INI CET Physiology

Sample Paper – 10

Duration: 14 Minutes **Maximum Marks: 15**

Instructions

- This paper contains **15** Multiple Choice Questions (Single Best Response), modelled on the Physiology content of **INI CET** (Institutes of National Importance Combined Entrance Test) conducted by AIIMS New Delhi.
- The **15-question** length matches the number of Physiology items you actually meet in the real 200-question paper.
- Each correct answer carries **+1 mark**. There is **negative marking of one-third (0.33) 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 **14 minutes**, the pace the real computer-based test demands.

General, Nerve and Muscle Physiology

Q1. The Gibbs-Donnan equilibrium across a cell membrane arises because:

(A) the membrane pumps every ion at an equal rate in both directions

(B) the membrane is completely impermeable to all ions

(C) non-diffusible, negatively charged proteins confined to one side force an unequal distribution of the diffusible ions

(D) water is unable to cross the membrane

Q2. In cardiac muscle, the trigger for release of calcium from the sarcoplasmic reticulum during excitation-contraction coupling is:

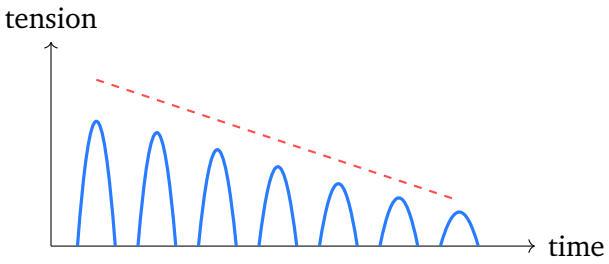
(A) direct mechanical coupling between the dihydropyridine and ryanodine receptors, exactly as in skeletal muscle

(B) depolarisation of the membrane alone, without any calcium entry

(C) entry of extracellular calcium through L-type channels, which then triggers further calcium release (calcium-induced calcium release)

(D) bulk release of calcium from the mitochondria

Q3. The tension record below shows a skeletal muscle fatiguing during repeated stimulation (the dashed line marks the falling peak force). The decline in force is best explained by:



(A) Depletion of ATP and phosphocreatine together with accumulation of lactate and H^+

(B) A rise in the resting membrane potential to $+30$ mV

(C) Increased acetylcholine release at the motor end plate

(D) An increase in the calcium stored in the sarcoplasmic reticulum

Blood and Body Fluids

Q4. Aggregation of platelets during primary haemostasis is actively promoted by which mediator released from the activated platelets themselves?

(A) Thromboxane A2

(B) Prostacyclin (PGI_2)

(C) Nitric oxide

(D) Antithrombin III

Q5. The intrinsic coagulation pathway is set off when which factor undergoes contact activation on a negatively charged surface (such as exposed collagen or glass)?

(A) Factor VII

(B) Factor XII (Hageman factor)

(C) Factor X

(D) Tissue factor (Factor III)

Cardiovascular System

Q6. Other factors remaining constant, an increase in total peripheral resistance will:

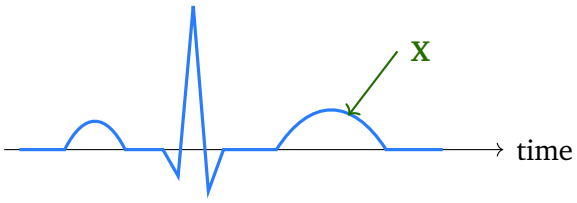
(A) decrease the mean arterial pressure

(B) leave the mean arterial pressure unchanged

(C) reduce cardiac output but not arterial pressure

(D) increase the mean arterial pressure

Q7. In the ECG trace below, the deflection marked **X** that follows the QRS complex represents:



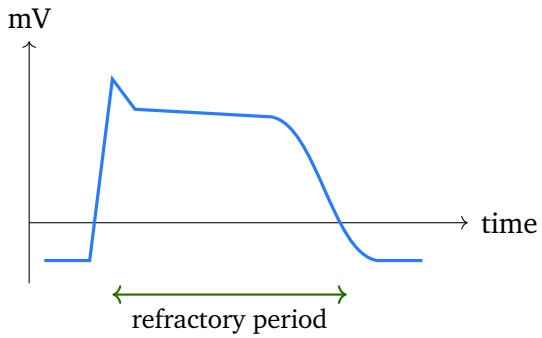
(A) Ventricular repolarisation

(B) Atrial depolarisation

(C) Ventricular depolarisation

(D) Atrial repolarisation

Q8. The cardiac action potential below carries a long refractory period spanning most of its duration (green arrow). This long refractory period is physiologically important because it:



(A) allows the heart muscle to be tetanised for a stronger contraction

(B) prevents tetanic (summated) contraction, so the heart can relax and refill between beats

(C) speeds conduction through the atrioventricular node

(D) has no meaningful effect on cardiac pumping

Respiratory System

Q9. Deficiency of pulmonary surfactant in a premature newborn produces neonatal respiratory distress syndrome mainly because surfactant normally:

(A) raises the surface tension inside the alveoli

(B) carries oxygen directly across the alveolar wall

(C) stimulates the medullary respiratory centre

(D) lowers alveolar surface tension and so prevents alveolar collapse (atelectasis)

Q10. The largest fraction of carbon dioxide is carried in the blood in which chemical form?

(A) Physically dissolved in the plasma

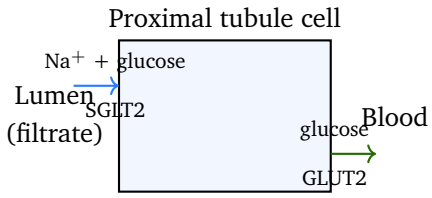
(B) Bound to haemoglobin as carbaminohaemoglobin

(C) As free carbonic acid inside the red cells

(D) As bicarbonate ions

Renal System

Q11. In the proximal tubule cell shown, glucose is reabsorbed from the filtrate across the apical (luminal) membrane mainly by:



(A) Simple diffusion straight down its own concentration gradient

(B) Sodium-glucose cotransport (SGLT2), driven secondarily by the inward sodium gradient

(C) Primary active transport that hydrolyses ATP directly on the glucose molecule

(D) Facilitated diffusion through GLUT2 placed on the apical membrane

Q12. The renal clearance of a substance is best defined as:

(A) the total amount of the substance filtered at the glomerulus each minute

(B) the volume of plasma completely cleared of the substance per unit time

(C) the concentration of the substance measured in the final urine

(D) the rate at which urine is produced by the kidney

Gastrointestinal, Endocrine and Neurophysiology

Q13. The same load of glucose given by mouth releases more insulin than when it is given intravenously. This incretin effect is produced largely by:

(A) glucose-dependent insulinotropic peptide (GIP) released from the gut, which augments insulin secretion

(B) glucagon secreted by the pancreatic alpha cells

(C) somatostatin secreted by the pancreatic delta cells

(D) cortisol released from the adrenal cortex

Q14. Antidiuretic hormone (vasopressin) is:

(A) made in the anterior pituitary and released from the posterior pituitary

(B) both made and released entirely by the anterior pituitary

(C) synthesised in the hypothalamic supraoptic nucleus and stored in, then released from, the posterior pituitary

(D) made in the adrenal medulla and stored in the hypothalamus

Q15. Body temperature is held around a regulated set-point. The integrating centre that acts as the body's thermostat is the:

(A) medulla oblongata

(B) cerebral cortex

(C) hypothalamus

(D) anterior pituitary

Detailed Solutions

Q1.

Solution

Concept – Gibbs-Donnan effect: It explains how a trapped charged species reshapes the distribution of the ions that can cross.

Step 1 – identify the trapped species: Cells hold large **negatively charged proteins** that cannot diffuse through the membrane.

Step 2 – work out the consequence: To keep each side electrically neutral, more diffusible cations are drawn to the protein side and diffusible anions are pushed out, so the diffusible ions end up **unequally distributed**.

Why the other options are wrong:

- A, equal pumping of every ion: describes active transport, not a passive Donnan equilibrium.
- B, impermeable to all ions: then no diffusible ion could redistribute at all.
- D, water cannot cross: water movement is osmosis, a separate consequence, not the cause.

Final Answer: Non-diffusible proteins force an unequal ion distribution $\Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – cardiac excitation-contraction coupling: Cardiac muscle needs extracellular calcium to start contraction, unlike skeletal muscle.

Step 1 – trace the trigger: During the plateau, Ca^{2+} enters through **L-type calcium channels**.

Step 2 – amplify the signal: This small influx opens ryanodine receptors on the sarcoplasmic reticulum, releasing a much larger store of calcium. This is **calcium-induced calcium release**.

Why the other options are wrong:

- A, direct mechanical coupling: that is the skeletal-muscle mechanism, which the question asks you to contrast.
- B, depolarisation alone: cardiac coupling cannot proceed without the calcium trigger.

- D, mitochondrial release: mitochondria buffer calcium but do not supply the contractile trigger.

Final Answer: Extracellular calcium entry triggering calcium-induced calcium release $\Rightarrow$ C

Answer: (C) Go Back to Q2

Q3.

Solution

Concept – muscle fatigue: The falling envelope of peak force signals failing energy supply.

Step 1 – read the trace: Each successive twitch peaks lower, and the dashed line shows the steady decline in force.

Step 2 – name the cause: Repeated activity **depletes ATP and phosphocreatine** while **lactate and H^+ accumulate**; the acid interferes with the contractile proteins and calcium handling, so force falls.

Why the other options are wrong:

- B, resting potential rising to $+30$ mV: not a feature of fatigue and not compatible with a resting cell.
- C, more acetylcholine: would tend to increase, not reduce, activation.
- D, more stored calcium: fatigue is associated with impaired calcium release, not larger stores.

Final Answer: ATP/phosphocreatine depletion with lactate and H^+ build-up $\Rightarrow$ A

Answer: (A) Go Back to Q3

Q4.

Solution

Concept – platelet aggregation mediators: Activated platelets release agents that recruit more platelets.

Step 1 – name the pro-aggregatory agent: Thromboxane A2, made by platelet cyclo-oxygenase, is a powerful stimulus for aggregation and vasoconstriction.

Step 2 – confirm the direction: It amplifies the growing platelet plug during primary haemostasis.

Why the other options are wrong:

- B, prostacyclin (PGI_2): from endothelium, it *inhibits* aggregation.
- C, nitric oxide: also inhibits platelet activation.
- D, antithrombin III: an anticoagulant that inactivates thrombin, not a platelet aggregator.

Final Answer: Thromboxane A2 $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – contact activation of the intrinsic pathway: The intrinsic pathway begins on a charged surface, without tissue factor.

Step 1 – name the initiating factor: Factor XII (Hageman factor) is activated on contact with a negatively charged surface such as exposed collagen.

Step 2 – follow the sequence: Factor XIIa then activates XI, which activates IX, carrying the cascade forward.

Why the other options are wrong:

- A, Factor VII: belongs to the extrinsic pathway.
- C, Factor X: sits on the shared common pathway, downstream of contact activation.
- D, tissue factor (III): triggers the extrinsic, not the intrinsic, pathway.

Final Answer: Factor XII $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – determinants of arterial pressure: Mean arterial pressure is the product of cardiac output and total peripheral resistance.

Step 1 – write the relationship: MAP $\approx$ cardiac output $\times$ total peripheral resistance.

Step 2 – apply the change: If resistance rises while cardiac output is held constant, the product rises, so **mean arterial pressure increases**.

Why the other options are wrong:

- A, decreased pressure: opposite of the relationship.
- B, unchanged: ignores the direct dependence of pressure on resistance.
- C, reduced cardiac output only: the question holds output constant and asks about pressure.

Final Answer: Mean arterial pressure increases $\Rightarrow$ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – reading the ECG: Each deflection maps to an electrical event of the cardiac cycle.

Step 1 – locate X: The rounded wave **X** comes right after the QRS complex; it is the **T wave**.

Step 2 – name the event: The T wave records **ventricular repolarisation**, as the ventricles reset electrically.

Why the other options are wrong:

- B, atrial depolarisation: that is the P wave, before the QRS.
- C, ventricular depolarisation: that is the QRS complex itself.
- D, atrial repolarisation: it is small and buried within the QRS, not the T wave.

Final Answer: Ventricular repolarisation $\Rightarrow$ A

Answer: (A) Go Back to Q7

Q8.

Solution

Concept – the long cardiac refractory period: The plateau keeps the muscle inexcitable for most of the contraction.

Step 1 – link duration to function: Because the refractory period lasts almost as long as the mechanical contraction, a second stimulus cannot land while the muscle is still contracting.

Step 2 – state the benefit: This **prevents tetanic (summated) contraction**, so

the heart relaxes and refills between beats and can work as a pump.

Why the other options are wrong:

- A, allows tetanus: a tetanised heart could not fill and would fail as a pump.
- C, speeds AV conduction: conduction speed is a separate property.
- D, no effect: the refractory period is central to rhythmic pumping.

Final Answer: Prevents tetanus so the heart can refill $\Rightarrow$ B

Answer: (B) Go Back to Q8

Q9.

Solution

Concept – pulmonary surfactant: Surfactant reduces the surface tension of the fluid lining the alveoli.

Step 1 – state the normal action: By **lowering alveolar surface tension**, surfactant keeps small alveoli open and stops them collapsing.

Step 2 – apply to the premature newborn: Without enough surfactant, surface tension is high, alveoli collapse (atelectasis), and the effort to breathe rises, producing neonatal respiratory distress syndrome.

Why the other options are wrong:

- A, raises surface tension: the opposite of what surfactant does.
- B, carries oxygen across the wall: gas crosses by diffusion, not on surfactant.
- C, stimulates the respiratory centre: surfactant has no such central role.

Final Answer: Lowers surface tension and prevents alveolar collapse $\Rightarrow$ D

Answer: (D) Go Back to Q9

Q10.

Solution

Concept – carbon dioxide transport: CO_2 travels in three forms, but one dominates.

Step 1 – recall the split: About 70% of CO_2 is carried as **bicarbonate ions**, roughly 20% as carbaminohaemoglobin, and about 7 to 10% dissolved.

Step 2 – explain the bicarbonate route: CO_2 enters red cells, carbonic anhydrase

makes carbonic acid, which dissociates to H^+ and HCO_3^-; the bicarbonate leaves into the plasma via the chloride shift.

Why the other options are wrong:

- A, dissolved: only a small fraction.
- B, carbaminohaemoglobin: an important but minority share.
- C, free carbonic acid: only a transient intermediate, not a storage form.

Final Answer: As bicarbonate ions $\Rightarrow$ D

Answer: (D) Go Back to Q10

Q11.

Solution

Concept – proximal tubular glucose reabsorption: Glucose is moved uphill using the energy stored in the sodium gradient.

Step 1 – name the apical carrier: The luminal membrane carries **SGLT2**, a sodium-glucose cotransporter.

Step 2 – explain the driving force: Na^+ flows down its gradient into the cell and drags glucose along with it (secondary active transport); glucose then leaves the basolateral side via GLUT2.

Why the other options are wrong:

- A, simple diffusion: glucose is reabsorbed against its gradient, so simple diffusion cannot do it.
- C, ATP directly on glucose: no pump hydrolyses ATP on glucose itself; the energy comes from the sodium gradient.
- D, apical GLUT2: GLUT2 sits on the *basolateral* membrane, not the apical one.

Final Answer: Sodium-glucose cotransport (SGLT2) $\Rightarrow$ B

Answer: (B) Go Back to Q11

Q12.

Solution

Concept – renal clearance: Clearance is a virtual volume, not a real amount or concentration.

Step 1 – state the definition: Clearance is the **volume of plasma completely freed of a substance per unit time**.

Step 2 – write the formula: Clearance = (urine concentration × urine flow) ÷ plasma concentration, which has units of volume per time.

Why the other options are wrong:

- A, amount filtered per minute: that is the filtered load, not clearance.
- C, urine concentration: only one term in the clearance equation.
- D, urine flow rate: again only one input, not the clearance itself.

Final Answer: Volume of plasma cleared per unit time ⇒ B

Answer: (B) Go Back to Q12

Q13.

Solution

Concept – the incretin effect: Gut hormones prime the pancreas before glucose even reaches it in force.

Step 1 – identify the incretin: GIP (glucose-dependent insulinotropic peptide), released from the duodenum in response to oral nutrients, augments insulin secretion.

Step 2 – explain the larger oral response: Because oral glucose releases GIP (and GLP-1) but intravenous glucose does not, the same load gives more insulin when taken by mouth.

Why the other options are wrong:

- B, glucagon: raises blood glucose, it is not the incretin that boosts insulin here.
- C, somatostatin: inhibits insulin secretion.
- D, cortisol: opposes insulin action and is not gut-derived.

Final Answer: GIP from the gut ⇒ A

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – where ADH is made and stored: Posterior pituitary hormones are made in the hypothalamus.

Step 1 – name the site of synthesis: ADH is synthesised by neurons of the **hypothalamic supraoptic nucleus** (with some from the paraventricular nucleus).

Step 2 – trace storage and release: The hormone travels down the axons and is **stored in the posterior pituitary**, from where it is released into the blood.

Why the other options are wrong:

- A, made in anterior pituitary: the anterior pituitary does not make ADH.
- B, wholly anterior pituitary: incorrect on both synthesis and release.
- D, adrenal medulla: that gland makes catecholamines, not ADH.

Final Answer: Made in the supraoptic nucleus, stored in the posterior pituitary $\Rightarrow$ C

Answer: (C) Go Back to Q14

Q15.

Solution

Concept – thermoregulation and the set-point: A control system needs a comparator that holds a reference value.

Step 1 – name the centre: The **hypothalamus** contains the thermoregulatory centre that acts as the body's thermostat.

Step 2 – explain the set-point: It compares actual core temperature with the set-point and drives shivering, sweating or vasomotor changes to correct any error.

Why the other options are wrong:

- A, medulla oblongata: controls cardiovascular and respiratory reflexes, not the thermostat.
- B, cerebral cortex: handles conscious behaviour, not the core set-point.
- D, anterior pituitary: an endocrine relay, not the temperature comparator.

Final Answer: Hypothalamus $\Rightarrow$ C

Answer: (C) Go Back to Q15

Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	C	2	C	3	A	4	A	5	B
6	D	7	A	8	B	9	D	10	D
11	B	12	B	13	A	14	C	15	C