NEET MDS General Physiology and Biochemistry

Sample Paper – 5

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 malnourished patient on a maize-based diet has a scaly pigmented rash over sun-exposed skin, chronic diarrhoea and progressive memory loss and confusion. This triad of dermatitis, diarrhoea and dementia is caused by deficiency of:

(A) Riboflavin

(B) Niacin

(C) Pyridoxine

(D) Cobalamin

Q2. A water-soluble vitamin is carried covalently on a lysine residue of its enzyme and serves as the carrier of an activated carbon dioxide group

for pyruvate carboxylase and propionyl-CoA carboxylase. This vitamin is:

(A) Biotin

(B) Pantothenic acid

(C) Riboflavin

(D) Ascorbic acid

Enzymes and Enzyme Kinetics

Q3. A 68-year-old man has bone pain and a slowly enlarging skull, and his hat no longer fits. Serum calcium and phosphate are normal, but one serum enzyme, a marker of osteoblastic activity, is grossly raised. In this Paget disease of bone the raised enzyme is:

(A) Acid phosphatase

(B) Serum amylase

(C) Creatine kinase

(D) Alkaline phosphatase

Q4. Methotrexate is given as a folate analogue in malignancy and in severe rheumatoid arthritis. It binds the active site of its target enzyme far more tightly than the natural substrate and thereby blocks the regeneration of tetrahydrofolate. The enzyme inhibited by methotrexate is:

(A) Thymidylate synthase

(B) Dihydrofolate reductase

(C) Xanthine oxidase

(D) Ribonucleotide reductase

Metabolism of Carbohydrate, Lipid and Protein

Q5. During a fast, glucagon raises hepatic glucose output by breaking stored glycogen down. The enzyme that removes glucose residues one at a time from the non-reducing ends of glycogen, using inorganic phosphate to release glucose-1-phosphate, is:

(A) Glycogen synthase

(B) Branching enzyme

(C) Debranching enzyme

(D) Glycogen phosphorylase

Q6. In the well-fed state, cytosolic acetyl-CoA is committed to fatty acid synthesis by a single biotin-dependent, citrate-activated step that is switched off by phosphorylation. The rate limiting enzyme of fatty acid synthesis is:

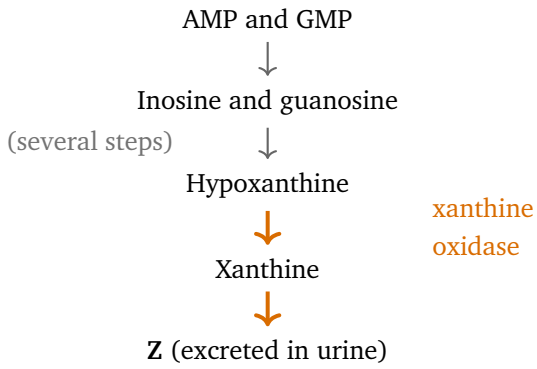
(A) Fatty acid synthase

(B) Acetyl-CoA carboxylase

(C) ATP-citrate lyase

(D) Carnitine palmitoyltransferase-1

Q7. The chain below outlines the catabolism of purine nucleotides in humans. The compound marked **Z** is the end product that is excreted, and it crystallises in the first metatarsophalangeal joint when its plasma level is high. Compound **Z** is:



(A) Uric acid

(B) Urea

(C) Beta-alanine

(D) Ammonia

Acid Base Balance and Body Fluids

Q8. In healthy arterial blood the pH sits between 7.35 and 7.45, plasma bicarbonate is about 24 mEq/L and dissolved carbonic acid is about 1.2 mEq/L. At this normal pH the ratio of bicarbonate to carbonic acid is:

(A) 20 : 1

(B) 10 : 1

(C) 1 : 20

(D) 4 : 1

Q9. The kidney handles the fixed acid produced each day by dietary protein. Its regulation of acid base balance is achieved mainly by:

(A) Excreting large amounts of free unbuffered hydrogen ions into the tubular urine

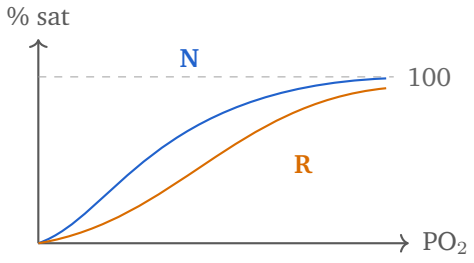
(B) Reabsorbing filtered hydrogen ions while allowing bicarbonate to be lost in urine

(C) Secreting hydrogen ions into the tubular lumen, reclaiming the filtered bicarbonate and trapping the secreted acid on ammonia and phosphate buffers

(D) Changing alveolar ventilation so that carbon dioxide is blown off

Blood and Haemostasis

Q10. The graph below shows the oxygen-haemoglobin dissociation curve. Curve **N** is normal and curve **R** is displaced as shown, so that haemoglobin gives up oxygen more readily to the tissues. The set of changes that produces curve **R** is:



(A) Fall in PCO_2, fall in temperature and rise in pH

(B) Rise in pH, fall in 2,3-BPG and fall in temperature

(C) Rise in PCO_2, rise in temperature, fall in pH and rise in 2,3-BPG

(D) Replacement of adult haemoglobin by fetal haemoglobin

Q11. A patient with a prosthetic heart valve takes an oral anticoagulant that acts as a vitamin K antagonist. Before extracting a tooth you must check the laboratory test used to monitor that drug. The drug and its monitoring test are:

(A) Warfarin, monitored by the prothrombin time reported as INR

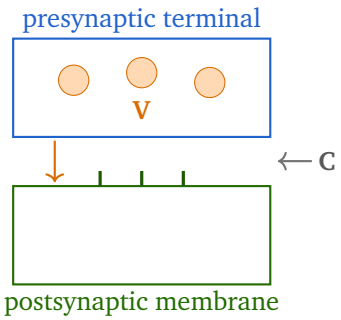
(B) Heparin, monitored by the prothrombin time reported as INR

(C) Warfarin, monitored by the activated partial thromboplastin time

(D) Aspirin, monitored by the activated partial thromboplastin time

Nerve, Muscle and Endocrine Physiology

Q12. The sketch below shows a chemical synapse, with the vesicles marked **V** in the presynaptic terminal and the cleft marked **C**. When an action potential reaches the terminal, the correct order of events is:



(A) Transmitter release, then calcium entry, then depolarisation of the terminal

(B) Depolarisation of the terminal, opening of voltage-gated calcium channels, calcium influx, exocytosis of vesicles, diffusion of transmitter across the cleft, binding to postsynaptic receptors

(C) Binding to postsynaptic receptors, then vesicle fusion, then calcium influx

(D) Calcium efflux from the terminal, then transmitter uptake, then hyperpolarisation

Q13. Compared with skeletal muscle, smooth muscle in the wall of a blood vessel differs in that:

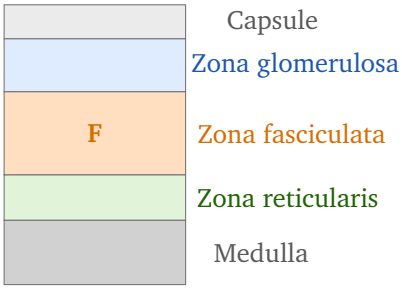
(A) It is striated and uses troponin C to bind calcium

(B) It contains no actin or myosin and contracts by cytoplasmic streaming

(C) It is non-striated, has no troponin, binds calcium to calmodulin to activate myosin light chain kinase, and produces slow sustained contraction

(D) It is non-striated but still uses troponin C, and it contracts and relaxes faster than skeletal muscle

Q14. The section below shows the layers of the adrenal gland. The zone marked **F** is the broad middle zone of the cortex, and it is the zone driven by ACTH from the anterior pituitary. The principal hormone secreted by zone **F** is:



(A) Aldosterone

(B) Adrenaline

(C) Dehydroepiandrosterone

(D) Cortisol

Detailed Solutions

Q1.

Solution

Concept – pellagra is the disease of the three Ds: Dermatitis, diarrhoea and dementia together name one deficiency and only one.

Step 1 – name the vitamin: The three Ds are the classical picture of **niacin (vitamin B3)** deficiency, that is pellagra.

Step 2 – explain why the diet matters: Maize is poor in available niacin and also poor in tryptophan, from which the body can make niacin at a rate of about 1 mg niacin per 60 mg tryptophan. A maize-based diet therefore starves the patient of both routes.

Step 3 – explain the dermatitis: The rash is symmetrical and appears only on **sun-exposed** skin, classically as a collar around the neck (Casal necklace), because photosensitive skin needs NAD-dependent repair.

Step 4 – explain the diarrhoea: Rapidly dividing gut mucosa fails without NAD^+ and $NADP^+$, giving atrophy, glossitis and chronic diarrhoea. The tongue is beefy red, which is the finding a dentist meets first.

Step 5 – explain the dementia: Neurons depend heavily on NAD-linked energy production, so deficiency gives irritability, memory loss and frank confusion.

Step 6 – confirm: All three features in the stem are accounted for by niacin deficiency, so option B is correct.

Why the other options are wrong:

- A, riboflavin: deficiency causes angular stomatitis, cheilosis, glossitis and a magenta tongue. It does not cause the dementia or the photosensitive rash.
- C, pyridoxine: deficiency causes peripheral neuropathy, convulsions and sideroblastic anaemia. Note that pyridoxine deficiency can worsen pellagra, since PLP is needed to convert tryptophan to niacin, but it is not the primary answer here.
- D, cobalamin: deficiency causes megaloblastic anaemia and subacute combined degeneration of the cord, not dermatitis and diarrhoea.

Final Answer: Niacin $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – one vitamin carries carbon dioxide: Every carboxylase in the body needs a way to hold an activated carbon dioxide group, and only one vitamin does that job.

Step 1 – read the first clue: The vitamin is bound **covalently** to a lysine residue of the enzyme. That covalent, permanent attachment is the hallmark of **biotin**, which forms a biocytin link.

Step 2 – read the second clue: It carries an activated carbon dioxide group. Biotin is carboxylated on its ureido nitrogen using bicarbonate and ATP, and it then donates that carbon dioxide to the substrate.

Step 3 – check the named enzymes: Pyruvate carboxylase (pyruvate to oxaloacetate, in gluconeogenesis) and propionyl-CoA carboxylase (propionyl-CoA to methylmalonyl-CoA) are both biotin-dependent carboxylases. So is acetyl-CoA carboxylase.

Step 4 – note the clinical link: Dietary deficiency is rare, but eating large amounts of raw egg white causes it, because avidin in the white binds biotin and blocks its absorption.

Why the other options are wrong:

- B, pantothenic acid: is part of **coenzyme A** and of the acyl carrier protein. It carries acyl groups, not carbon dioxide.
- C, riboflavin: becomes FAD and FMN, which carry **hydrogen** in redox reactions.
- D, ascorbic acid: is a reducing agent for hydroxylation reactions and is not a carboxyl carrier at all.

Final Answer: Biotin $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – serum enzymes report the tissue they leak from: Match the enzyme to the cell that makes it, then match that cell to the disease.

Step 1 – identify the cell in the stem: The question states the enzyme is a marker of **osteoblastic activity**, so the source cell is the osteoblast.

Step 2 – name the osteoblast enzyme: Osteoblasts are rich in **alkaline phosphatase**, which they use to hydrolyse pyrophosphate and allow mineral to be laid down.

Step 3 – fit the disease: Paget disease of bone is disordered remodelling: osteoclasts resorb wildly, and osteoblasts lay down new woven bone just as fast. That furious osteoblastic response floods the serum with alkaline phosphatase.

Step 4 – explain the normal calcium and phosphate: Because resorption and formation are coupled in Paget disease, calcium released is promptly reused, so serum calcium and phosphate stay normal while alkaline phosphatase is grossly raised. That dissociation is the exam clue.

Step 5 – note the dental relevance: Paget disease of the jaws causes hypercementosis and loss of the lamina dura, so extraction is difficult and bleeds heavily from the vascular bone.

Why the other options are wrong:

- A, acid phosphatase: comes largely from the **prostate** and from osteoclasts as the tartrate-resistant form. It is not the osteoblastic marker asked for.
- B, serum amylase: rises in acute pancreatitis and in salivary gland disease such as mumps. It has nothing to do with bone.
- C, creatine kinase: rises with damage to skeletal muscle, cardiac muscle and brain. Bone does not release it.

Final Answer: Alkaline phosphatase $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q3

Q4.

Solution

Concept – a drug that looks like a substrate blocks that substrate's enzyme: Name the natural substrate the drug imitates, and the target enzyme falls out at once.

Step 1 – read what the drug resembles: Methotrexate is a **folate analogue**. Its structure is close to that of dihydrofolate.

Step 2 – read what the drug blocks: The stem says it blocks the **regeneration of tetrahydrofolate**. So the reaction blocked is dihydrofolate to tetrahydrofolate.

Step 3 – name the enzyme of that reaction: Dihydrofolate is reduced to tetrahydrofolate by **dihydrofolate reductase (DHFR)**, using NADPH.

Step 4 – explain the tight binding: Methotrexate occupies the DHFR active site with an affinity roughly a thousand times that of the natural substrate, which is why the block is so effective in practice even though the mechanism is reversible.

Step 5 – explain the consequence: Without tetrahydrofolate the cell cannot make thymidylate or purines, so DNA synthesis stops. Rapidly dividing cells suffer first, which gives both the anti-tumour effect and the oral mucositis a dentist sees.

Step 6 – note the rescue: Folinic acid (leucovorin) bypasses the block because it is already a reduced folate and needs no DHFR.

Why the other options are wrong:

- A, thymidylate synthase: is inhibited by **5-fluorouracil**, not by methotrexate. Methotrexate starves it of its cofactor indirectly, but that is not the enzyme the drug binds.
- C, xanthine oxidase: is the target of **allopurinol** in gout. It has no role in folate metabolism.
- D, ribonucleotide reductase: is inhibited by **hydroxyurea**, and it converts ribonucleotides to deoxyribonucleotides.

Final Answer: Dihydrofolate reductase $\Rightarrow$ B

Answer: (B) Go Back to Q4

Q5.

Solution

Concept – glycogen is built by one enzyme and dismantled by another: Synthesis and breakdown use separate enzymes so that the two can be regulated in opposite directions.

Step 1 – read the direction: The stem describes a **fast**, with glucagon acting and glucose leaving the liver. So the pathway is glycogenolysis, that is breakdown.

Step 2 – read the chemistry: The enzyme uses **inorganic phosphate**, not water, to cleave the bond. That is phosphorolysis rather than hydrolysis.

Step 3 – read the product: The product is **glucose-1-phosphate**, which is what phosphorolysis of an alpha-1,4 bond gives.

Step 4 – name the enzyme: An enzyme that attacks the non-reducing ends of glycogen, uses inorganic phosphate and releases glucose-1-phosphate is **glycogen phosphorylase**.

Step 5 – confirm it is the regulated one: Glycogen phosphorylase is the rate

limiting enzyme of glycogenolysis. Glucagon and adrenaline raise cAMP, which activates protein kinase A, which activates phosphorylase kinase, which phosphorylates phosphorylase b to the active phosphorylase a. That cascade is exactly what the stem sets up.

Step 6 – follow the product on: Glucose-1-phosphate becomes glucose-6-phosphate through phosphoglucomutase, and in the liver glucose-6-phosphatase then frees glucose for the blood.

Why the other options are wrong:

- A, glycogen synthase: adds glucose **to** glycogen from UDP-glucose. It is the opposite direction, and glucagon switches it off.
- B, branching enzyme: transfers a block of residues to create an alpha-1,6 branch during synthesis, so it also builds rather than breaks.
- C, debranching enzyme: is needed in glycogenolysis, but only to clear the alpha-1,6 branch points once phosphorylase stalls four residues away. It releases **free glucose**, not glucose-1-phosphate, and it is not the main or rate limiting enzyme.

Final Answer: Glycogen phosphorylase $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – the committed step of a pathway is its rate limiting step: The first step that cannot be used by any other pathway is the one worth regulating.

Step 1 – find the committed step: Cytosolic acetyl-CoA can go to cholesterol or to fatty acids. The moment it becomes **malonyl-CoA** it is committed to fatty acid synthesis and to nothing else.

Step 2 – name the enzyme of that step: Acetyl-CoA plus bicarbonate plus ATP gives malonyl-CoA, catalysed by **acetyl-CoA carboxylase (ACC)**.

Step 3 – check the biotin clue: ACC is a **biotin-dependent** carboxylase, exactly as the stem says.

Step 4 – check the citrate clue: Citrate is the allosteric **activator** of ACC. When the fed cell has plenty of energy, citrate leaves the mitochondrion, which both supplies acetyl-CoA and switches ACC on. Palmitoyl-CoA, the end product, inhibits it.

Step 5 – check the phosphorylation clue: Glucagon and adrenaline phosphory-

late ACC and switch it off; insulin dephosphorylates it and switches it on. That fits the well-fed state described.

Step 6 – note the elegant cross-talk: Malonyl-CoA also inhibits CPT-1, so while fat is being made, fat cannot simultaneously be burned. One enzyme therefore controls both directions.

Why the other options are wrong:

- A, fatty acid synthase: is the multi-enzyme complex that does the seven cycles of condensation and reduction to build palmitate. It works hard but it is **downstream** of the committed step and is not rate limiting.
- C, ATP-citrate lyase: splits citrate in the cytosol to give acetyl-CoA and oxaloacetate. It supplies the substrate but the acetyl-CoA it makes is still free to go to cholesterol, so this step is not committed.
- D, carnitine palmitoyltransferase-1: carries fatty acids **into** the mitochondrion for beta-oxidation. It is the rate limiting step of fat breakdown, the opposite pathway.

Final Answer: Acetyl-CoA carboxylase $\Rightarrow$ B

Answer: (B) Go Back to Q6

Q7.

Solution

Concept – humans cannot break the purine ring down completely: Unlike many animals, we lack uricase, so purine catabolism stops one step early.

Step 1 – follow the chain in the diagram: AMP and GMP lose their phosphate and their sugar to give the free bases, reaching hypoxanthine and then xanthine.

Step 2 – take the last two steps: Xanthine oxidase converts hypoxanthine to xanthine, and then xanthine to **uric acid**. Both arrows marked in the figure are that one enzyme.

Step 3 – stop where humans stop: Most mammals go on to convert uric acid to the far more soluble allantoin using uricase. Humans have lost that enzyme, so **uric acid is the end product** and it must be excreted as it is.

Step 4 – explain the clinical clue: Uric acid is poorly soluble. When plasma urate rises, monosodium urate crystals precipitate in cool peripheral joints, classically the first metatarsophalangeal joint (podagra), triggering the acute inflammation of **gout**.

Step 5 – link back to the diagram: Because xanthine oxidase makes the offending

product, blocking it with allopurinol lowers urate. That is why the figure names that enzyme.

Why the other options are wrong:

- B, urea: is the end product of **amino acid** nitrogen disposal through the urea cycle, not of purine rings.
- C, beta-alanine: is an end product of **pyrimidine** catabolism (from cytosine and uracil), which is the other nucleotide family.
- D, ammonia: is released at intermediate deamination steps and is promptly detoxified. It is not the excreted end product of purine breakdown.

Final Answer: Uric acid $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – normal pH is set by a ratio, not by an absolute amount: Blood pH depends on how much bicarbonate there is *relative to* carbonic acid, so the ratio is the number to remember.

Step 1 – take the two values given: Bicarbonate = 24 mEq/L. Carbonic acid = 1.2 mEq/L.

Step 2 – form the ratio: Ratio $= \dfrac{24}{1.2}$

Step 3 – clear the decimal: $\dfrac{24}{1.2} = \dfrac{240}{12}$

Step 4 – divide: $\dfrac{240}{12} = 20$

Step 5 – state the ratio: So bicarbonate to carbonic acid is **20 : 1** at normal pH.

Step 6 – see why that ratio gives 7.4: The pK of the bicarbonate system is 6.1. Adding the log of the ratio, $\log 20 = 1.3$, gives $6.1 + 1.3 = 7.4$, which is exactly the normal arterial pH quoted in the stem.

Step 7 – read the physiology out of it: Any change that alters the ratio alters the pH. The lung controls the carbonic acid on the bottom by changing ventilation; the kidney controls the bicarbonate on the top. Keep the ratio at 20:1 and the pH holds at 7.4 whatever the absolute values are.

Why the other options are wrong:

- B, 10:1: would give pH $= 6.1 + \log 10 = 6.1 + 1 = 7.1$, a severe acidosis, not

a normal blood gas.

- C, 1:20: is the ratio inverted. It would give pH $= 6.1 - 1.3 = 4.8$, which is incompatible with life.
- D, 4:1: would give pH $= 6.1 + \log 4 = 6.1 + 0.6 = 6.7$, again far too acidic.

Final Answer: 20 : 1 $\Rightarrow$ A

Answer: (A) Go Back to Q8

Q9.

Solution

Concept – the kidney is the slow but permanent line of defence: Buffers act in seconds, the lung in minutes, but only the kidney can actually remove fixed acid from the body.

Step 1 – state the load: An ordinary diet generates roughly 50 to 100 mEq of non-volatile acid a day from sulphur-containing and phosphate-containing foods. The lung cannot touch this, because it is not carbon dioxide.

Step 2 – describe hydrogen ion secretion: Tubular cells generate H^+ and bicarbonate from carbon dioxide and water using carbonic anhydrase. The H^+ is pumped into the lumen, through the Na^+-H^+ exchanger in the proximal tubule and through H^+-ATPase in the alpha intercalated cells of the collecting duct.

Step 3 – describe bicarbonate reclamation: About 4300 mEq of bicarbonate is filtered daily and essentially all of it is reabsorbed, most in the proximal tubule. Losing that bicarbonate would itself be an acid load, so reclaiming it is half the job.

Step 4 – explain why buffers are needed: Free H^+ cannot be excreted in quantity. Urine pH can fall only to about 4.5, and even at that pH the free H^+ excreted is a fraction of a milliequivalent. The acid must therefore be trapped on something.

Step 5 – name the buffers: Filtered phosphate accepts H^+ to become titratable acid, and **ammonia**, made from glutamine in the proximal tubule, traps H^+ as ammonium, which is charged and cannot diffuse back. Ammonium excretion is the adaptable component and rises several fold in chronic acidosis.

Step 6 – combine: Secreting H^+, reclaiming bicarbonate and buffering the secreted acid on ammonia and phosphate is precisely option C.

Why the other options are wrong:

- A, large amounts of free hydrogen ion: is impossible. The minimum urine pH of 4.5 limits free H^+ excretion to a trivial amount, which is exactly why

buffers exist.

- B, reabsorbing hydrogen ions and losing bicarbonate: is backwards on both counts. The kidney secretes H^+ and reabsorbs bicarbonate. Doing what this option says would cause an acidosis, as happens in renal tubular acidosis.
- D, changing alveolar ventilation: is **respiratory** compensation, done by the lung. The question asks about the renal mechanism.

Final Answer: Hydrogen ion secretion with bicarbonate reclamation and ammonia and phosphate buffering $\Rightarrow$ C

Answer: (C) Go Back to Q9

Q10.

Solution

Concept – a right shift means haemoglobin lets oxygen go: Read the direction of the shift first, then ask what conditions a working tissue creates.

Step 1 – read the graph: Curve R lies to the right of curve N, so at any given PO_2 the saturation is lower. The P_{50}, the PO_2 at which haemoglobin is half saturated, is raised above its normal 27 mmHg.

Step 2 – translate that into affinity: A right shift means **reduced** affinity of haemoglobin for oxygen, so oxygen is unloaded more readily. That matches the stem.

Step 3 – ask where this should happen: It should happen in an actively metabolising tissue, which is exactly where oxygen is needed.

Step 4 – list what an active tissue produces: It makes more carbon dioxide, it is warmer, it is more acidic from carbon dioxide and lactate, and chronic hypoxia raises red cell 2,3-BPG.

Step 5 – match to the options: Rise in PCO_2, rise in temperature, fall in pH and rise in 2,3-BPG is the standard right-shift set, which is option C. The pH effect is the **Bohr effect**.

Step 6 – keep the mnemonic: Everything that goes up (CO_2, temperature, H^+, 2,3-BPG) shifts the curve right, and 2,3-BPG works by binding deoxyhaemoglobin and holding it in the low-affinity T state.

Why the other options are wrong:

- A, fall in PCO_2, fall in temperature, rise in pH: is every factor reversed, so it shifts the curve **left** and makes haemoglobin hold oxygen tighter.
- B, rise in pH, fall in 2,3-BPG, fall in temperature: is also a left-shift set.

Stored blood loses 2,3-BPG for this very reason.

- D, fetal haemoglobin: has **higher** affinity because its gamma chains bind 2,3-BPG poorly, so it shifts the curve left. That is how the fetus pulls oxygen from maternal blood.

Final Answer: Rise in PCO_2, rise in temperature, fall in pH and rise in 2,3-BPG $\Rightarrow$ C

Answer: (C) Go Back to Q10

Q11.

Solution

Concept – each anticoagulant has its own test: Pair the drug with the limb of the cascade it hits, and the monitoring test follows.

Step 1 – identify the drug: The stem says an **oral** anticoagulant that is a **vitamin K antagonist**. That is **warfarin**.

Step 2 – state how it acts: Warfarin blocks vitamin K epoxide reductase, so the reduced vitamin K needed to activate clotting factors II, VII, IX and X is not regenerated. Those factors are made but are functionally useless.

Step 3 – decide which test detects that: Factor VII has the shortest half-life, about 6 hours, so it falls first. Factor VII sits in the tissue factor limb, and the test that reads that limb is the **prothrombin time**.

Step 4 – explain the INR: Raw PT varies between laboratories because thromboplastin reagents differ. The International Normalised Ratio corrects for this, so the same patient gives the same number anywhere. Warfarin is therefore monitored by PT reported as INR.

Step 5 – contrast with heparin: Heparin is parenteral, potentiates antithrombin III, and mainly hits thrombin and factor Xa along with the intrinsic limb, so unfractionated heparin is monitored by the **aPTT**. That contrast is the trap in this question.

Step 6 – apply it in the dental chair: For simple extractions an INR up to about 3.5 is usually acceptable with local measures such as sutures and tranexamic acid mouthwash. Stopping warfarin risks thromboembolism, which is far worse than a socket that oozes.

Why the other options are wrong:

- B, heparin with PT/INR: gets both halves wrong. Heparin is injected, not oral, it is not a vitamin K antagonist, and it is monitored by aPTT.

- C, warfarin with aPTT: names the right drug but the wrong test. The aPTT does move a little on warfarin, but it is not the test used to titrate the dose.
- D, aspirin with aPTT: aspirin is an antiplatelet drug that irreversibly acetylates cyclooxygenase-1. It is not a vitamin K antagonist and it has no routine monitoring test.

Final Answer: Warfarin, monitored by PT reported as INR $\Rightarrow$ A

Answer: (A) Go Back to Q11

Q12.

Solution

Concept – a chemical synapse converts electricity to chemistry and back: The order never changes, and calcium is the hinge between the two halves.

Step 1 – the action potential arrives: It depolarises the presynaptic terminal shown in the diagram.

Step 2 – calcium channels open: Depolarisation opens **voltage-gated calcium channels** in the terminal membrane.

Step 3 – calcium enters: Extracellular calcium is about 10 000 times more concentrated than cytosolic calcium, so calcium floods in down a very steep gradient.

Step 4 – vesicles fuse: Calcium binds synaptotagmin on the vesicles marked **V**, which drives the SNARE proteins to pull vesicle and terminal membranes together. The vesicles fuse and release transmitter by exocytosis.

Step 5 – transmitter crosses the cleft: The transmitter diffuses across the cleft marked **C**, a gap of only about 20 to 40 nm, which it crosses in well under a millisecond.

Step 6 – receptors are occupied: It binds receptors on the postsynaptic membrane, opening ion channels and producing an excitatory or inhibitory postsynaptic potential.

Step 7 – match to the options: Depolarisation, calcium channel opening, calcium influx, exocytosis, diffusion, receptor binding is exactly the order in option B.

Why the other options are wrong:

- A: puts transmitter release **before** calcium entry and before depolarisation. Nothing triggers release without calcium, so this order is impossible.
- C: starts at the postsynaptic receptors, which is the last event, and it runs the whole synapse backwards.
- D: has calcium leaving the terminal instead of entering. Calcium **influx** is

the trigger, and transmitter uptake is a termination step, not an initiating one.

Final Answer: Depolarisation → calcium influx → exocytosis → receptor binding ⇒ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – both muscles slide actin on myosin, but they switch it on differently: The contractile proteins are similar; the calcium sensor is not.

Step 1 – compare the architecture: Skeletal muscle has sarcomeres in register, which produces visible **striations**. Smooth muscle has actin and myosin anchored to dense bodies in an oblique lattice, so there are no striations.

Step 2 – compare the calcium sensor: Skeletal muscle has **troponin C** on the thin filament. Smooth muscle has **no troponin** at all; it uses **calmodulin** in the cytosol.

Step 3 – follow the smooth muscle pathway: Calcium binds calmodulin, the calcium-calmodulin complex activates **myosin light chain kinase**, and that kinase phosphorylates the regulatory light chain of myosin. Only then can myosin engage actin.

Step 4 – contrast the skeletal pathway: In skeletal muscle calcium binds troponin C, tropomyosin moves off the actin binding site, and myosin heads attach at once. Regulation is on the **thin** filament, whereas smooth muscle regulates the **thick** filament.

Step 5 – explain the speed: Phosphorylating myosin takes time, and smooth muscle myosin ATPase is far slower, so contraction is slow in onset and slow to relax.

Step 6 – explain the economy: Smooth muscle can enter the latch state, holding tension with very slow cross-bridge cycling and very little ATP. That is how a vessel or a sphincter maintains tone for hours without fatigue.

Step 7 – match: Non-striated, no troponin, calmodulin and myosin light chain kinase, slow sustained contraction is option C.

Why the other options are wrong:

- A, striated and using troponin C: describes **skeletal** muscle, that is the muscle being compared against, not smooth muscle.

- B, no actin or myosin: is simply false. Smooth muscle has both; only the orderly sarcomere arrangement is missing.
- D, non-striated but using troponin C and faster than skeletal: is right about the striations and wrong about everything else. Smooth muscle has no troponin, and it is markedly **slower**, not faster.

Final Answer: Non-striated, no troponin, calmodulin-dependent, slow sustained contraction ⇒ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – each adrenal layer makes its own class of steroid: Three cortical zones from outside in, three hormone classes in the same order.

Step 1 – name the layers from the diagram: Under the capsule lie the zona glomerulosa, then the zona fasciculata, then the zona reticularis, and inside all of them the medulla.

Step 2 – identify the zone marked F: The stem says F is the broad **middle** zone of the cortex. That is the **zona fasciculata**, which makes up roughly 75 percent of the cortex.

Step 3 – use the ACTH clue: The stem says F is driven by **ACTH**. The fasciculata and the reticularis are ACTH-dependent, while the glomerulosa answers mainly to angiotensin II and potassium.

Step 4 – name the hormone: The zona fasciculata secretes **glucocorticoids**, principally **cortisol**.

Step 5 – fix the order with a mnemonic: GFR from outside in gives Salt, Sugar, Sex: glomerulosa makes mineralocorticoids (salt), fasciculata makes glucocorticoids (sugar), reticularis makes androgens (sex).

Step 6 – note the dental relevance: Because cortisol output rises with stress, a patient on long-term steroids has a suppressed axis and may need cover for a surgical extraction. This is why the fasciculata matters in the dental chair.

Why the other options are wrong:

- A, aldosterone: comes from the **zona glomerulosa**, the thin outer zone just under the capsule, and it is regulated by the renin-angiotensin system, not chiefly by ACTH.
- B, adrenaline: comes from the **medulla**, which is modified sympathetic tis-

sue of neural crest origin, not cortex at all.

- C, dehydroepiandrosterone: comes from the **zona reticularis**, the innermost cortical zone lying against the medulla.

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

Answer: (D) Go Back to Q14

Answer Key

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