (TRAP)** into the resorption lacuna under the ruffled border. Its activity in serum or in a bone section marks how much resorption is going on, which matters in Paget disease, in metastatic bone lesions and in periodontal bone loss.

Step 3 – use the prostate clue: Prostatic acid phosphatase is the isoform that **is** inhibited by tartrate. It rises in carcinoma of the prostate, especially once the tumour has spread to bone, and it was the classic marker before PSA replaced it.

Step 4 – see how tartrate separates the two: Add tartrate to the sample. The prostatic isoform is switched off, and whatever activity survives is the bone or osteoclast isoform. That single reagent is what tells the two sources apart.

Step 5 – confirm: Acid optimum plus a tartrate-resistant osteoclast form plus a tartrate-sensitive prostatic form fits acid phosphatase exactly.

Why the other options are wrong:

- B, alkaline phosphatase: works best at an **alkaline** pH near 9 to 10. It is a marker of osteoblastic bone **formation** and of cholestasis, not of osteoclastic resorption, and it has no tartrate-resistant prostatic story.
- C, lactate dehydrogenase: is a cytosolic enzyme of anaerobic glycolysis with five isoenzymes separated by **electrophoresis**, not by tartrate. It has no acid optimum and no bone-specific form.
- D, gamma-glutamyl transferase: is a membrane enzyme of the biliary tree used to confirm that a raised alkaline phosphatase is hepatic in origin. It is unrelated to osteoclasts or to the prostate.

Final Answer: Acid phosphatase $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – the temperature curve has two limbs and two different reasons: Enzyme activity against temperature is a bell that climbs gently and falls sharply. The rise and the fall are governed by two separate physical facts.

Step 1 – read the rising limb: Up to the peak, heating adds kinetic energy. Enzyme and substrate collide more often and more of those collisions carry the activation energy, so velocity rises.

Step 2 – read the peak: The peak is the **optimum temperature**. For most human enzymes it sits close to normal body temperature, near 37°C, which is what the label on the axis marks.

Step 3 – read the falling limb: Beyond the optimum, activity does not tail off gently. It collapses over a few degrees, which is far steeper than the rise. A steep collapse means the enzyme molecules themselves are being destroyed, not merely slowed.

Step 4 – give the mechanism: The tertiary and quaternary structure of a protein is held together by weak forces: hydrogen bonds, ionic bonds, hydrophobic interactions and van der Waals contacts. Heat energy shakes those weak bonds apart.

Step 5 – state the consequence: The chain unfolds, the precisely shaped **active site** is lost, substrate can no longer bind, and activity disappears. This is **denaturation**.

Step 6 – note that it is irreversible: Cooling the sample back to 37°C does not restore activity, because the unfolded chains aggregate rather than refold. Note that the peptide bonds are untouched, so the primary structure survives. Only the folding is lost.

Step 7 – apply it: This is why a fever above about 41°C is dangerous, and why autoclaving at 121°C sterilises by denaturing microbial proteins.

Why the other options are wrong:

- A, substrate exhaustion: initial velocity is measured at a fixed saturating substrate concentration, so substrate is not limiting. Exhaustion would also affect both limbs equally, not just the region beyond the optimum.
- C, molecules moving too fast to collide: faster movement means **more** collisions, not fewer. This reverses the kinetic theory that explains the rising limb.
- D, K_m falling to zero: a K_m of zero would mean infinitely tight binding and maximal activity at any substrate concentration. That is the opposite of what

the graph shows, and K_m is meaningless once the enzyme is denatured.

Final Answer: Irreversible denaturation destroys the active site above the optimum $\Rightarrow$ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – the pentose phosphate pathway is a biosynthetic pathway, not an energy pathway: Glucose-6-phosphate can go down glycolysis for energy or down this pathway for building blocks. The question asks what the pathway hands the cell.

Step 1 – note what it does not make: The question itself says the pathway consumes no ATP and generates no ATP. So its purpose cannot be energy.

Step 2 – follow the oxidative phase: Glucose-6-phosphate is oxidised and decarboxylated to ribulose-5-phosphate. Two dehydrogenase steps in this phase each reduce $NADP^+$, so the phase yields **2 NADPH** and one CO_2 per glucose-6-phosphate.

Step 3 – follow the non-oxidative phase: Ribulose-5-phosphate is isomerised to **ribose-5-phosphate**. Transketolase and transaldolase can then reshuffle the sugars back into fructose-6-phosphate and glyceraldehyde-3-phosphate, so the cell takes only as much ribose as it needs.

Step 4 – state the use of ribose-5-phosphate: It is the sugar backbone of **nucleotides**, so it goes into purines, pyrimidines, DNA, RNA, ATP, NAD, FAD and coenzyme A. Demand is highest in dividing tissue such as marrow and oral epithelium.

Step 5 – state the use of NADPH: NADPH is the cell's **reducing power for synthesis**, distinct from NADH which is for energy. It drives fatty acid and cholesterol synthesis, steroid hormone synthesis in the adrenal cortex and gonads, the respiratory burst in neutrophils through NADPH oxidase, and it keeps glutathione reduced so that peroxides are cleared.

Step 6 – combine: Ribose-5-phosphate for nucleotides plus NADPH for reductive biosynthesis is exactly option B.

Why the other options are wrong:

- A, ATP and NADH: those are the currencies of **glycolysis** and the TCA cycle. The stem rules ATP out explicitly, and this pathway reduces $NADP^+$, not

NAD^+.

- C, acetyl-CoA and $FADH_2$: acetyl-CoA comes from the link reaction and from beta-oxidation, and $FADH_2$ from the TCA cycle and beta-oxidation. Neither is a product of this pathway.
- D, glycerol-3-phosphate and NADH: glycerol-3-phosphate comes from the dihydroxyacetone phosphate of glycolysis. The pathway does supply NADPH for fat synthesis, but the pair as written is wrong on both counts.

Final Answer: Ribose-5-phosphate and NADPH $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – translation needs an adaptor between nucleic acid language and protein language: A codon is three bases. An amino acid has no way of recognising three bases by itself, so something must read the codon at one end and hold the amino acid at the other.

Step 1 – name the adaptor: That molecule is **transfer RNA (tRNA)**, a small clover-leaf RNA of about 75 to 90 nucleotides that folds into an L shape.

Step 2 – describe its reading end: One arm of the clover leaf carries the **anticodon**, three bases complementary and antiparallel to the mRNA codon. Base pairing between codon and anticodon in the ribosomal A site is what selects the right tRNA.

Step 3 – describe its carrying end: The other end is the 3′ **CCA** tail, to which the amino acid is esterified.

Step 4 – state who loads it: Aminoacyl-tRNA synthetase charges the tRNA, using ATP and matching each amino acid to its own tRNA. There is one synthetase per amino acid, and its proofreading is the main guarantee of accuracy.

Step 5 – place it on the ribosome: The charged tRNA enters the **A site**, the peptidyl transferase activity of the large subunit forms the peptide bond, the tRNA shifts to the **P site**, and the uncharged tRNA leaves from the **E site**.

Step 6 – confirm against the question: The question asks for the molecule that reads the codon **and** delivers the amino acid. Only tRNA does both jobs, which is option C.

Why the other options are wrong:

- A, mRNA: carries the **codons**, not the anticodon. It is the message being

read, not the reader.

- B, ribosomal RNA: the small subunit rRNA positions the mRNA and the large subunit rRNA catalyses the peptide bond, but rRNA has no anticodon and carries no amino acid.
- D, aminoacyl-tRNA synthetase: is an **enzyme** that charges tRNA in the cytosol. It does not sit in the A site and it does not base pair with the codon.

Final Answer: Transfer RNA $\Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – classify an amino acid by where its carbon skeleton lands: Strip off the nitrogen and follow the carbons. Where they enter metabolism decides the label.

Step 1 – define glucogenic: If the skeleton yields pyruvate or a TCA intermediate such as alpha-ketoglutarate, succinyl-CoA, fumarate or oxaloacetate, it can be built back into glucose. That amino acid is **glucogenic**.

Step 2 – define ketogenic: If the skeleton yields **acetyl-CoA** or acetoacetyl-CoA, it cannot become glucose, because the pyruvate dehydrogenase step is irreversible and the two carbons of acetyl-CoA are lost as CO_2 in the TCA cycle. Such carbons can only go to ketone bodies or to fat. That amino acid is **ketogenic**.

Step 3 – find the purely ketogenic ones: Only two of the twenty standard amino acids give acetyl-CoA or acetoacetate and nothing else: **leucine** and **lysine**. Both L's, which is the usual memory hook.

Step 4 – list the ones that are both: Isoleucine, phenylalanine, tyrosine, tryptophan and threonine are **both** glucogenic and ketogenic, because their skeletons split and one part goes each way.

Step 5 – note the rest: Every remaining amino acid is purely glucogenic.

Step 6 – apply the clinical point: In prolonged starvation the glucogenic amino acids feed gluconeogenesis and hold the blood glucose up, while leucine and lysine can only add to the ketone load. That is why option A is the answer.

Why the other options are wrong:

- B, alanine and glutamine: both are purely **glucogenic**. Alanine gives pyruvate through the glucose-alanine cycle and glutamine gives alpha-ketoglutarate. These are in fact the two main nitrogen carriers from muscle.

- C, isoleucine and phenylalanine: both are **glucogenic and ketogenic**, not purely ketogenic, so the pair fails the word "purely".
- D, glycine and serine: both are purely **glucogenic**, converging on pyruvate through serine.

Final Answer: Leucine and lysine $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – a metabolic acidosis with a normal anion gap is a short list: Once the gap is normal, the acid was not added from outside. Bicarbonate was lost, and the loss is either from the gut or from the kidney.

Step 1 – read the acid base picture: Low pH with a low bicarbonate is a **metabolic acidosis**.

Step 2 – read the gap: The anion gap is **normal**, and the chloride is raised to fill the space the lost bicarbonate left behind. This is a hyperchloraemic acidosis.

Step 3 – understand what that rules out: A normal gap means no extra unmeasured anion is present. So there is no ketoacid, no lactate, no salicylate and no retained sulphate or phosphate.

Step 4 – narrow to the source: Bicarbonate has therefore simply been lost or has failed to be regenerated. The two routes are the gut, as in severe diarrhoea or a ureteric diversion, and the kidney.

Step 5 – use the tubular clue: The stem states that renal function is otherwise preserved, meaning the glomerular filtration rate is normal, and that the defect lies in tubular handling of hydrogen ion and bicarbonate. That is the definition of **renal tubular acidosis (RTA)**.

Step 6 – name the two common types: Distal (type 1) RTA is a failure of the alpha-intercalated cell to secrete H^+, so urine pH stays above 5.5, and stones, nephrocalcinosis and hypokalaemia follow. Proximal (type 2) RTA is a failure to reabsorb filtered bicarbonate, so bicarbonate is wasted until the plasma level drops low enough for the remaining capacity to cope.

Step 7 – add the dental angle: Chronic acidosis is buffered by bone, which drives calcium out of the skeleton, and type 1 RTA has a recognised association with enamel defects and delayed eruption, so the dental surgeon does meet these patients.

Why the other options are wrong:

- B, diabetic ketoacidosis: acetoacetate and beta-hydroxybutyrate are unmeasured anions, so the gap is **raised**, not normal, and the patient would be hyperglycaemic with ketones.
- C, lactic acidosis: lactate is itself the unmeasured anion, so it too gives a **high** gap, typically after shock, hypoxia or sepsis. There is no tubular defect.
- D, salicylate poisoning: gives a **high** gap metabolic acidosis, classically mixed with a respiratory alkalosis from direct stimulation of the respiratory centre. Again the gap is wrong and the mechanism is a poisoning, not a tubule.

Final Answer: Renal tubular acidosis $\Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – fluid across a capillary wall is settled by four pressures: Two pressures push fluid out, two hold it in, and oedema appears when the balance tips outward beyond what the lymphatics can carry away.

Step 1 – name the four Starling forces from the diagram: P_c, the capillary hydrostatic pressure, pushes fluid **out**. π_c, marked **Y**, the plasma colloid osmotic pressure, pulls fluid **in**. P_i, the interstitial hydrostatic pressure, pushes **in**. π_i, the interstitial colloid osmotic pressure, pulls **out**.

Step 2 – write the net filtration: Net filtration pressure $= (P_c - P_i) - (\pi_c - \pi_i)$.

Step 3 – state who generates π_c: The plasma proteins hold water in the capillary, and **albumin** supplies roughly 75 to 80 percent of that pull because it is small and by far the most numerous protein molecule.

Step 4 – apply nephrotic syndrome: The damaged glomerular filtration barrier leaks albumin into the urine. Plasma albumin falls, so π_c **falls**.

Step 5 – put the fall back into the equation: A smaller π_c makes the subtracted term smaller, so the net filtration pressure rises. More fluid leaves the capillary all along its length, and even the venous end stops reabsorbing.

Step 6 – explain why it becomes visible: The lymphatics take up the first part of the extra load, which is the safety factor. Once filtration outruns lymph drainage, fluid collects in the interstitium and pitting oedema appears, typically periorbital in the morning in a nephrotic child.

Step 7 – note the second hit: The falling plasma volume also triggers renin,

angiotensin and aldosterone, so sodium and water are retained and the oedema worsens. That is why option B is correct.

Why the other options are wrong:

- A, a rise in P_c: albumin loss does not raise capillary hydrostatic pressure. If anything the plasma volume falls. A raised P_c is the mechanism of oedema in heart failure and venous obstruction, not in hypoalbuminaemia.
- C, a fall in π_i: π_i is an **outward** force. If it fell, less fluid would be filtered and oedema would improve. The option also reverses the direction of the force.
- D, lymphatic obstruction: blocked lymphatics do cause oedema, but that is lymphoedema, as in filariasis or after node clearance. Albumin does not form casts that block lymphatics, and the mechanism here is the loss of π_c.

Final Answer: Force Y, the plasma colloid osmotic pressure, falls $\Rightarrow$ B

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – one stem cell, but a moving address: Every blood cell descends from the pluripotent haematopoietic stem cell shown as X, yet the tissue that houses those stem cells changes three times before birth is over.

Step 1 – read the diagram: X is self-renewing and pluripotent. It commits either to the myeloid progenitor, which gives red cells, platelets, granulocytes and monocytes, or to the lymphoid progenitor, which gives T and B lymphocytes and NK cells.

Step 2 – take the first site, the mesoblastic phase: From about the third week the **yolk sac** blood islands make primitive nucleated red cells. This carries the embryo to roughly the sixth week.

Step 3 – take the second site, the hepatic phase: From about the sixth week the **liver** takes over and is the chief site through mid fetal life, peaking around the fifth month. The **spleen** and the lymph nodes help.

Step 4 – take the third site, the myeloid phase: From about the fifth to seventh month the **bone marrow** takes over, and by birth it is doing essentially all of it.

Step 5 – follow it after birth: In a young child every marrow cavity is red and active. With age the long bone marrow turns to yellow fat, so that in an adult active haematopoiesis is confined to the axial skeleton: vertebrae, sternum, ribs,

pelvis, skull and the proximal ends of femur and humerus.

Step 6 – note the reversal: Under heavy chronic demand, as in thalassaemia, the liver and spleen can restart, giving extramedullary haematopoiesis, and the marrow expands, which is what produces the frontal bossing and maxillary overgrowth seen in the dental chair.

Step 7 – match the sequence: Yolk sac, then liver and spleen, then bone marrow. That is option D.

Why the other options are wrong:

- A, marrow throughout: the marrow does not even exist in a useful form in the early embryo, since the skeleton has not ossified. It is the last site to take over, not the first.
- B, liver then yolk sac then spleen: the order is inverted. The yolk sac is first, and the spleen works alongside the liver rather than after it.
- C, thymus first: the thymus never makes red cells or platelets. It is a site where lymphoid progenitors that arrive from elsewhere mature into T cells, so it is not a general haematopoietic site at all.

Final Answer: Yolk sac, then liver and spleen, then bone marrow $\Rightarrow \boxed{D}$

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – the graveyard of red cells: An ageing red cell has to be caught somewhere. The organ built to catch it is the one whose architecture forces every red cell to prove it is still flexible.

Step 1 – identify the organ: The **spleen** is the answer, and it is called the graveyard of red cells for exactly this reason.

Step 2 – explain the architecture: In the red pulp, blood enters the cords of Billroth and must then squeeze through slits between the endothelial cells of the sinusoids that are only a few micrometres wide.

Step 3 – explain the test that imposes: A young red cell is a deformable biconcave disc and slips through. An old cell of about 120 days has lost membrane, lost ATP and lost flexibility, and it becomes stuck.

Step 4 – explain what happens to the stuck cell: Macrophages lining the cords phagocytose it. This destruction outside the blood vessel lumen is **extravascular haemolysis**, and it accounts for the great majority of normal red cell turnover.

Step 5 – follow the breakdown products: Globin is hydrolysed to amino acids and reused. Iron from haem is returned on transferrin to the marrow. The porphyrin ring becomes biliverdin, then unconjugated bilirubin, which travels on albumin to the liver.

Step 6 – apply it clinically: In hereditary spherocytosis, thalassaemia and autoimmune haemolytic anaemia the spleen destroys defective cells faster still, which is why the spleen enlarges and why splenectomy helps. Splenectomy in turn leaves the patient at risk from encapsulated organisms, since the spleen also filters them.

Step 7 – separate it from intravascular haemolysis: Lysis inside the vessels, as in a mismatched transfusion or PNH, releases free haemoglobin, drops haptoglobin and gives haemoglobinuria. That is the minor route.

Why the other options are wrong:

- A, liver: its Kupffer cells do clear some damaged red cells, and the liver is where bilirubin is conjugated, but it has no cord and sinusoid slit system, so it is not the chief site of red cell removal.
- C, bone marrow: is the site of red cell **production**. It clears only defective precursors, which is ineffective erythropoiesis.
- D, thymus: is a lymphoid organ for T cell maturation. It has no role in red cell destruction and it involutes after puberty.

Final Answer: Spleen $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – chewing is largely automatic: The stem describes a self-sustaining loop that keeps running without a conscious decision at each cycle, which tells you the circuit sits low in the neuraxis.

Step 1 – follow the loop as written: Food presses on the mouth, which reflexly **inhibits** the muscles of mastication.

Step 2 – take the next stage: Inhibition lets the mandible **drop** under gravity.

Step 3 – take the next stage: The drop **stretches** the jaw closing muscles, masseter, temporalis and medial pterygoid.

Step 4 – take the next stage: Stretch fires the muscle spindles, whose afferents run in the mandibular nerve to the **mesencephalic nucleus** of the trigeminal, the one place in the body where primary sensory neurons sit inside the central nervous

system.

Step 5 – close the loop: They synapse onto the **trigeminal motor nucleus** in the pons, producing a rebound contraction that snaps the jaw shut on the bolus, which presses on the mouth again and starts the next cycle.

Step 6 – locate the pattern generator: The circuit that sequences all of this is the masticatory central pattern generator in the **reticular formation of the brainstem**, close to the trigeminal motor nucleus. Stimulating it produces rhythmic chewing.

Step 7 – fit the higher centres in: The cortical masticatory area in the lower precentral region, the amygdala and the hypothalamus can start, stop or modulate the rhythm, which is how chewing can be brought under voluntary control. But the rhythm itself is generated in the brainstem, which is option D.

Why the other options are wrong:

- A, cerebellum: coordinates and smooths jaw movement and helps grade the force, but it generates no rhythm. Cerebellar lesions cause dysmetria and ataxia, not loss of the chewing reflex.
- B, hypothalamus: houses the feeding and satiety centres that decide **whether** to eat. It does not organise the mechanics of the chewing cycle.
- C, cervical spinal cord: the whole reflex is trigeminal. Its afferents and efferents are cranial nerve V, which enters and leaves the pons. The spinal cord is not part of the arc.

Final Answer: The brainstem reticular formation near the trigeminal motor nucleus $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – cardiac muscle is built to beat, never to be tetanised: Three properties define it, and all three come straight out of the structure marked Z and out of the shape of its action potential.

Step 1 – name Z: The dark bands joining cell to cell are **intercalated discs**. They contain desmosomes and fascia adherens, which hold the cells together mechanically against the pull of contraction, and **gap junctions**, which are low resistance channels of connexon protein.

Step 2 – state what the gap junctions do: They let ions, and therefore current,

flow straight from the cytoplasm of one cell into the next. An action potential started anywhere spreads to every connected cell.

Step 3 – name the result: The heart is therefore a **functional syncytium**. It is anatomically many cells but functionally behaves as one, which is why the heart obeys the all or none law as a whole chamber. The atria and ventricles are two separate syncytia, insulated by the fibrous skeleton, with the AV node as the only bridge.

Step 4 – look at the refractory period: The cardiac action potential has a **plateau** of about 200 to 300 milliseconds, held up by slow calcium influx through L-type channels. The absolute refractory period lasts almost as long as the contraction itself.

Step 5 – state what the long refractory period buys: A second stimulus arriving during systole finds the membrane inexcitable. So the twitches cannot summate and the muscle **cannot be tetanised**. This is essential, since a tetanised heart could not relax to fill and would pump nothing.

Step 6 – add automaticity: Pacemaker cells in the SA node have no stable resting potential. The funny current drifts them up to threshold on their own, so the heart beats **without any nerve supply**. Nerves only modulate the rate.

Step 7 – confirm the option: Syncytium, long refractory period, automaticity. All three sit in option C.

Why the other options are wrong:

- A, electrically insulated cells: this describes **skeletal** muscle, where each fibre has its own nerve terminal and no gap junctions. The gap junctions in Z make cardiac muscle the opposite.
- B, short refractory period allowing tetanus: the refractory period is **long**, precisely so that tetanus is impossible. Skeletal muscle has the short one and can be tetanised.
- D, dependence on nerve supply: false. A denervated or transplanted heart keeps beating at its intrinsic SA nodal rate, only faster than usual because vagal tone is gone. This is the whole point of automaticity.

Final Answer: Functional syncytium, long refractory period and automaticity $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – the medulla is a sympathetic ganglion that squirts into the blood: Once you accept that its cells are postganglionic sympathetic neurons that lost their axons, everything about its secretion follows.

Step 1 – take the developmental clue: Both the medulla and the sympathetic ganglia come from **neural crest**. The cortex, in contrast, comes from mesoderm, which is why one gland has two entirely different halves.

Step 2 – take the innervation clue: Preganglionic sympathetic fibres from T5 to T9 run in the greater splanchnic nerve, pass straight through the coeliac plexus without synapsing, and end directly on the chromaffin cells. They release acetylcholine onto nicotinic receptors, exactly as at any sympathetic ganglion.

Step 3 – draw the conclusion: The chromaffin cell is therefore the postganglionic neuron. Having no axon, it releases its transmitter into the **bloodstream** instead of onto a target organ, making it an endocrine gland rather than a nerve ending.

Step 4 – follow the synthesis: Tyrosine goes to DOPA by tyrosine hydroxylase, the rate limiting step, then to dopamine, then to **noradrenaline**.

Step 5 – take the final step, the one unique to the medulla: The enzyme **PNMT** methylates noradrenaline to **adrenaline**. PNMT is induced by the high concentration of cortisol arriving from the cortex through the portal sinusoids, which is why this last step happens here and nowhere else.

Step 6 – give the proportions: About **80 percent adrenaline** and about 20 percent noradrenaline, with a trace of dopamine. So adrenaline is the bulk secretion, which is option D.

Step 7 – note the dental relevance: Adrenaline in local anaesthetic is the same molecule, and a phaeochromocytoma is a chromaffin cell tumour that oversecretes it, giving episodic headache, sweating, palpitations and hypertension.

Why the other options are wrong:

- A, cortisol: is a glucocorticoid from the zona **fasciculata** of the cortex, under ACTH control. It reaches the medulla and induces PNMT, but it is not a medullary product.
- B, aldosterone: is a mineralocorticoid from the zona **glomerulosa** of the cortex, under renin and angiotensin control. Wrong layer and wrong axis.
- C, acetylcholine: is the transmitter of the **preganglionic** fibre arriving at the chromaffin cell, not what the cell secretes. It is the input, not the output.

Final Answer: Adrenaline $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q14

Answer Key

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