

## NEET PG Physiology Sample Paper-5

Duration: 15 Minutes

Maximum Marks: 68

### Instructions

- This paper contains 17 Multiple Choice Questions.
- Each correct answer carries +4 mark. Incorrect answer: -1 marks. Only one correct option.
- Unattempted questions carry 0 marks.
- Use of mobile phones, smartwatches, or any electronic gadgets is strictly prohibited.

**Q1.** A clinical research team is studying the biophysical properties of a novel synthetic ion channel expressed in a mammalian cell line. Electrophysiological tracking shows that the channel is selectively permeable to a divalent cation  $X^{2+}$ . At a steady-state experimental temperature of  $37^{\circ}\text{C}$ , the intracellular concentration of  $X^{2+}$  is maintained at 12 mEq/L while the extracellular concentration is fixed at 1,200 mEq/L. Assuming the ion behaves ideally, calculate the precise equilibrium membrane potential ( $E_X$ ) required to halt the net flux of this cation across the lipid bilayer.

- (A) +30.5 mV
- (B) +61.0 mV
- (C) +122.0 mV
- (D) -61.0 mV

**Q2.** During an experimental patch-clamp assay analyzing skeletal muscle sarcolemmal fatigue dynamics, a scientist applies a specific pharmacological agent that selectively blocks the voltage-gated chloride channels (ClC-1). Which of the following electrical anomalies is most likely to be observed in the muscle fiber action potential propagation profile immediately following repetitive high-frequency stimulation?



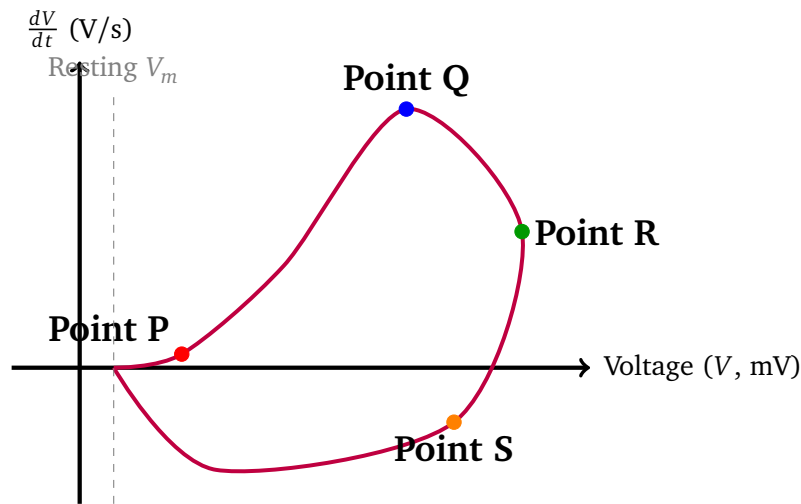
- (A) Accelerated repolarization with an exaggerated, shortened absolute refractory period
- (B) Persistent after-depolarization leading to spontaneous, repetitive runs of action potentials
- (C) Complete failure of the membrane to reach the firing threshold due to massive hyperpolarization
- (D) Prolonged plateau phase mimicking the mechanical properties of ventricular cardiomyocytes

**Q3.** An investigator constructs an artificial membrane containing a dense population of  $\text{Na}^+$ - $\text{K}^+$  ATPase pumps operating under standard physiological conditions. If the external medium is experimentally manipulated to completely deplete extracellular potassium ( $\text{K}^+$ ) while maintaining an optimal intracellular ATP concentration, which specific conformational state or step of the transport cycle will the pump freeze or accumulate into?

- (A) The unphosphorylated  $\text{E}_1$  state with high affinity for intracellular  $\text{Na}^+$  ions
- (B) The transient  $\text{E}_1$ -P state immediately prior to the occlusion of  $\text{Na}^+$
- (C) The phosphorylated  $\text{E}_2$ -P state with outward-facing ion-binding pockets
- (D) The dephosphorylated  $\text{E}_2$  state bound to intracellular ADP units

**Q4.** An automated microfluidic patch-clamp system records the membrane potential deviations of an isolated spherical node during a sequence of high-velocity current injections. Analyze the simulated phase-plane trace of the neuronal action potential cycle ( $\frac{dV}{dt}$  vs.  $V$ ) plotted below. Identify which specific marked region or intercept point precisely reflects the maximum rate of regenerative inward sodium flux ( $I_{\text{Na}}$  max):





- (A) Point P
- (B) Point Q
- (C) Point R
- (D) Point S

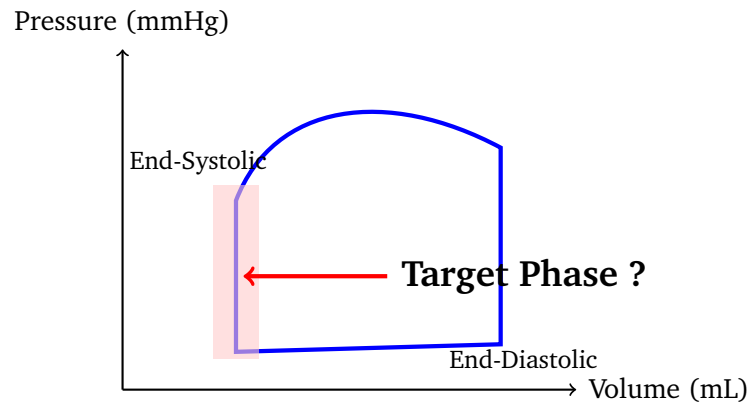
**Q5.** A 58-year-old male with severe calcific aortic stenosis undergoes an advanced cardiac catheterization procedure to evaluate left ventricular performance indices. During the assessment, a sudden transient baroreceptor-mediated burst causes an isolated, systemic peripheral vasoconstriction that acutely elevates the effective arterial elastance ( $E_A$ ) by 40%, while leaving the end-diastolic volume (EDV) and ventricular contractility ( $E_{ES}$ ) completely unaltered. Which of the following alterations will be visualized on the subsequent Left Ventricular Pressure-Volume loop?

- (A) A parallel leftward shift of the end-systolic pressure-volume relationship (ESPVR) line
- (B) An increase in stroke volume accompanied by a distinct reduction in the end-systolic volume
- (C) An increased end-systolic volume leading directly to a compromised, reduced stroke volume
- (D) A downward collapse of the diastolic filling curve along the passive compliance boundary



- Q6.** A clinical trial analyzes the cardiovascular responses to a novel selective  $I_f$  current inhibitor in patients experiencing refractory sinus tachycardia. The drug acts exclusively on the hyperpolarization-activated cyclic nucleotide-gated (HCN) channels within the sinoatrial node. Which of the following mechanical or electrical metrics will remain strictly unchanged following a therapeutic steady-state infusion of this agent?
- (A) The slope of the Phase 4 spontaneous diastolic depolarization phase
  - (B) The maximum negative diastolic potential achieved by the pacemaking cells
  - (C) The net duration of the total cardiac cycle ( $R - R$  interval length)
  - (D) The myocardial contractility ( $dP/dt$  max) of the working ventricular myocytes
- Q7.** During a high-altitude physiological simulation study, an investigator evaluates coronary hemodynamic autoregulation profiles. If the local partial pressure of oxygen ( $P_{O_2}$ ) drops acutely within a localized segment of the left anterior descending coronary territory, which of the following secondary downstream intracellular signaling loops is primarily responsible for inducing the requisite homeostatic metabolic arteriolar vasodilation?
- (A) Accumulation of interstitial adenosine leading to  $A_{2A}$  receptor activation and increased intracellular cAMP production
  - (B) Clearance of localized nitric oxide via endothelial activation causing a decrease in protein kinase G activity
  - (C) Hyperpolarization of vascular smooth muscle via the selective closure of ATP-sensitive  $K^+$  ( $K_{ATP}$ ) channels
  - (D) Activation of the endothelin-1 ( $ET_A$ ) G-protein coupled cascade promoting calcium-calmodulin binding
- Q8.** A clinical physiology laboratory maps the simultaneous mechanical, electrical, and acoustic events of the cardiac cycle. Identify the specific phase of the high-fidelity ventricular pressure-volume loop marked by the shaded rectangular box block highlighted in the schematic model diagram below:





- (A) Isovolumetric Contraction Phase
- (B) Rapid Ventricular Ejection Phase
- (C) Isovolumetric Relaxation Phase
- (D) Diastasis (Slow Ventricular Filling Phase)

- Q9.** A patient presenting with acute respiratory distress syndrome (ARDS) is placed on positive end-expiratory pressure (PEEP) mechanical ventilation. A bedside physiological calculation reveals a total physiological dead space ( $V_D/V_T$ ) ratio of 0.55. Concurrently, arterial blood gas analyses show an arterial  $P_{CO_2}$  ( $P_aCO_2$ ) of 48 mmHg. Based on the modified Bohr equation, what is the precise expected partial pressure of carbon dioxide measured in the mixed expired air ( $P_ECO_2$ ) collected from this patient?
- (A) 18.4 mmHg
  - (B) 21.6 mmHg
  - (C) 26.4 mmHg
  - (D) 31.2 mmHg

- Q10.** An experimental hyperbaric challenge tests the oxygen-hemoglobin dissociation dynamics of an individual possessing a rare hemoglobin variant characterized by a highly structurally stabilized R-state configuration. Which of the following physiological developments will manifest as a direct result of this structural variant when compared to normal adult hemoglobin (HbA)?
- (A) A pronounced rightward shift of the oxygen dissociation curve, facilitating systemic oxygen offloading at tissue levels

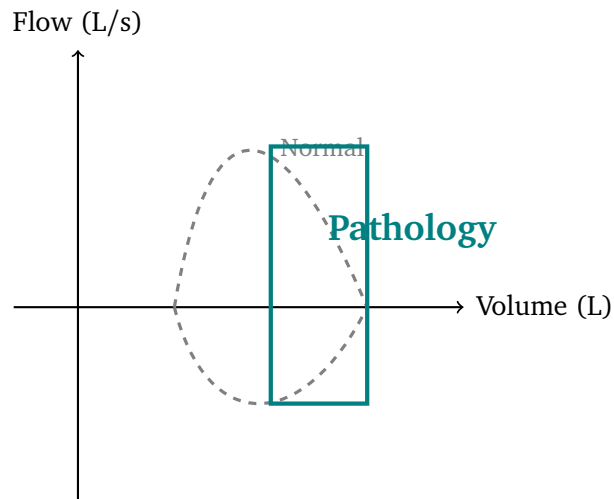
- (B) A severe decrease in the overall oxygen affinity of the molecule accompanied by an elevated  $P_{50}$  value
- (C) A distinct leftward shift of the curve, yielding an impaired, restricted unloading of oxygen in peripheral capillary systems
- (D) Complete elimination of the cooperative binding kinetics resulting in a strictly linear, non-sigmoidal curve profiles

**Q11.** A group of medical students are scaling an extreme mountain range without supplemental oxygen cylinders. At an altitude where the ambient atmospheric barometric pressure drops exactly to 380 mmHg, they measure their alveolar air composition. Assuming the fractional concentration of oxygen ( $F_{I}O_2$ ) remains at 21%, the respiratory quotient (R) is 0.8, and the alveolar  $P_{CO_2}$  ( $P_A CO_2$ ) is homeostatically driven down to 24 mmHg via hyperventilation, compute the true Alveolar  $P_{O_2}$  ( $P_A O_2$ ) given that water vapor pressure at body temperature is 47 mmHg.

- (A) 40.0 mmHg
- (B) 50.0 mmHg
- (C) 60.0 mmHg
- (D) 70.0 mmHg

**Q12.** A pulmonary fellow utilizes a comprehensive body plethysmograph system to evaluate a series of flow-volume dynamic contours. Analyze the spirometric loop topology illustrated below, which shows a baseline normal trace against an unknown pathological manifestation:





Based on the distinct flat-cutoff configuration affecting both the expiratory and inspiratory maxima, select the most accurate diagnosis represented by this dynamic configuration:

- (A) Severe pulmonary emphysema with premature dynamic airway collapse
- (B) Fixed upper airway obstruction (e.g., subglottic tracheal stenosis)
- (C) Variable extrathoracic obstruction (e.g., unilateral vocal cord paralysis)
- (D) Idiopathic pulmonary fibrosis with highly restricted parenchymal lung compliance

**Q13.** An intensive care patient with septic shock displays a sudden drop in urine output. To calculate the renal plasma flow (RPF), the medical team conducts a continuous baseline para-aminohippuric acid (PAH) infusion. The measured plasma concentration of PAH is 0.02 mg/mL, the urine concentration is 12.0 mg/mL, and the urine flow rate is 1.5 mL/min. If the patient's concurrent hematocrit reading is exactly 50%, what is the true calculated Total Renal Blood Flow (RBF) assuming a standard PAH extraction ratio ( $E_{\text{PAH}}$ ) of 0.90?

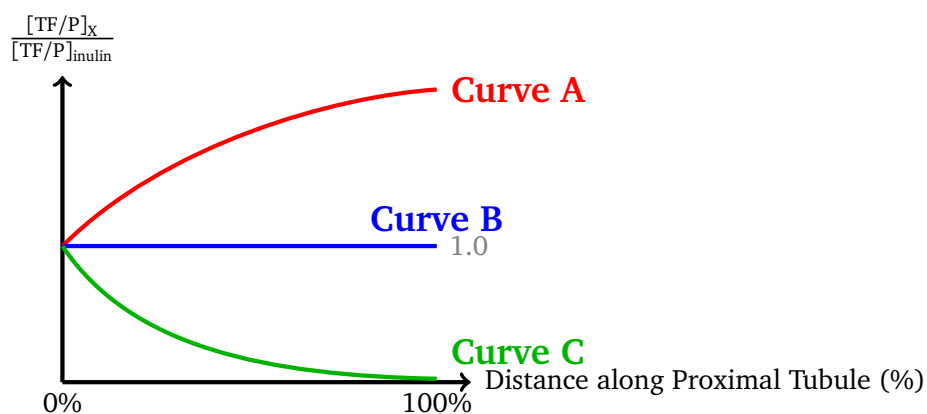
- (A) 500 mL/min
- (B) 900 mL/min
- (C) 1,000 mL/min
- (D) 2,000 mL/min



**Q14.** A transgenic mouse model exhibits a complete loss-of-function mutation in the gene encoding the luminal  $\text{Na}^+ - \text{H}^+$  Exchanger 3 (NHE3) within the proximal convoluted tubule cells. Which of the following explicit renal transport or systemic acid-base abnormalities will be characteristically present in this animal model?

- (A) Severe metabolic alkalosis due to unregulated hypersecretion of hydrogen ions
- (B) Profound metabolic acidosis associated with a massive wasting of filtered bicarbonate ions
- (C) Marked hyperkalemia driven by the secondary activation of distal cortical intercalated intercalated discs
- (D) Complete cessation of glucose reabsorption due to downstream down-regulation of SGLT2 complexes

**Q15.** An advanced nephron modeling software correlates solute concentration parameters along different anatomic sub-segments of the renal tubular architecture. Examine the classic clearance ratio profile graph ( $[\text{TF}/\text{P}]_{\text{solute}}/[\text{TF}/\text{P}]_{\text{inulin}}$  vs. Nephron length) displayed below:



Which option accurately identifies the physiological solutes whose handling matches **Curve A** and **Curve C** respectively?

- (A) Curve A = Glucose; Curve C = Bicarbonate
- (B) Curve A = Inulin; Curve C = Sodium
- (C) Curve A = Para-aminohippuric acid (PAH); Curve C = Glucose

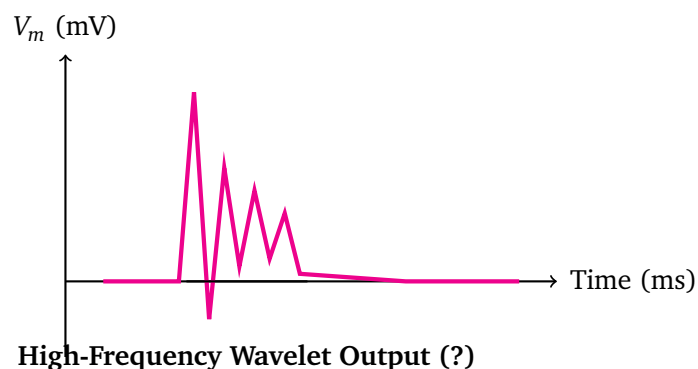


(D) Curve A = Sodium; Curve C = Chloride

**Q16.** A 34-year-old female presents with severe muscle weakness, recurrent polyuria, and resistant hypertension. Laboratory assays establish markedly elevated plasma aldosterone levels accompanied by an undetectable, suppressed plasma renin activity (PRA). A therapeutic trial of an epithelial sodium channel (ENaC) blocker is initiated. Which of the following cell populations located inside the renal parenchymal tissue is directly driven to hypersecrete potassium ions under the initial primary pathophysiological mechanism of this condition?

- (A) Macula densa cells of the juxtaglomerular apparatus
- (B) Principal cells of the cortical collecting duct system
- (C) Type A intercalated cells of the outer medullary collecting tubule
- (D) Podocyte foot processes covering the glomerular capillary loops

**Q17.** Neurophysiologists recording intracellular potentials from a Purkinje cell in the cerebellar cortex observe distinctive electrical responses during motor learning training protocols. The diagram below represents an extracellular and intracellular dual-channel tracking layout of a specific firing pattern:



Which specific afferent neural pathway projecting directly into the cerebellar architecture is responsible for generating this unique high-amplitude "Complex Spike" waveform structural event?

- (A) Mossy fibers originating from the pontine nuclei activating granule cell pathways



- (B) Climbing fibers originating exclusively from the contralateral inferior olivary nucleus
- (C) Parallel fibers forming excitatory glutamatergic synapses on Purkinje dendrites
- (D) Inhibitory GABAergic projections originating within the basket cell network



## Detailed Solutions

Q1.

## Solution

**Concept:** The equilibrium membrane potential ( $E_X$ ) for a selectively permeable ion is calculated using the Nernst equation. For any ion, the potential represents the electrical force required to exactly balance the chemical concentration gradient, resulting in zero net flux across the membrane.

**Solution:**

Let's calculate the precise equilibrium potential using the Nernst equation:

$$E_X = \frac{RT}{zF} \ln \left( \frac{[X^{2+}]_{\text{outside}}}{[X^{2+}]_{\text{inside}}} \right)$$

At 37°C (310.15 K), the constant factor using base-10 logarithms ( $2.303 \times \frac{RT}{F}$ ) simplifies to approximately 61.5 mV. Dividing by the valence ( $z$ ) of the cation:

- (a) The valence  $z$  for the divalent cation  $X^{2+}$  is +2.  
 (b) The concentration ratio is given by:

$$\frac{[X^{2+}]_{\text{outside}}}{[X^{2+}]_{\text{inside}}} = \frac{1,200 \text{ mEq/L}}{12 \text{ mEq/L}} = 100$$

- (c) Substituting these values into the simplified Nernst formula:

$$E_X = \frac{61.0 \text{ mV}}{2} \log_{10}(100) = 30.5 \text{ mV} \times 2 = +61.0 \text{ mV}$$

Since the extracellular concentration is substantially higher than the intracellular concentration, the interior of the cell must be electrically positive to repel the chemical influx of the positive  $X^{2+}$  ions.

**Final Answer:** +61.0 mV

**Answer: (B)**

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Q2.

**Solution**

**Concept:** In skeletal muscle sarcolemma, voltage-gated chloride channels (ClC-1) provide a massive resting chloride conductance ( $G_{Cl}$ ), contributing up to 70–80% of the total resting membrane stability. This dense chloride influx serves to rapidly clamp the membrane potential back down toward its resting level during repetitive, high-frequency motor unit firings.

**Solution:**

Let's analyze the pathophysiology of blocking ClC-1 channels:

- (a) During high-frequency physiological stimulation, potassium ions ( $K^+$ ) rapidly accumulate inside the narrow transverse tubule (T-tubule) network, which systematically depolarizes the local sarcolemmal membrane.
- (b) Under normal conditions, the baseline open ClC-1 channels counteract this localized accumulation-induced depolarization.
- (c) Pharmacological blockade of ClC-1 removes this critical electrical buffer. Consequently, the accumulated T-tubular potassium induces a persistent after-depolarization. This baseline depolarization keeps voltage-gated sodium channels transitioning through recovery states, resulting in spontaneous, repetitive runs of action potentials (myotonia).

**Final Answer:**

Persistent after-depolarization leading to spontaneous, repetitive runs of action potentials

**Answer: (B)**[Go Back to Question 2](#)

Q3.

**Solution**

**Concept:** The  $\text{Na}^+\text{-K}^+$  ATPase pump operates via a structural Albers-Post ( $\text{E}_1\text{-E}_2$ ) conformational cycle. The  $\text{E}_1$  conformation faces internally with high affinity for three  $\text{Na}^+$  ions, leading to phosphorylation ( $\text{E}_1\text{-P}$ ). This transforms into the outward-facing  $\text{E}_2\text{-P}$  state, which releases  $\text{Na}^+$  and displays high affinity for two external  $\text{K}^+$  ions.

**Solution:**

Let's evaluate the molecular transport mechanics when extracellular potassium is absent:

- (a) Intracellular ATP is available, allowing normal binding of internal  $\text{Na}^+$ , phosphorylation of the pump, and its subsequent conversion to the outward-facing  $\text{E}_2\text{-P}$  conformation.
- (b) Once in the  $\text{E}_2\text{-P}$  state, the pump releases its three bound  $\text{Na}^+$  ions into the external medium.
- (c) However, dephosphorylation of  $\text{E}_2\text{-P}$  back to the unphosphorylated  $\text{E}_2$  and  $\text{E}_1$  states strictly requires the binding of extracellular  $\text{K}^+$  ions. Because the extracellular medium is completely depleted of  $\text{K}^+$ , the transport mechanism is broken and stalls cleanly at this stage.

**Final Answer:** The phosphorylated  $\text{E}_2\text{-P}$  state with outward-facing ion-binding pockets

**Answer: (C)**

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Q4.

**Solution**

**Concept:** A phase-plane trajectory tracks the rate of change of membrane voltage ( $\frac{dV}{dt}$ ) directly against the instantaneous absolute voltage ( $V$ ). Because the total membrane current during the upstroke is dominated by ionic current, the peak vertical amplitude ( $\frac{dV}{dt}$  max) is mathematically proportional to the maximum net inward current flowing across the capacitive bilayer.

**Solution:**

Let's analyze the coordinate features along the phase-loop curve:

- (a) **Point P** represents the early voltage threshold step where the loop leaves the horizontal axis as voltage-gated sodium channels initially open.
- (b) **Point Q** marks the global maximum vertical point on the trajectory curve ( $\frac{dV}{dt}$  max). At this spatial point, the rate of membrane depolarization reaches its absolute peak velocity, driven directly by the maximum rate of regenerative inward  $\text{Na}^+$  flux ( $I_{\text{Na}}$  max).
- (c) **Point R** represents the peak action potential amplitude (overshoot potential), where  $\frac{dV}{dt}$  falls back to zero before switching polarity.
- (d) **Point S** marks the maximum rate of repolarization mediated by outward potassium channels.

**Final Answer:**

**Answer: (B)**

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Q5.

**Solution**

**Concept:** Effective arterial elastance ( $E_A$ ) serves as a comprehensive structural measure of ventricular afterload, geometrically represented on a Pressure-Volume (PV) loop as the negative slope of a line extending from the end-diastolic volume point on the horizontal axis to the end-systolic coordinate point.

**Solution:**

Let's analyze the homeostatic shift in the loop properties under the given experimental parameters:

- (a) Because the end-systolic pressure-volume relationship (ESPVR) is a fundamental metric of intrinsic contractility ( $E_{ES}$ ), and contractility is explicitly unchanged, the linear slope of the ESPVR line remains locked in its baseline position.
- (b) An acute 40% elevation in  $E_A$  represents a severe increase in ventricular afterload. This forces the ventricle to meet the fixed ESPVR line at a higher end-systolic coordinate point.
- (c) This shift directly expands the dynamic **End-Systolic Volume (ESV)**. Because the End-Diastolic Volume (EDV) remains entirely constant, the absolute Stroke Volume ( $SV = EDV - ESV$ ) must decrease.

**Final Answer:**

An increased end-systolic volume leading directly to a compromised, reduced stroke volume

**Answer: (C)**[Go Back to Question 5](#)

Q6.

**Solution**

**Concept:** The hyperpolarization-activated cyclic nucleotide-gated (HCN) channels carry the funny current ( $I_f$ ), which dictates the rate of Phase 4 spontaneous diastolic depolarization in pacemaking tissues. Ivabradine or selective  $I_f$  inhibitors slow the pacemaker firing rate without directly modifying other cardiac currents.

**Solution:**

Let's examine how this selective current inhibition impacts cardiodynamic variables:

- (a) Inhibiting  $I_f$  flattens the Phase 4 depolarization slope, thereby lengthening the time required to reach the action potential threshold, which lengthens the overall  $R - R$  interval.
- (b) Because HCN channels are predominantly operational in the specialized pacemaking tissues (SA and AV nodes) during diastole, blocking them does not alter working myocardial calcium release cycles.
- (c) Consequently, the contractility of the working ventricular myocytes—measured via the peak rate of systolic pressure development ( $\frac{dP}{dt}_{\max}$ )—is left completely intact and unchanged.

**Final Answer:** The myocardial contractility ( $\frac{dP}{dt}_{\max}$ ) of the working ventricular myocytes.

**Answer: (D)**

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Q7.

**Solution**

**Concept:** Coronary autoregulation is highly responsive to metabolic demands. When tissue hypoxia occurs due to a drop in local vascular  $P_{O_2}$ , myocytes rapidly hydrolyze adenosine triphosphate (ATP) down to adenosine, which diffuses out into the local interstitial microenvironment to initiate vasodilation.

**Solution:**

Let's map the downstream metabolic signaling cascade:

- (a) The accumulated extracellular adenosine binds to vascular smooth muscle  $A_{2A}$  G-protein coupled receptors.
- (b) This receptor activation stimulates adenylyl cyclase, driving an increase in intracellular cyclic adenosine monophosphate (cAMP) levels.
- (c) Elevated cAMP activates protein kinase A (PKA), which promotes smooth muscle hyperpolarization and relaxation, ensuring homeostatic local hyperemic flow matching the hypoxic stress.

**Final Answer:**

Accumulation of interstitial adenosine leading to  $A_{2A}$  receptor activation and increased intracellular cAMP production.

**Answer: (A)**[Go Back to Question 7](#)

Q8.

**Solution**

**Concept:** A Left Ventricular Pressure-Volume loop represents the sequential opening and closing of the cardiac valves during a single complete cardiac cycle, dividing it into four distinct mechanical operational phases.

**Solution:**

Let's trace the loop starting from the bottom right corner clockwise:

- (a) From the maximum volume point (End-Diastolic Volume), the mitral valve closes, and the vertical ascending segment represents **Isovolumetric Contraction**.
- (b) When ventricular pressure exceeds aortic pressure, the aortic valve opens, tracing the upper curved path of **Ventricular Ejection**.
- (c) At the top left coordinate point, the aortic valve snaps shut (End-Systolic Volume). The subsequent near-vertical descending drop highlighted by the shaded rectangular box block shows ventricular pressure falling rapidly while total intraventricular volume remains strictly fixed.
- (d) This precise segment represents the **Isovolumetric Relaxation Phase**, preceding the opening of the mitral valve for diastolic filling.

**Final Answer:**

**Answer:**

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Q9.

**Solution****Concept:**

The physiological dead space ratio ( $V_D/V_T$ ) quantifies the fraction of the total tidal volume that does not participate in respiratory gas exchange. It can be calculated using the modified Bohr equation (Enghoff modification), which uses arterial and mixed expired carbon dioxide tensions.

**Solution:**

Let's write out the modified Bohr equation format and solve for  $P_E\text{CO}_2$ :

$$\frac{V_D}{V_T} = \frac{P_a\text{CO}_2 - P_E\text{CO}_2}{P_a\text{CO}_2}$$

Given parameters:  $\frac{V_D}{V_T} = 0.55$  and  $P_a\text{CO}_2 = 48$  mmHg. Substituting these into the formula:

(a) Multiply both sides by  $P_a\text{CO}_2$ :

$$0.55 \times 48 = 48 - P_E\text{CO}_2$$

(b) Compute the left side value:

$$26.4 = 48 - P_E\text{CO}_2$$

(c) Rearrange the equation to isolate  $P_E\text{CO}_2$ :

$$P_E\text{CO}_2 = 48 - 26.4 = 21.6 \text{ mmHg}$$

**Final Answer:**

**Answer: (B)**

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Q10.

**Solution**

**Concept:** Hemoglobin transitions between two structural states: the T-state (Tense, low oxygen affinity) and the R-state (Relaxed, high oxygen affinity). Stabilizing the high-affinity R-state locks the molecule in its oxygen-binding conformation.

**Solution:**

Let's analyze the physiological consequences of a structurally stabilized R-state configuration:

- (a) Because the R-state is locked or heavily favored, hemoglobin binds oxygen with extremely high affinity, meaning it requires a much lower partial pressure of oxygen to achieve 50% saturation (a reduced  $P_{50}$  value).
- (b) This high affinity produces a distinct **leftward shift** of the oxygen dissociation curve.
- (c) While oxygen loading in the lungs remains highly efficient, the tightly bound oxygen cannot be easily offloaded or released in the lower oxygen environments of peripheral capillary tissue beds, severely limiting tissue oxygenation.

**Final Answer:**

A distinct leftward shift of the curve, yielding an impaired, restricted unloading of oxygen in peripheral capillary systems

**Answer: (C)**

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Q11.

**Solution**

**Concept:** The composition of alveolar oxygen ( $P_AO_2$ ) is calculated using the Alveolar Gas Equation, which accounts for the drop in oxygen partial pressure due to humidification in the airways and the respiratory exchange ratio in the alveoli.

**Solution:**

Let's state the Alveolar Gas Equation formula:

$$P_AO_2 = [(P_{\text{atm}} - P_{H_2O}) \times F_{IO_2}] - \frac{P_ACO_2}{R}$$

Given values:  $P_{\text{atm}} = 380$  mmHg,  $P_{H_2O} = 47$  mmHg,  $F_{IO_2} = 0.21$ ,  $P_ACO_2 = 24$  mmHg, and  $R = 0.8$ .

(a) Calculate the inspired oxygen partial pressure ( $P_{IO_2}$ ):

$$P_{IO_2} = (380 - 47) \times 0.21 = 333 \times 0.21 = 69.93 \approx 70.0 \text{ mmHg}$$

(b) Calculate the carbon dioxide metabolic correction factor:

$$\frac{P_ACO_2}{R} = \frac{24}{0.8} = 30.0 \text{ mmHg}$$

(c) Subtract the values to find  $P_AO_2$ :

$$P_AO_2 = 70.0 \text{ mmHg} - 30.0 \text{ mmHg} = 40.0 \text{ mmHg}$$

**Final Answer:**

**Answer:**

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Q12.

**Solution**

**Concept:** Flow-volume loops provide structural information regarding airway resistance and compliance. A flattening or severe truncation of specific sectors of the flow profile signifies the anatomical location and mechanical flexibility of an obstruction.

**Solution:**

Let's analyze the visual morphology of the pathological trace:

- (a) The pathological curve features a strict, equal truncation (flattening) affecting both the maximal expiratory flow and maximal inspiratory flow limits.
- (b) This symmetrical flat-cutoff profile is characteristic of a **fixed upper airway obstruction**. Because the obstruction cannot expand or compress during different phases of respiration, it restricts flow equally during both expiration and inspiration.
- (c) Examples include mature structural lesions like subglottic tracheal stenosis or fixed foreign objects. (Variable obstructions affect only one limb, while restrictive conditions reduce volume dimensions while preserving high proportional expiratory peak flows).

**Final Answer:** Fixed upper airway obstruction (e.g., subglottic tracheal stenosis)

**Answer: (B)**

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Q13.

**Solution**

**Concept:** Renal Blood Flow (RBF) is calculated by first determining the effective renal plasma flow via para-aminohippuric acid (PAH) clearance, correcting for its extraction efficiency ( $E_{PAH}$ ), and then accounting for the cellular fraction (hematocrit).

**Solution:**

Let's systematically solve the multi-step calculation:

- (a) Calculate the clearance of PAH ( $C_{PAH}$ ) using the mass balance formula:

$$C_{PAH} = \frac{U_{PAH} \times V}{P_{PAH}} = \frac{12.0 \text{ mg/mL} \times 1.5 \text{ mL/min}}{0.02 \text{ mg/mL}} = \frac{18}{0.02} = 900 \text{ mL/min}$$

- (b) Correct for the true Renal Plasma Flow (RPF) using the extraction ratio ( $E_{PAH} = 0.90$ ):

$$RPF = \frac{C_{PAH}}{E_{PAH}} = \frac{900 \text{ mL/min}}{0.90} = 1,000 \text{ mL/min}$$

- (c) Calculate the total Renal Blood Flow (RBF) by accounting for the hematocrit (Hct = 50%):

$$RBF = \frac{RPF}{1 - \text{Hct}} = \frac{1,000 \text{ mL/min}}{1 - 0.50} = \frac{1,000}{0.50} = 2,000 \text{ mL/min}$$

**Final Answer:**

**Answer: (D)**

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Q14.

**Solution**

**Concept:** The luminal  $\text{Na}^+\text{-H}^+$  Exchanger 3 (NHE3) in proximal tubule cells plays a major role in both sodium reabsorption and bicarbonate reclamation. NHE3 secretes  $\text{H}^+$  ions into the tubular lumen, where they combine with filtered  $\text{HCO}_3^-$  to allow its reabsorption.

**Solution:**

Let's analyze the effects of a complete loss-of-function mutation in NHE3:

- (a) Without functional NHE3, proximal hydrogen ion secretion drops drastically. As a result, filtered bicarbonate cannot combine with  $\text{H}^+$  to form carbonic acid in the lumen, halting its reabsorption.
- (b) This leads to a massive wasting of filtered bicarbonate ions in the urine, a condition analogous to Proximal (Type 2) Renal Tubular Acidosis.
- (c) The loss of systemic bicarbonate buffer stores directly causes profound metabolic acidosis.

**Final Answer:**

Profound metabolic acidosis associated with a massive wasting of filtered bicarbonate ions

**Answer: (B)**[Go Back to Question 14](#)

Q15.

### Solution

**Concept:** The fraction  $\frac{[TF/P]_{\text{solute}}}{[TF/P]_{\text{inulin}}}$  normalizes solute clearance against water reabsorption along the proximal tubule. If this ratio remains at 1.0, the solute is reabsorbed at a rate exactly matching water reabsorption (e.g., sodium). If it rises above 1.0, the solute is net secreted. If it falls below 1.0, the solute is reabsorbed more rapidly than water.

**Solution:**

Let's evaluate the three profiles illustrated in the graph:

- (a) **Curve A** starts at 1.0 and rises steeply. This signifies a substance that is actively and highly secreted into the early lumen, such as para-aminohippuric acid (PAH).
- (b) **Curve B** remains flat at 1.0, matching sodium ( $\text{Na}^+$ ) or total osmolarity, which are reabsorbed isosmotically with water.
- (c) **Curve C** drops rapidly down toward zero. This reflects solutes that are avidly reabsorbed in the first half of the proximal tubule via specialized co-transporters, such as glucose or amino acids.

**Final Answer:** Curve A = Para-aminohippuric acid (PAH); Curve C = Glucose

Answer: (C)

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Q16.

### Solution

**Concept:** Primary hyperaldosteronism (Conn's syndrome) presents with hypertension, hypokalemia, and metabolic alkalosis due to autonomous oversecretion of aldosterone from the adrenal cortex, which suppresses renin activity via negative feedback.

**Solution:**

Let's localize the cellular site of aldosterone action within the nephron:

- (a) Aldosterone acts primarily on the **\*\*principal cells of the cortical collecting duct\*\***.
- (b) It binds to intracellular mineralocorticoid receptors, increasing the expression and activity of apical epithelial sodium channels (ENaC) and basolateral  $\text{Na}^+$ - $\text{K}^+$  ATPase pumps.
- (c) The resulting increase in sodium reabsorption generates a lumen-negative electrical potential that drives the hypersecretion of potassium ( $\text{K}^+$ ) ions through apical ROMK channels into the urine, causing systemic hypokalemia.

**Final Answer:** Principal cells of the cortical collecting duct system

Answer: (B)

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Q17.

**Solution**

**Concept:** Purkinje cells receive two primary excitatory inputs: mossy fibers (via granule cells and parallel fibers) and climbing fibers. These pathways produce distinct electrophysiological waveforms.

**Solution:**

Let's analyze the properties of the complex spike waveform:

- (a) Parallel fibers fire at high frequencies to produce standard, uniform "Simple Spikes."
- (b) Conversely, **climbing fibers**, which originate exclusively from the contralateral inferior olivary nucleus, form highly branched, powerful synapses directly on a single Purkinje cell dendrite tree.
- (c) A single action potential from a climbing fiber delivers a prolonged depolarizing burst that triggers a high-amplitude initial action potential followed by a characteristic train of high-frequency small-amplitude wavelets. This distinct electrical signature is known as a **Complex Spike**.

**Final Answer:** Climbing fibers originating exclusively from the contralateral inferior olivary nucleus

**Answer: (B)**

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## Answer Key

Q	Ans	Q	Ans	Q	Ans	Q	Ans	Q	Ans
1	B	2	B	3	C	4	B	5	C
6	D	7	A	8	C	9	B	10	C
11	A	12	B	13	D	14	B	15	C
16	B	17	B						

