NEET MDS General Physiology and Biochemistry

Sample Paper – 8

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 breastfed baby of 4 days presents with bleeding from the umbilical stump and melaena, with a normal platelet count. The single intramuscular injection routinely given to every newborn at birth to prevent this haemorrhagic disease is:

(A) Vitamin C

(B) Vitamin K

(C) Vitamin D

(D) Vitamin B12

Q2. A patient complains of a blunted sense of taste and reports that a healing extraction socket is taking far longer than expected to close. The

trace element whose deficiency classically causes hypogeusia together with impaired wound healing is:

(A) Iron

(B) Zinc

(C) Iodine

(D) Fluoride

Enzymes and Enzyme Kinetics

Q3. A patient on long-term rifampicin needs a much larger dose of a second drug that is cleared by the hepatic cytochrome P450 enzyme CYP3A4. The mechanism behind this loss of effect is that rifampicin:

(A) Competes with the second drug for the active site of CYP3A4

(B) Irreversibly destroys the haem group of CYP3A4

(C) Induces the liver to synthesise more CYP3A4 enzyme protein

(D) Blocks the renal tubular secretion of the second drug

Q4. Pepsin is secreted by the gastric chief cells as the inactive precursor pepsinogen and is switched on by the hydrochloric acid of the stomach. The pH at which pepsin works best is:

(A) pH 7.4

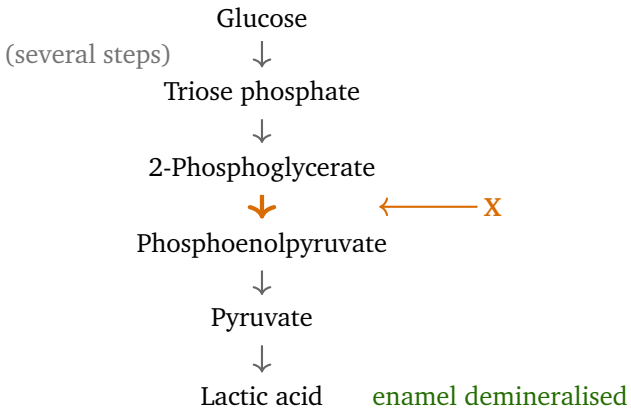
(B) pH 6.8

(C) pH 8.0 to 9.0

(D) pH 1.5 to 2.0

Metabolism of Carbohydrate, Lipid and Protein

Q5. The chain below is the glycolytic route that plaque bacteria use to turn dietary sugar into acid. Fluoride from toothpaste and varnish blocks the single step marked **X**, so acid production falls. The enzyme catalysing **X** is:



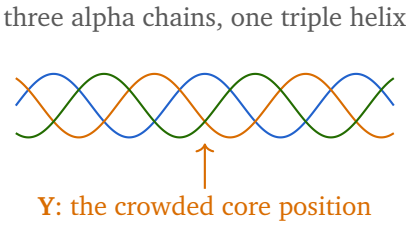
(A) Hexokinase

(B) Enolase

(C) Lactate dehydrogenase

(D) Aldolase

Q6. The figure shows the three left-handed alpha chains of collagen wound into a right-handed superhelix. At the position marked **Y** the three chains pack tightest, so only the smallest amino acid fits there. The repeating tripeptide of the collagen alpha chain is therefore:



(A) Pro-X-Y, with proline at every third position

(B) Gly-Gly-Gly, an unbroken run of glycine

(C) Gly-X-Y, with X often proline and Y often hydroxyproline

(D) Lys-X-Y, with lysine at every third position

Q7. During DNA replication the lagging strand is copied in short pieces called Okazaki fragments. After the RNA primers are removed and the gaps filled, the enzyme that seals the remaining nick and joins one fragment to the next is:

(A) DNA ligase

(B) DNA helicase

(C) DNA polymerase III

(D) Primase

Acid Base Balance and Body Fluids

Q8. The pKa of the bicarbonate system is 6.1, more than one pH unit away from the blood pH of 7.4, which on paper makes it a poor buffer. Yet it is the most important buffer of the extracellular fluid. The reason is that:

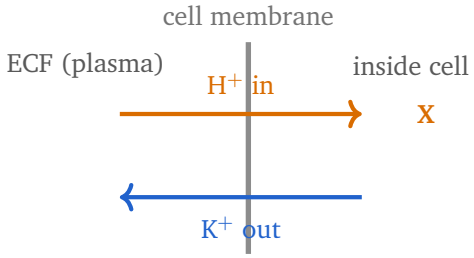
(A) Its pKa shifts to 7.4 once it is dissolved in plasma

(B) It is present at a higher concentration than any other buffer inside the cell

(C) It is an open system, because the lungs can blow off carbon dioxide and the kidney can retain or excrete bicarbonate, so both halves of the pair are adjusted independently and the system never gets used up

(D) It is the only buffer able to handle carbonic acid produced by respiration

Q9. The figure shows what happens at the cell membrane when a patient becomes acidotic. Excess hydrogen ions move into the cell along the arrow marked **X**, and electrical neutrality is maintained by the paired movement shown. The expected change in serum potassium is:



(A) Serum potassium falls, because potassium follows hydrogen into the cell

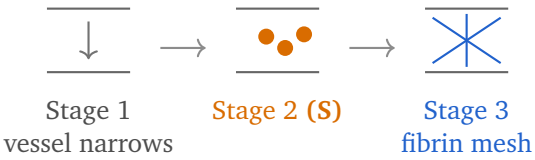
(B) Serum potassium rises, because potassium leaves the cell in exchange for the hydrogen entering it

(C) Serum potassium is unchanged, because the exchange is confined to the cell interior

(D) Serum potassium falls, because acidosis suppresses aldosterone

Blood and Haemostasis

Q10. The panels below follow a cut vessel in an extraction socket through the three stages of haemostasis, left to right in time order. The correct sequence, and hence the event at the stage marked **S**, is:



(A) Vascular spasm, then platelet plug formation at **S**, then coagulation with a fibrin clot

(B) Coagulation, then vascular spasm at **S**, then platelet plug formation

(C) Platelet plug formation, then vascular spasm at **S**, then coagulation

(D) Vascular spasm, then coagulation at **S**, then platelet plug formation

Q11. A young woman gives a long history of heavy periods, easy bruising and prolonged oozing after a dental extraction. Her disorder is caused by a deficiency of von Willebrand factor. The two functions this molecule normally performs are:

(A) It converts fibrinogen to fibrin and then cross-links the fibrin strands

(B) It is a vitamin K dependent factor and also activates protein C

(C) It activates plasminogen to plasmin and also degrades fibrin

(D) It anchors platelets to subendothelial collagen and also carries factor VIII in the plasma, protecting it from breakdown

Nerve, Muscle and Endocrine Physiology

Q12. The statement that correctly describes the outflow and the transmitters of the two divisions of the autonomic nervous system is:

(A) The parasympathetic outflow is craniosacral and uses acetylcholine at both the ganglion and the target organ, while the sympathetic outflow is thoracolumbar and uses acetylcholine at the ganglion but noradrenaline at most target organs

(B) The parasympathetic outflow is thoracolumbar and the sympathetic outflow is craniosacral, and both use noradrenaline throughout

(C) Both divisions leave the cord in the thoracolumbar segments, the parasympathetic using noradrenaline and the sympathetic using acetylcholine at every synapse

(D) The sympathetic outflow is craniosacral and uses acetylcholine at the target organ, while the parasympathetic outflow is thoracolumbar and uses noradrenaline at the target organ

Q13. Both autonomic divisions supply the salivary glands, but the saliva they produce is quite different. The correct description of that difference is:

(A) Parasympathetic stimulation gives scanty viscous saliva and sympathetic stimulation gives copious watery saliva

(B) Both divisions give copious watery saliva, differing only in the speed of onset

(C) Only the sympathetic supply reaches the salivary glands, so all saliva is viscous

(D) Parasympathetic stimulation gives copious watery saliva through vasodilatation and heavy fluid secretion, while sympathetic stimulation gives scanty viscous saliva rich in protein

Q14. The mineralocorticoid that is secreted from the outermost layer of the adrenal cortex and acts on the distal tubule and collecting duct is best described as:

(A) Aldosterone, from the zona glomerulosa, which reabsorbs sodium and water and excretes potassium and hydrogen

(B) Cortisol, from the zona fasciculata, which reabsorbs potassium and excretes sodium

(C) Dehydroepiandrosterone, from the zona reticularis, which reabsorbs sodium and excretes potassium

(D) Adrenaline, from the adrenal medulla, which reabsorbs sodium and excretes potassium

Detailed Solutions

Q1.

Solution

Concept – haemorrhagic disease of the newborn: Every newborn starts life short of one particular vitamin, and that shortage shows itself as bleeding.

Step 1 – read the clinical picture: A baby of a few days old bleeds from the umbilical stump and passes melaena, yet the platelet count is normal. So the platelets are fine and the fault lies in coagulation.

Step 2 – ask why a newborn is short of vitamin K: Vitamin K crosses the placenta poorly, the newborn gut is still sterile so no gut bacteria are making it, and breast milk contains very little of it. All three point the same way.

Step 3 – add the feeding clue: The baby is breastfed, which is the classic setting, because formula is fortified with vitamin K while breast milk is not.

Step 4 – name the prophylaxis: A single intramuscular dose of **vitamin K** is given to every newborn at birth. It is universal practice precisely because this bleeding can be fatal and is entirely preventable.

Step 5 – note the dental relevance: The same vitamin K dependent factors are the ones you worry about later in life when a patient on warfarin comes for an extraction.

Why the other options are wrong:

- A, vitamin C: its deficiency does cause bleeding, but only after months of a deficient diet, and it presents as scurvy in older children and adults, never as a bleed on day 4 of life.
- C, vitamin D: is given as drops for rickets prophylaxis, not as an injection at birth, and its deficiency causes bone disease and not bleeding.
- D, vitamin B12: its deficiency causes megaloblastic anaemia and neurological signs. It is not given routinely at birth and does not prevent neonatal bleeding.

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

Answer: (B) Go Back to Q1

Q2.

Solution

Concept – zinc is the trace element of taste and of healing: Two complaints, blunted taste and a socket that will not close, point to one mineral.

Step 1 – take the taste complaint: Zinc is a component of gustin, the salivary protein needed for taste bud maturation. Without zinc the taste buds do not develop properly, so taste is blunted. That is **hypogeusia**.

Step 2 – take the healing complaint: Zinc is a cofactor for over 300 enzymes, including the DNA and RNA polymerases and the matrix metalloproteinases that remodel a healing wound.

Step 3 – link the two: Cells cannot divide quickly without those polymerases, so epithelium and granulation tissue are slow to cover the socket. Impaired wound healing follows.

Step 4 – recall the rest of the syndrome: Zinc deficiency also gives dermatitis around the mouth and anus, diarrhoea, alopecia, growth retardation and hypogonadism, the picture of acrodermatitis enteropathica.

Step 5 – confirm the fit: Hypogeusia plus poor healing is the classic pairing for **zinc**, and both features are ones a dentist meets directly.

Why the other options are wrong:

- A, iron: deficiency gives microcytic hypochromic anaemia, angular cheilitis, atrophic glossitis and koilonychia. It gives a sore tongue, not a loss of taste, and it does not classically delay wound healing.
- C, iodine: deficiency causes goitre, hypothyroidism and cretinism. It has no direct link with taste or healing.
- D, fluoride: deficiency raises caries risk. Excess causes fluorosis. Neither affects taste or wound healing.

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

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – induction and inhibition pull a drug level in opposite directions: The cytochrome P450 system in the smooth endoplasmic reticulum of the hepatocyte carries out phase I oxidation of most drugs. Its activity is not fixed.

Step 1 – define enzyme induction: An inducer acts on gene transcription so the liver makes **more enzyme protein**. More enzyme means faster metabolism, lower plasma drug level and loss of effect.

Step 2 – define enzyme inhibition: An inhibitor blocks the enzyme that already exists. Metabolism slows, the plasma level rises and toxicity follows.

Step 3 – read what the question describes: The dose has to be raised for the effect to be kept, so the second drug is being cleared *faster*. That is induction, not inhibition.

Step 4 – match rifampicin to that: Rifampicin is the classic strong **inducer** of CYP3A4. Phenobarbitone, phenytoin, carbamazepine, griseofulvin and chronic alcohol behave the same way.

Step 5 – note the time course, which confirms the mechanism: Induction takes days to build up, because new enzyme protein has to be synthesised, and it wears off over days after the drug stops. A simple active site block would act within hours. The question says long-term rifampicin, which fits induction.

Step 6 – note the clinical relevance: An inducer lowers the level of oral contraceptives and of warfarin. An inhibitor such as erythromycin, ketoconazole or cimetidine does the opposite and can push a patient into bleeding.

Why the other options are wrong:

- A, competition for the active site: is a form of **inhibition**. It would slow metabolism and raise the drug level, so the dose would need to be cut, not raised.
- B, destruction of the haem group: is suicide inhibition, seen with drugs such as chloramphenicol. Again this is inhibition, and it would raise the level of the second drug.
- D, blocking renal secretion: would reduce excretion and so **raise** the plasma level. This is the probenecid mechanism, and in any case the question names a hepatic CYP3A4 route, not a renal one.

Final Answer: Induction of CYP3A4 synthesis ⇒ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – every enzyme has an optimum pH set by where it works: An enzyme is only fully active over a narrow pH band, because pH controls the ionisation of the active site residues.

Step 1 – locate pepsin: Pepsin works in the lumen of the **stomach**.

Step 2 – recall the pH of that lumen: Parietal cells secrete hydrochloric acid, which takes gastric juice to a pH of about 1 to 2.

Step 3 – match the enzyme to its surroundings: An enzyme that has to digest protein in that lumen must be built to work in acid, so pepsin has an optimum of about **pH 1.5 to 2.0**.

Step 4 – link the acid to activation as well: Pepsin is secreted as the zymogen pepsinogen. Gastric acid cleaves an inhibitory peptide off pepsinogen to give active pepsin, and pepsin then activates more pepsinogen autocatalytically. So the same acid both activates the enzyme and provides its working pH.

Step 5 – follow it out of the stomach: Once chyme passes into the duodenum and pancreatic bicarbonate lifts the pH above about 5, pepsin is irreversibly inactivated and the pancreatic proteases take over.

Why the other options are wrong:

- A, pH 7.4: is the pH of blood and of the extracellular fluid, not of gastric juice. Pepsin is dead at this pH.
- B, pH 6.8: is close to the pH of resting saliva, which suits salivary amylase, not pepsin.
- C, pH 8.0 to 9.0: is the alkaline range that suits trypsin and chymotrypsin in the duodenum, and alkaline phosphatase. It is the opposite of what pepsin needs.

Final Answer: pH 1.5 to 2.0 $\Rightarrow$ D

Answer: (D) Go Back to Q4

Q5.

Solution

Concept – fluoride works on the bacterium as well as on the enamel: Caries needs plaque bacteria to ferment sugar into acid. Anything that slows that fermentation slows caries.

Step 1 – read the marked step from the diagram: X converts 2-phosphoglycerate into phosphoenolpyruvate.

Step 2 – name the enzyme of that step: That dehydration is catalysed by **enolase**.

Step 3 – state how fluoride blocks it: Fluoride together with magnesium and inorganic phosphate forms a fluorophosphate complex that ties up the magnesium enolase needs, so the enzyme stops working.

Step 4 – follow the consequence down the chain: With enolase blocked, phosphoenolpyruvate is not formed. No phosphoenolpyruvate means no pyruvate, and no pyruvate means no lactic acid.

Step 5 – add the second hit: Phosphoenolpyruvate is also the energy source for the bacterial phosphotransferase system, the pump that carries sugar into the cell. So blocking enolase starves the bacterium of the very molecule it needs to take up more sugar.

Step 6 – tie it to the clinic: Less acid means the plaque pH stays above the critical pH of about 5.5, so enamel is not demineralised. This anti-enzyme action sits alongside the better known formation of fluorapatite.

Why the other options are wrong:

- A, hexokinase: catalyses the first step of the chain, glucose to glucose-6-phosphate, at the top of the diagram. The marked step is far lower down, and hexokinase is not the fluoride target.
- C, lactate dehydrogenase: converts pyruvate into lactic acid, which is the **last** arrow in the diagram, not the marked one.
- D, aldolase: splits fructose-1,6-bisphosphate into triose phosphates, which sits in the upper "several steps" block, well before the marked arrow.

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

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – the shape of collagen dictates its sequence: Collagen is three polypeptide chains twisted together, and the geometry of that twist decides which amino acids are allowed where.

Step 1 – describe the architecture: Each alpha chain is a **left-handed** helix. Three of them wind around one another into a **right-handed** superhelix, which is

the triple helix in the figure.

Step 2 – look at the core, the position marked Y: Where the three chains cross, the space at the centre of the helix is extremely tight. Only a side chain of a single hydrogen atom will fit.

Step 3 – name the only amino acid that small: Glycine is the only one whose side chain is a hydrogen atom. So glycine must occupy every third residue.

Step 4 – write the repeat: The sequence therefore reads **Gly-X-Y** over and over, where X is frequently proline and Y is frequently hydroxyproline.

Step 5 – explain why proline and hydroxyproline sit there: The rigid ring of proline forces the individual chain into its left-handed twist, and hydroxyproline forms hydrogen bonds between adjacent chains that lock the triple helix together.

Step 6 – confirm from disease: If a bulkier amino acid replaces even one glycine, the helix cannot close at that point. That single substitution is the basis of osteogenesis imperfecta, which proves how strict the glycine rule is.

Why the other options are wrong:

- A, Pro-X-Y: puts proline at the crowded core. Proline has a bulky pyrrolidine ring and would not fit there. Proline is common, but at the X position, not at the core.
- B, Gly-Gly-Gly: an unbroken run of glycine would leave no room for the proline and hydroxyproline residues that give the helix its rigidity and its interchain hydrogen bonds.
- D, Lys-X-Y: lysine has a long charged side chain, far too big for the core. Lysine matters in collagen, but as the residue that is hydroxylated and then cross-linked between molecules, not as the repeat unit.

Final Answer: Gly-X-Y $\Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – replication needs a team of enzymes, each with one job: Name the job first, then the enzyme that does it.

Step 1 – set the scene: DNA polymerase can only add nucleotides in the 5' to 3' direction. The two parent strands run in opposite directions, so one new strand is made continuously and the other in short pieces.

Step 2 – name those pieces: The short pieces on the lagging strand are the **Okazaki fragments**.

Step 3 – follow what happens to them: Each fragment starts with an RNA primer. DNA polymerase I removes that primer and fills the gap with DNA. That leaves a break in the sugar-phosphate backbone, called a nick.

Step 4 – name the sealing enzyme: DNA ligase forms the phosphodiester bond across the nick, joining one fragment to the next and making the lagging strand continuous.

Step 5 – confirm from the question wording: The question already states that the primers are removed and the gaps are filled, so only the sealing job is left, and that is ligase alone.

Why the other options are wrong:

- B, DNA helicase: unwinds the double helix at the replication fork by breaking the hydrogen bonds between the strands. It opens DNA, it does not join it.
- C, DNA polymerase III: is the main enzyme that adds nucleotides and extends the new strand. It cannot make the final phosphodiester bond across a nick, which is exactly why ligase is needed.
- D, primase: is an RNA polymerase that lays down the short RNA primer at the start of each fragment. It works at the beginning of a fragment, not at the join.

Final Answer: DNA ligase $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – an open buffer beats a closed one: Buffer theory says a buffer is best when the pH is close to its pKa. The bicarbonate system breaks that rule, so something else must be carrying it.

Step 1 – state the apparent problem: The pKa is 6.1 and the blood pH is 7.4. The gap is 1.3 pH units, so on paper the pair is far from its best working point.

Step 2 – define a closed system: In a closed system, such as the phosphate buffer, the acid and the base forms simply interconvert. Once the base form is spent, buffering stops.

Step 3 – define what makes bicarbonate open: Its acid form is carbonic acid, which is in equilibrium with dissolved carbon dioxide, and carbon dioxide is a **gas**

that the lungs can remove from the body altogether.

Step 4 – follow an acid load through the system: Added H^+ is mopped up by bicarbonate to form carbonic acid, which becomes carbon dioxide and water. Ventilation then blows the carbon dioxide away, so the acid is physically removed rather than stored.

Step 5 – add the kidney: The kidney independently regenerates bicarbonate and excretes hydrogen ions, so the base half of the pair is topped up as fast as it is consumed.

Step 6 – put the two together: Because both halves of the pair are separately controlled and neither ever runs out, the effective buffering power is far greater than the pKa alone predicts. That is why bicarbonate accounts for the majority of extracellular buffering despite its unfavourable pKa.

Why the other options are wrong:

- A, a pKa shift to 7.4: does not happen. The pKa of the bicarbonate system stays at 6.1 in plasma. The whole point of the question is that the system works *despite* that value.
- B, highest concentration inside cells: is wrong on two counts. The dominant intracellular buffers are proteins, haemoglobin and phosphate, and in any case the question asks about the **extracellular** fluid.
- D, handling carbonic acid: is exactly backwards. Carbonic acid is one half of the bicarbonate pair itself, so the system cannot buffer it. Respiratory acid loads are buffered mainly by haemoglobin and other proteins.

Final Answer: It is an open system, with lungs and kidney adjusting the two halves independently $\Rightarrow$ C

Answer: (C) Go Back to Q8

Q9.

Solution

Concept – hydrogen and potassium trade places across the cell membrane: About 98 percent of body potassium sits inside cells, so tiny shifts across the membrane make large changes in the serum value.

Step 1 – start with the acid load: In acidosis the extracellular hydrogen ion concentration rises.

Step 2 – follow the hydrogen ion: Hydrogen moves down its gradient into the cell, where intracellular proteins buffer it. That is the arrow marked **X**.

Step 3 – apply electroneutrality: Hydrogen carries a positive charge. If positive charge enters the cell and nothing leaves, the cell interior would become positive, which cannot happen.

Step 4 – identify the ion that leaves: To keep the balance, **potassium moves out** of the cell into the extracellular fluid, as the second arrow shows.

Step 5 – read the serum result: Potassium has moved from the cells into the plasma, so the serum potassium **rises**. This is **hyperkalaemia**.

Step 6 – note the trap: Total body potassium may be normal or even low. The hyperkalaemia is a redistribution, not an excess, which is why correcting the acidosis can unmask a real potassium deficit.

Step 7 – reverse the logic to check: In alkalosis hydrogen leaves the cell and potassium moves in, giving hypokalaemia. The mirror image confirms the mechanism.

Why the other options are wrong:

- A, potassium follows hydrogen in: breaks electroneutrality. Two positive ions cannot both flood into the cell together with no anion, and the figure shows the arrows pointing in opposite directions.
- C, unchanged: ignores the exchange completely. The whole point is that potassium crosses the membrane into the plasma, where it is measured.
- D, acidosis suppresses aldosterone: is not the mechanism. Aldosterone acts over hours in the kidney, whereas the shift here happens in minutes at the cell membrane, and the direction given is wrong anyway.

Final Answer: Serum potassium rises $\Rightarrow$ B

Answer: (B) Go Back to Q9

Q10.

Solution

Concept – haemostasis runs in a fixed order, fastest first: The body stops bleeding in three steps, and they are arranged so the quickest mechanism buys time for the slowest.

Step 1 – the first stage, vascular spasm: The cut vessel constricts within seconds. This is partly a direct myogenic response to injury and partly local release of endothelin and of thromboxane A_2 and serotonin from platelets. The narrower lumen cuts blood flow immediately.

Step 2 – the second stage, the platelet plug, which is S: Collagen is now ex-

posed under the damaged endothelium. Platelets adhere to it through von Willebrand factor, become activated, change shape and release their granules, and then aggregate with one another through fibrinogen bridges. Within a few minutes a soft plug fills the gap. This is the stage marked **S** in the middle panel, where the platelets are drawn inside the vessel.

Step 3 – the third stage, coagulation: The cascade converts prothrombin to thrombin, and thrombin converts fibrinogen to fibrin. The fibrin mesh in the third panel traps red cells and stitches the soft platelet plug into a firm clot.

Step 4 – put them in order: Vascular spasm, then platelet plug, then coagulation. The order matches how fast each acts: seconds, then minutes, then several minutes more.

Step 5 – apply it to an extraction socket: The socket vessels constrict, platelets plug them, and a fibrin clot then organises into the granulation tissue that heals the socket. This is exactly why you tell the patient to bite firmly on a gauze pack rather than rinse. Losing that clot early is what produces a dry socket.

Why the other options are wrong:

- B: puts coagulation first. The cascade takes minutes to generate thrombin, so it cannot be the immediate response, and there would be nothing to reinforce.
- C: puts the platelet plug before vascular spasm. Spasm is myogenic and instantaneous, and it happens before platelets have had time to adhere and aggregate.
- D: puts coagulation at S, before the platelet plug. The fibrin mesh exists to reinforce a plug that is already there, so it cannot come first. The figure also shows platelets in the middle panel and fibrin only in the last one.

Final Answer: Vascular spasm, then platelet plug, then coagulation $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – von Willebrand factor has a foot in both camps: It is the one molecule that belongs to the platelet phase and to the coagulation phase at the same time.

Step 1 – where it comes from: Von Willebrand factor is a large multimeric glycoprotein made by endothelial cells and by megakaryocytes, and stored in the

Weibel-Palade bodies of endothelium and the alpha granules of platelets.

Step 2 – its first job, adhesion: When endothelium is stripped away, vWF binds the exposed subendothelial collagen at one end and the platelet GpIb receptor at the other. It is the glue that sticks the first layer of platelets to the wound under high shear.

Step 3 – its second job, carrying factor VIII: Free factor VIII in plasma has a half life of only a few hours. Bound to vWF it is protected from degradation, so a vWF deficiency drags the factor VIII level down with it.

Step 4 – read both jobs into the patient: Losing the adhesion job gives a **platelet type** bleed: mucosal bleeding, menorrhagia, easy bruising and a prolonged bleeding time. Losing the carrier job lowers factor VIII, which can also lengthen the aPTT. Both are present in this woman.

Step 5 – name the disease: This is **von Willebrand disease**, the commonest inherited bleeding disorder, autosomal in inheritance so men and women are affected alike. Desmopressin releases stored vWF from endothelium and is the usual cover for a dental extraction in the mild type.

Why the other options are wrong:

- A, converting fibrinogen to fibrin: is the job of **thrombin**, and the cross-linking that follows is done by factor XIII. Neither is vWF.
- B, a vitamin K dependent factor that activates protein C: mixes two things up. The vitamin K dependent factors are II, VII, IX and X plus proteins C and S, and protein C is activated by thrombin bound to thrombomodulin. vWF is not vitamin K dependent at all.
- C, activating plasminogen: describes tissue plasminogen activator, which belongs to **fibrinolysis**, that is clot breakdown. vWF helps build the plug, it does not dissolve it.

Final Answer: Platelet adhesion to collagen and carriage of factor VIII $\Rightarrow$ D

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – learn the autonomic system by outflow and by transmitter: Each division has its own exit from the central nervous system and its own chemical at the end of the line.

Step 1 – the parasympathetic outflow: It is **craniosacral**. The cranial part trav-

els in the oculomotor, facial, glossopharyngeal and vagus nerves, that is cranial nerves III, VII, IX and X. The sacral part leaves from S2, S3 and S4.

Step 2 – the sympathetic outflow: It is **thoracolumbar**, leaving the cord from T1 to L2 through the lateral horn.

Step 3 – the transmitter at the ganglion: Both divisions use **acetylcholine** at the ganglion, acting on nicotinic receptors. This is the same for sympathetic and parasympathetic, so it never separates them.

Step 4 – the transmitter at the target organ: The parasympathetic postganglionic fibre releases **acetylcholine** onto muscarinic receptors. The sympathetic postganglionic fibre releases **noradrenaline** onto adrenergic receptors. This is the step that separates the two.

Step 5 – note the two sympathetic exceptions: Sympathetic fibres to sweat glands are cholinergic, and the adrenal medulla is supplied directly by preganglionic fibres that release acetylcholine onto chromaffin cells, which then pour adrenaline into the blood.

Step 6 – check the option: Craniosacral parasympathetic with acetylcholine throughout, thoracolumbar sympathetic with acetylcholine at the ganglion and noradrenaline at the target, is exactly option A.

Why the other options are wrong:

- B: swaps the two outflows and then claims both use noradrenaline throughout. No ganglion in either division uses noradrenaline, and the outflows are the wrong way round.
- C: says both divisions leave in the thoracolumbar segments, which erases the craniosacral parasympathetic outflow entirely, and it reverses the transmitters as well.
- D: reverses both the outflows and the target transmitters, so it is wrong twice over.

Final Answer: Craniosacral parasympathetic, thoracolumbar sympathetic $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – salivary glands take orders from both divisions, and both increase secretion: This is the classic exception. Sympathetic and parasympathetic do not oppose each other here, but the saliva they produce is not the same.

Step 1 – trace the parasympathetic supply: The submandibular and sublingual glands are supplied through the chorda tympani branch of the facial nerve and the submandibular ganglion. The parotid is supplied through the glossopharyngeal nerve, the lesser petrosal nerve and the otic ganglion.

Step 2 – state what parasympathetic stimulation does: Acetylcholine acts on M3 receptors on the acinar cells, raising intracellular calcium. This drives heavy movement of water and electrolytes into the acinus.

Step 3 – add the blood flow effect: Parasympathetic fibres also release vasoactive intestinal peptide and drive kallikrein release, both of which cause **vasodilatation**. More blood flow supplies the plasma from which saliva is made.

Step 4 – combine steps 2 and 3: Heavy fluid secretion plus a rich blood supply gives **copious watery saliva**, low in protein. This is why atropine, a muscarinic blocker, dries the mouth.

Step 5 – now the sympathetic supply: Fibres arise from T1 to T3, relay in the superior cervical ganglion, and reach the glands along the arteries.

Step 6 – state what sympathetic stimulation does: Noradrenaline acts mainly on beta receptors to trigger **exocytosis of protein granules**, while alpha mediated vasoconstriction cuts the blood supply. Plenty of protein, very little water.

Step 7 – combine and compare: The result is **scanty viscous saliva rich in protein**. That is the dry sticky mouth of fear or anxiety, and it is exactly option D.

Why the other options are wrong:

- A: reverses the two. Parasympathetic gives the copious watery saliva, not the viscous one. This is the commonest error in this question.
- B: says both give copious watery saliva. Sympathetic stimulation gives a small volume, because alpha mediated vasoconstriction limits the water available.
- C: denies the parasympathetic supply, which is in fact the dominant one. Chorda tympani, glossopharyngeal, submandibular ganglion and otic ganglion all supply these glands.

Final Answer: Parasympathetic copious and watery, sympathetic scanty and viscous $\Rightarrow$ $\boxed{D}$

Answer: (D) Go Back to Q13

Q14.

Solution

Concept – each zone of the adrenal cortex makes its own class of steroid: Read the zone off the question and the hormone follows.

Step 1 – lay out the three zones from outside in: Zona glomerulosa makes mineralocorticoids, zona fasciculata makes glucocorticoids, zona reticularis makes androgens. The medulla, deeper still, makes catecholamines.

Step 2 – read the clue in the question: It names the **outermost** layer, so this is the zona glomerulosa, and it names a mineralocorticoid.

Step 3 – name the hormone: The mineralocorticoid of the zona glomerulosa is **aldosterone**.

Step 4 – name its target: Aldosterone acts on the principal cells of the **distal convoluted tubule and collecting duct**, which is what the question states.

Step 5 – state what it does there: It increases the number of epithelial sodium channels on the luminal side and of Na^+/K^+ ATPase pumps on the basal side. Sodium is reabsorbed, and water follows the sodium.

Step 6 – state what is lost in exchange: Sodium reabsorption leaves the lumen negative, which drives **potassium** out through the intercalated and principal cells and **hydrogen** out through the intercalated cells. So aldosterone retains sodium and excretes potassium and hydrogen.

Step 7 – confirm from the pathology: Conn syndrome, an aldosterone excess, gives hypertension, hypokalaemia and metabolic alkalosis. Addison disease, a deficiency, gives hyponatraemia, hyperkalaemia and hypotension. Both mirror the actions above.

Why the other options are wrong:

- B, cortisol from the zona fasciculata: is a glucocorticoid from the **middle** zone, and its actions are described backwards here. Cortisol has weak mineralocorticoid activity that retains sodium, it does not excrete it.
- C, dehydroepiandrosterone from the zona reticularis: is an adrenal androgen from the **innermost** cortical zone. It has no mineralocorticoid action on the distal tubule.
- D, adrenaline from the adrenal medulla: is a catecholamine, not a steroid, and the medulla is not part of the cortex at all. It acts within seconds on adrenergic receptors, not on distal tubular sodium handling.

Final Answer: Aldosterone from the zona glomerulosa ⇒ A

Answer: (A) Go Back to Q14

Answer Key

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