

NEET PG Physiology Sample Paper-9

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 24-year-old medical student is researching the transport kinetics of a novel therapeutic peptide across cell membranes. She observes that the intracellular-to-extracellular concentration ratio stabilizes at a value far exceeding electrochemical equilibrium. However, when the cell is exposed to a competitive inhibitor of mitochondrial complex IV, the transport rate drops to zero, and the accumulation gradient collapses. Which of the following thermodynamic equations properly defines the transport system's maximum work capacity per mole under active states, where R is the universal gas constant, T is temperature, F is Faraday's constant, and z is valence?

(A) $\Delta G = RT \ln \left(\frac{[C_{in}]}{[C_{out}]} \right) + zF \Delta \Psi$

(B) $\Delta G = RT \ln \left(\frac{[C_{out}]}{[C_{in}]} \right) - zF \Delta \Psi$

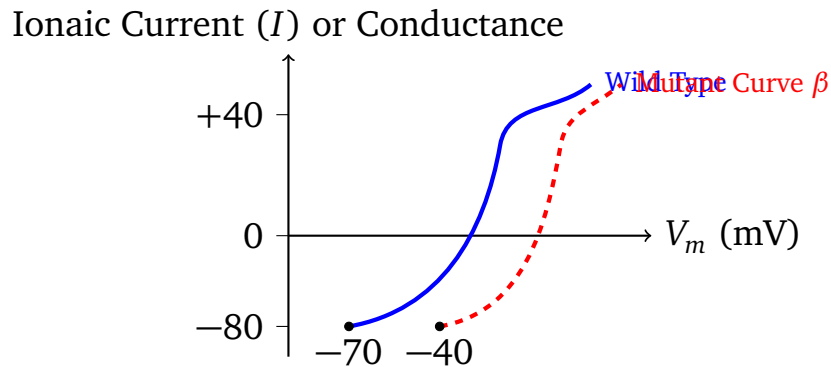
(C) $\Delta G = zF \Delta \Psi - RT \ln \left(\frac{[C_{in}]}{[C_{out}]} \right)$

(D) $\Delta G = RT \ln \left(\frac{[C_{in}]}{[C_{out}]} \right)$

Q2. A multi-system channelopathy is discovered where a mutation in the voltage-sensing S4 segment of a specific ion channel alters its gating properties. Patch-clamp experiments demonstrate that the activation threshold shifted from -70 mV to -40 mV. Given the biological parameters below plotted on the membrane potential graph, evaluate which trace accurately defines the



modified chord conductance (G) relative to the wild-type channel performance:



- (A) Rightward shift with an altered activation slope matching Mutant Curve β
- (B) Hyperpolarizing leftward shift with enhanced voltage sensitivity
- (C) Unchanged voltage dependence but a selective drop in absolute maximal peak I_{\max}
- (D) Linear ohmic conductance profile independent of voltage gating configurations

Q3. An experimental cell culture is treated with an irreversible inhibitor of the $\text{Na}^+ - \text{K}^+$ ATPase pump. Immediately after execution, the cell is placed into a hypertonic bath solution. Over a prolonged incubation phase, which of the following combined trends represents the sequence of changes in intracellular volume and intracellular pH (pH_i) due to subsequent loss of secondary active transport drivers?

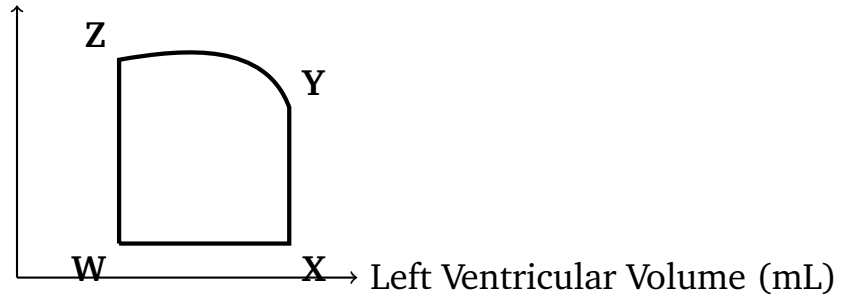
- (A) Initial shrinkage followed by secondary swelling; Intracellular acidification
- (B) Continuous cell swelling; Intracellular alkalinization
- (C) Continuous cell shrinkage; Intracellular acidification
- (D) Initial swelling followed by secondary shrinkage; Intracellular alkalinization

Q4. A 62-year-old male with severe calcific aortic stenosis undergoes comprehensive cardiac catheterization. The interventional cardiologist acquires



synchronous pressure-volume loops to gauge myocardial performance metrics. Analyze the structural configuration loop provided below. Identify which specific coordinate intersection corresponds precisely to the point of opening of the aortic valve:

Left Ventricular Pressure (mmHg)



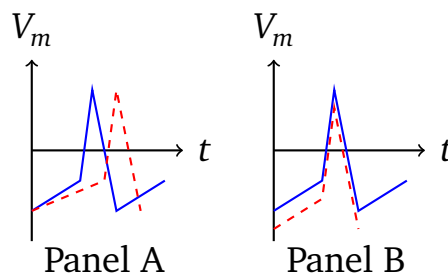
- (A) Coordinate point W
- (B) Coordinate point X
- (C) Coordinate point Y
- (D) Coordinate point Z
- Q5.** During a physiological simulation of isolated hemorrhagic shock, arterial baroreceptor firing frequencies drop precipitously. A computational mathematical model graphs the baroreceptor afferent output signal versus mean arterial pressure (MAP). If the system baseline sensitivity slope ($\Delta\text{Freq}/\Delta\text{MAP}$) is drastically attenuated by endotoxins, what is the net compensation impact on the setpoint and the systemic vascular resistance (SVR) curve?
- (A) Setpoint shifts rightwards; SVR spikes violently to maintain cardiac output
- (B) Setpoint shifts leftwards; SVR drops because of sympathetic withdrawal
- (C) Setpoint remains steady; SVR demonstrates fixed unresponsiveness to modifications
- (D) Setpoint shifts rightwards; SVR decreases due to paradoxically elevated parasympathetic drive
- Q6.** A 45-year-old patient presents with an atypical supraventricular tachycardia. Electrophysiological testing demonstrates a delayed phase 0 depolarization



velocity in the atrioventricular (AV) nodal cells. A molecular pathology report points to a selective modification of an ion channel pore. Which of the following ionic currents is primarily responsible for generating the upstroke phase of action potentials within these specific specialized cardiac pacemaker nodes?

- (A) Fast inward Na^+ current (I_{Na}) via voltage-gated Na^+ channels
- (B) L-type Ca^{2+} current ($I_{\text{Ca,L}}$) via voltage-gated Ca^{2+} channels
- (C) Transient outward K^+ current (I_{to}) via voltage-gated K^+ channels
- (D) Inward rectifier K^+ current (I_{K1})

Q7. An experimental drug under trial blocks the funny current (I_f) in the sinoatrial node without modifying any other cardiac conductances. A researcher records the baseline and drug-induced pacemaker potentials. Identify the diagram profile below that reflects the exact electrical alteration produced by this pharmacological block agent:



- (A) Panel A, showing a decreased slope of diastolic depolarization (Phase 4)
- (B) Panel B, showing an isolated reduction in the peak amplitude of Phase 0
- (C) Panel A, showing a marked hyperpolarization of the maximum diastolic potential
- (D) Panel B, showing a shortening of the overall action potential duration profile

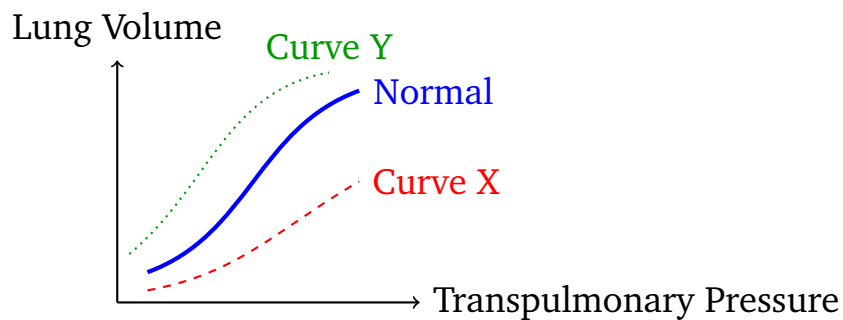
Q8. A senior high-altitude mountaineer attempts a rapid, unacclimatized ascent to an altitude where the barometric pressure is exactly half of sea-level value (380 mmHg). Assuming his alveolar PCO_2 drops to 20 mmHg due to hyperventilation and his respiratory quotient (RQ) is 0.8, calculate his exact



Alveolar PO_2 (PAO_2) value using the standard alveolar gas equation (Water vapor pressure at body temperature = 47 mmHg):

- (A) 45.0 mmHg
- (B) 69.9 mmHg
- (C) 39.2 mmHg
- (D) 54.7 mmHg

Q9. A patient with advanced idiopathic pulmonary fibrosis undergoes pulmonary function testing. The laboratory presents a static compliance curve mapping lung volume against transpulmonary pressure (P_{tp}). Which of the options correctly describes the position shift of this patient's compliance trace on the comparative axes relative to a normal healthy control?



- (A) Shifted downward and to the right, corresponding to Curve X (decreased compliance)
 - (B) Shifted upward and to the left, corresponding to Curve Y (increased compliance)
 - (C) Identical matching to the normal curve, but with early expiratory termination
 - (D) Shifted horizontally to the left without changes in maximum lung volume
- Q10.** During an experiment evaluating the Bohr effect versus Haldane effect kinetics in capillary systems, blood gas profiles are processed across varying hemoglobin parameters. Which of the following molecular descriptions correctly clarifies the underlying mechanism of the Haldane effect within functional systemic capillary beds?

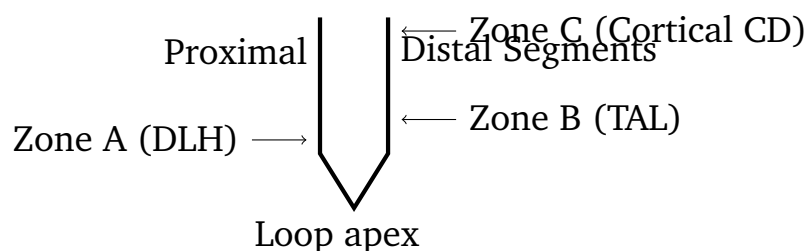


- (A) Deoxygenation of hemoglobin increases its affinity for CO_2 and protons, facilitating CO_2 loading.
- (B) High concentrations of CO_2 displace oxygen from oxyhemoglobin complexes via carbamino-group transformation.
- (C) Binding of 2,3-BPG directly induces structural transitions from the R-state to the T-state layout.
- (D) Carbonic anhydrase activation shifts localized cellular equilibrium points toward bicarbonate ion excretion channels.

Q11. A laboratory rat is given an intravenous infusion of a novel chemical compound that selectively constricts the efferent arteriole while keeping afferent arteriolar resistance completely constant. Assuming no activation of intrinsic autoregulation loops or tubuloglomerular feedback mechanisms, what will be the resulting changes in Glomerular Filtration Rate (GFR), Renal Plasma Flow (RPF), and the overall Filtration Fraction (FF)?

- (A) GFR increases, RPF decreases, FF increases
- (B) GFR decreases, RPF increases, FF decreases
- (C) GFR increases, RPF increases, FF remains unchanged
- (D) GFR decreases, RPF decreases, FF increases

Q12. A clinical trial participant is administered a loop diuretic agent that inhibits the outer medullary $\text{Na}^+ - \text{K}^+ - 2\text{Cl}^-$ co-transporter. Researchers track the changes across the longitudinal tubular fluid-to-plasma osmolarity profile ratio along different segments. Which point on the anatomical schematic path representation below loses its normal hyper-diluting profile status following this drug intervention?



- (A) Zone A (Descending Limb of Henle)



- (B) Zone B (Thick Ascending Limb of Henle)
- (C) Zone C (Cortical Collecting Ducts)
- (D) The entire proximal segment exclusively

Q13. A patient presents with a plasma sodium concentration of 118 mEq/L and an abnormally high urine osmolarity of 650 mOsm/kg H₂O. A clinical diagnosis of SIADH is established. At the cellular level within principal cells of the collecting duct, what molecular rearrangement occurs regarding aquaporin-2 channel trafficking vectors?

- (A) Persistent V2-receptor stimulation elevates intracellular cAMP, driving vesicle fusion and exocytosis of aquaporin-2 into the apical membrane.
- (B) V1a-receptor activation triggers PKC phosphorylation cascades, which reclaim aquaporin-2 into storage endosomes.
- (C) Basolateral aquaporin-3 and aquaporin-4 channels are selectively down-regulated via ubiquitination.
- (D) Apical clathrin-coated pits undergo rapid structural disassembly, preventing trans-cellular water reabsorption pathways.

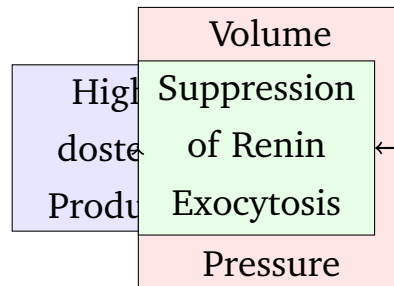
Q14. A molecular endocrinologist isolates a mutant variant of the Thyroid Hormone Receptor located in the nucleus. This mutant receptor binds its ligand T₃ with normal affinity, but fails to dissociate from its Co-repressor protein complex even when fully saturated with T₃. What baseline transcription status profile will be seen at the target Thyroid Hormone Response Element (TRE) locus?

- (A) Continuous and unremitting baseline gene transcription suppression
- (B) Uncontrolled, constitutive activation of target gene transcription
- (C) Hyper-responsiveness to changing levels of active free thyroxine concentrations
- (D) Spontaneous splicing destruction of the transcribed pre-mRNA molecules

Q15. A 38-year-old female presents with severe muscle weakness, hypertension, and recurrent hypokalemia. Lab values reveal a plasma aldosterone-to-renin activity ratio (ARR) greater than 40. A CT scan confirms a unilateral



aldosterone-producing adrenal adenoma (Conn syndrome). Which of the following physiological response maps correctly traces the expected direct feedback changes occurring inside her juxtaglomerular apparatus cells?



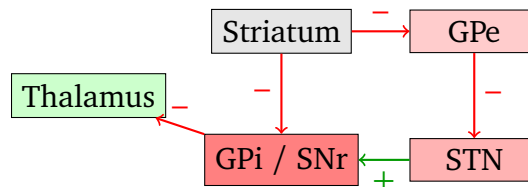
- (A) High Aldosterone → ECF expansion → Increased renal perfusion → Downregulation of renin release
- (B) High Aldosterone → Hyperkalemia → Direct chemical suppression of the juxtaglomerular cells
- (C) High Aldosterone → Decreased distal Na^+ delivery → Macula densa-mediated renin surge
- (D) High Aldosterone → Systemic vasodilation → Baroreceptor-mediated sympathetic renin discharge

Q16. A neurophysiology experiment records postsynaptic potentials from a pyramidal neuron in the hippocampus. Stimulating an input pathway at a high frequency (100 Hz) triggers Long-Term Potentiation (LTP). Which molecular event sequence is mandatory to clear the magnesium block from NMDA receptor pores during this induction phase?

- (A) Sustained activation of AMPA receptors leads to sufficient localized dendritic depolarization to electrostatically repel Mg^{2+} ions out of the NMDA channel pore.
- (B) Direct binding of retrograde nitric oxide gas to the channel's extracellular regulatory domain.
- (C) Metabotropic glutamate receptor activation shifts intracellular calcium storage targets toward receptor phosphorylation.
- (D) Calcineurin activation uncouples structural anchor frameworks via endocytic internalization processes.



Q17. A 55-year-old patient exhibits a resting tremor, bradykinesia, and postural instability. A functional neuroimaging study shows a selective loss of dopaminergic projections from the substantia nigra pars compacta to the striatum. In the basic functional wiring diagram of the basal ganglia circuitry below, which pathway element shifts into an abnormally hyperactive state as a direct mechanical consequence of this disease?



- (A) Tonic inhibitory output from the Subthalamic Nucleus (STN) to the GPi/SNr complex
- (B) Inhibitory output from the GPi/SNr complex to the Thalamus, suppressing cortical drive
- (C) Excitatory GABAergic feedback transmission originating from the Thalamic projection neurons
- (D) Inhibitory output from the Striatum directly to the GPe segment



Detailed Solutions

Q1.

Solution

Concept:

Peptide transport that establishes an intracellular concentration far exceeding electrochemical equilibrium represents primary or secondary active transport. The total maximum work capacity per mole required to move a solute against both a chemical concentration gradient and an electrical potential gradient is defined by its electrochemical potential change (ΔG).

Solution:

Let's analyze the components of the thermodynamic work equation:

- (a) The total free energy change (ΔG) represents the sum of the chemical work and the electrical work performed during transport.
- (b) The chemical work required to move a substance from the outside to the inside of a cell is given by:

$$\Delta G_{\text{chem}} = RT \ln \left(\frac{[C_{\text{in}}]}{[C_{\text{out}}]} \right)$$

- (c) The electrical work required to move a charged particle across an electrical potential difference ($\Delta\Psi = \Psi_{\text{in}} - \Psi_{\text{out}}$) is determined by its valence (z) and Faraday's constant (F):

$$\Delta G_{\text{elect}} = zF\Delta\Psi$$

Combining both thermodynamic quantities yields the complete electrochemical potential equation: $\Delta G = RT \ln \left(\frac{[C_{\text{in}}]}{[C_{\text{out}}]} \right) + zF\Delta\Psi$. When mitochondrial complex IV is inhibited, ATP synthesis halts, causing this active transport system's energy capacity to drop to zero and the gradient to collapse.

Final Answer: $\Delta G = RT \ln \left(\frac{[C_{\text{in}}]}{[C_{\text{out}}]} \right) + zF\Delta\Psi$

Answer: (A)

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Q2.

Solution**Concept:**

The voltage-sensing S4 segments of voltage-gated ion channels contain positively charged amino acid residues that move in response to changes in membrane potential, controlling channel gating. A shift in the activation threshold to a more positive voltage (from -70 mV to -40 mV) means the channel requires greater depolarization to open.

Solution:

Let's analyze the graphical shift in chord conductance (G) resulting from this gating channelopathy:

- Shifting the voltage activation threshold from -70 mV to -40 mV represents a depolarizing, **rightward shift** along the membrane potential axis (V_m).
- This specific modification maps precisely to Mutant Curve β , where the voltage threshold is displaced to -40 mV and exhibits an altered activation slope profile.
- Leftward shifts correspond to hyperpolarizing mutations, structural drops in I_{\max} alter maximum current without modifying threshold coordinates, and linear profiles describe voltage-independent leak pathways.

Final Answer: Rightward shift with an altered activation slope matching Mutant Curve β

Answer: (A)

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Q3.

Solution**Concept:**

Inhibiting the primary active $\text{Na}^+ - \text{K}^+$ ATPase pump disrupts the fundamental ion gradients that sustain secondary active transport mechanisms across the cell membrane, including the sodium-hydrogen exchanger (NHE) and osmotic balance vectors.

Solution:

Let's break down the timeline of changes following pump inhibition and placement in a hypertonic environment:

- (a) **Volume Dynamics:** Placed into a hypertonic bath, water initially moves out of the cell down its osmotic gradient, causing an **initial shrinkage**. However, because the $\text{Na}^+ - \text{K}^+$ pump is permanently blocked, intracellular Na^+ gradually accumulates, and the normal resting membrane potential is lost. This intracellular solute accumulation creates a secondary hyperosmotic force that drives water back into the cell, causing a **secondary swelling**.
- (b) **pH Dynamics:** The gradual loss of the extracellular-to-intracellular Na^+ gradient eliminates the driving force for the secondary active Na^+ / H^+ exchanger (NHE). Without functional NHE operation, metabolically produced protons cannot be extruded, leading to **intracellular acidification**.

Final Answer: Initial shrinkage followed by secondary swelling; Intracellular acidification

Answer: (A)

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Q4.

Solution**Concept:**

A left ventricular pressure-volume (PV) loop charts the mechanical changes that occur within the ventricular chamber during a single complete cardiac cycle. The four corners of the loop reflect the opening and closing points of the cardiac valves.

Solution:

Let's track the mechanical sequence around the four corners of the provided PV loop:

- (a) **Point W:** Marks the opening of the mitral valve, initiating the diastolic ventricular filling phase along the bottom segment (from W to X).
- (b) **Point X:** Marks the closure of the mitral valve at the end-diastolic volume point. The vertical segment from X to Y represents isovolumetric contraction.
- (c) **Point Y:** At this intersection, left ventricular pressure exceeds aortic diastolic pressure, forcing the **aortic valve to open** and initiating the systolic ejection phase (from Y to Z).
- (d) **Point Z:** Marks the closure of the aortic valve, which initiates the vertical drop of isovolumetric relaxation (from Z to W).

Final Answer:

Answer: (C)

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Q5.

Solution**Concept:**

The arterial baroreceptor reflex serves as a primary homeostatic mechanism regulating systemic blood pressure. Endotoxins can BW impair this reflex by altering baroreceptor sensitivity ($\Delta\text{Freq}/\Delta\text{MAP}$), shifting the operational setpoint and changing downstream vascular compensation profiles.

Solution:

Let's analyze the physiological consequences of endotoxin-induced baroreceptor desensitization:

- (a) Attenuating the sensitivity slope reduces the afferent firing frequency for any given mean arterial pressure. This causes the neural control center to perceive the blood pressure as abnormally low, shifting the operational **setpoint rightwards**.
- (b) Because the central nervous system misinterprets the signal as a hypotensive state, it triggers a massive increase in sympathetic outflow to blood vessels.
- (c) This sympathetic discharge causes widespread vasoconstriction, causing the **systemic vascular resistance (SVR) to spike violently** in an attempt to normalize systemic perfusion and maintain blood pressure.

Final Answer: Setpoint shifts rightwards; SVR spikes violently to maintain cardiac output

Answer: (A)

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Q6.

Solution**Concept:**

Cardiac action potentials vary by tissue type. While working myocardial cells and Purkinje fibers rely on fast voltage-gated sodium channels (I_{Na}) for their rapid Phase 0 upstroke, specialized pacemaker tissues like the atrioventricular (AV) and sinoatrial (SA) nodes utilize distinct channel profiles.

Solution:

Let's identify the ionic current that drives depolarization in specialized nodal tissue:

- (a) AV nodal cells lack functional fast voltage-gated sodium channels due to their relatively depolarized resting membrane potentials.
- (b) Instead, the Phase 0 upstroke velocity in these pacemaking nodes is driven exclusively by the activation of **L-type Ca^{2+} channels ($I_{Ca,L}$)**.
- (c) Because L-type calcium channels open more slowly than fast sodium channels, the resulting Phase 0 upstroke has a lower maximum velocity (dV/dt_{max}), which accounts for the slower conduction velocity characteristic of AV nodal tissue.

Final Answer: L-type Ca^{2+} current ($I_{Ca,L}$) via voltage-gated Ca^{2+} channels

Answer: (B)

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Q7.

Solution**Concept:**

The funny current (I_f), carried by hyperpolarization-activated cyclic nucleotide-gated (HCN) channels, is an inward current activated at hyperpolarized potentials. It provides a major depolarizing drive during Phase 4 spontaneous diastolic depolarization in sinoatrial node pacemaker cells.

Solution:

Let's examine how blocking this current alters the pacemaker potential profile:

- Pharmacological blockade of I_f reduces the influx of positive ions during diastole, which flattens or **decreases the slope of Phase 4 diastolic depolarization**.
- This flattened slope means it takes longer for the pacemaker potential to reach the threshold required to activate L-type calcium channels for Phase 0.
- This change is illustrated in **Panel A**, where the dashed mutant line shows a prolonged Phase 4 slope, which slows the pacemaking frequency without altering the peak amplitude or maximum diastolic potential.

Final Answer: Panel A, showing a decreased slope of diastolic depolarization (Phase 4)

Answer: (A)

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Q8.

Solution**Concept:**

The partial pressure of oxygen within the alveoli (PAO_2) is calculated using the standard Alveolar Gas Equation. This formula accounts for the reduction in oxygen pressure due to water vapor humidification in the upper airways and the metabolic consumption of oxygen relative to carbon dioxide production in the lungs.

Solution:

Let's state the Alveolar Gas Equation formula and calculate the values:

$$PAO_2 = [(P_{\text{baro}} - P_{\text{H}_2\text{O}}) \times FiO_2] - \frac{PACO_2}{RQ}$$

Given the parameters: $P_{\text{baro}} = 380$ mmHg, $P_{\text{H}_2\text{O}} = 47$ mmHg, $FiO_2 = 0.21$ (atmospheric oxygen fraction remains constant at 21%), $PACO_2 = 20$ mmHg, and $RQ = 0.8$.

(a) Calculate the humidified inspired oxygen tension (P_1O_2):

$$P_1O_2 = (380 \text{ mmHg} - 47 \text{ mmHg}) \times 0.21 = 333 \times 0.21 = 69.93 \text{ mmHg}$$

(b) Calculate the metabolic gas exchange factor:

$$\frac{PACO_2}{RQ} = \frac{20 \text{ mmHg}}{0.8} = 25.0 \text{ mmHg}$$

(c) Subtract the metabolic factor from the inspired oxygen tension to find PAO_2 :

$$PAO_2 = 69.93 \text{ mmHg} - 25.0 \text{ mmHg} = 44.93 \text{ mmHg} \approx 45.0 \text{ mmHg}$$

Final Answer:

Answer: (A)

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Q9.

Solution**Concept:**

Static compliance curves map changes in lung volume against transpulmonary pressure (P_{tp}). The slope of this curve ($\Delta V/\Delta P$) reflects the elasticity of the lung parenchyma. Fibrotic processes deposit rigid collagen matrix, increasing restrictive inward elastic recoil and decreasing compliance.

Solution:

Let's determine the position shift of the compliance curve under fibrotic pathology:

- (a) Because the fibrotic lung is stiff and non-compliant, it requires much higher transpulmonary pressures to achieve a given change in lung volume, flattening the slope of the compliance curve.
- (b) This mechanical restriction reduces total lung volumes (including Total Lung Capacity and Functional Residual Capacity), shifting the compliance trace **downward and to the right**.
- (c) This corresponds to **Curve X**, which illustrates a classical restrictive lung profile with decreased compliance, contrasting with the upward and leftward shift seen in conditions with increased compliance like emphysema (Curve Y).

Final Answer:

Shifted downward and to the right, corresponding to Curve X (decreased compliance)

Answer: (A)[Go Back to Question 9](#)

Q10.

Solution**Concept:**

The Haldane effect describes how oxygen levels alter hemoglobin's affinity for carbon dioxide (CO_2) and protons (H^+). This mechanism complements the Bohr effect to optimize respiratory gas transport and exchange within both systemic and pulmonary capillary networks.

Solution:

Let's trace the molecular mechanism of the Haldane effect in systemic capillary beds:

- (a) As hemoglobin unloads oxygen to peripheral tissues, it transitions from its oxygenated form to its deoxygenated conformation (T-state).
- (b) This **deoxygenation of hemoglobin increases its affinity for both protons and carbon dioxide**. Deoxyhemoglobin acts as a more effective buffer for H^+ ions and binds CO_2 more readily to form carbaminohemoglobin complexes.
- (c) This conformation change facilitates the loading of metabolic CO_2 from active tissues into the blood, allowing it to be transported back to the lungs.

Final Answer:

Deoxygenation of hemoglobin increases its affinity for CO_2 and protons, facilitating CO_2 loading.

Answer: (A)[Go Back to Question 10](#)

Q11.

Solution**Concept:**

Glomerular filtration parameters are regulated by the relative resistances of the afferent and efferent arterioles. The Filtration Fraction ($FF = \frac{GFR}{RPF}$) describes the proportion of renal plasma flow that is filtered across the glomerular filtration barrier.

Solution:

Let's evaluate the hemodynamic effects of selectively constricting the efferent arteriole while keeping afferent resistance constant:

- (a) Constricting the efferent arteriole restricts blood outflow from the glomerulus, which increases the upstream hydrostatic pressure within the glomerular capillaries (P_G), driving an **increase in the Glomerular Filtration Rate (GFR)**.
- (b) This constriction increases total renal vascular resistance, which reduces the total volume of blood flowing through the kidney, resulting in a **decrease in Renal Plasma Flow (RPF)**.
- (c) Because GFR increases while RPF decreases, the ratio of filtration to plasma flow—the **Filtration Fraction (FF)**—must increase.

Final Answer: GFR increases, RPF decreases, FF increases

Answer: (A)

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Q12.

Solution**Concept:**

The thick ascending limb (TAL) of the loop of Henle is impermeable to water but actively reabsorbs solutes (Na^+ , K^+ , and Cl^-) via apical $\text{Na}^+ - \text{K}^+ - 2\text{Cl}^-$ (NKCC2) co-transporters. This separation of solute and water reabsorption dilutes the tubular fluid, giving the TAL its designation as the “diluting segment.”

Solution:

Let's analyze the effects of loop diuretic intervention on the different nephron segments:

- (a) Under normal physiological conditions, fluid entering the descending limb becomes concentrated, while fluid moving up the TAL (**Zone B**) is progressively diluted as solutes are reabsorbed, dropping its osmolarity below that of plasma.
- (b) Administering a loop diuretic blocks these apical NKCC2 co-transporters within the TAL (**Zone B**), halting active solute reabsorption.
- (c) Inhibiting this transporter prevents the normal dilution of tubular fluid within this segment, causing the TAL to lose its hyper-diluting status and altering the corticopapillary osmotic gradient.

Final Answer: Zone B (Thick Ascending Limb of Henle)

Answer: (B)

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Q13.

Solution**Concept:**

The Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH) is characterized by autonomous, unregulated release of ADH (vasopressin). This leads to excessive water reabsorption in the collecting ducts, causing concentrated urine and dilutional hyponatremia.

Solution:

Let's map the intracellular signaling pathway of ADH in the principal cells of the collecting duct:

- (a) Circulating ADH binds to basolateral V_2 receptors on the principal cells, which are G_{α_s} -coupled receptors.
- (b) This binding activates adenylyl cyclase, raising intracellular cAMP levels and activating Protein Kinase A (PKA).
- (c) PKA phosphorylates target proteins that regulate vesicle trafficking, driving the **exocytosis and insertion of aquaporin-2 (AQP2) water channels into the apical membrane**. This insertion increases the water permeability of the membrane, allowing water to flow down the osmotic gradient into the medullary interstitium.

Final Answer:

Persistent V_2 -receptor stimulation elevates intracellular cAMP, driving vesicle fusion and exocytosis of aquaporin-2 into the apical membrane.

Answer: (A)

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Q14.

Solution**Concept:**

The nuclear thyroid hormone receptor (TR) regulates transcription by binding to Thyroid Hormone Response Elements (TRE) in target DNA. In its un-liganded state, TR associates with co-repressor protein complexes to actively repress baseline gene transcription.

Solution:

Let's analyze the molecular transcription status of the mutant receptor variant:

- (a) Under normal conditions, thyroid hormone (T_3) binding induces a conformational change in the TR that causes the co-repressor complex to dissociate. This allows co-activators to bind and initiate gene transcription.
- (b) The mutant receptor variant binds T_3 normally but fails to dissociate from its co-repressor complex, blocking the recruitment of co-activators.
- (c) Because the co-repressor complex remains bound to the TRE locus regardless of hormone levels, target gene expression experiences **continuous and unremitting baseline gene transcription suppression**.

Final Answer: Continuous and unremitting baseline gene transcription suppression

Answer: (A)

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Q15.

Solution**Concept:**

Primary hyperaldosteronism (Conn syndrome) is characterized by autonomous production of aldosterone from an adrenal adenoma. This excessive aldosterone acts on principal cells in the collecting duct to increase sodium reabsorption, causing volume expansion and hypertension.

Solution:

Let's trace the homeostatic feedback loops inside the juxtaglomerular apparatus (JGA) cells:

- (a) Autonomous high aldosterone production drives excessive renal sodium retention and extracellular fluid (ECF) volume expansion.
- (b) This volume expansion elevates systemic blood pressure and increases renal perfusion pressure, which is detected by baroreceptors in the afferent arterioles.
- (c) The increased perfusion pressure and the resulting delivery of solute to the macula densa generate inhibitory signals that **suppress renin exocytosis and release** from JGA cells, leading to the low plasma renin activity characteristic of Conn syndrome.

Final Answer:

High Aldosterone → ECF expansion → Increased renal perfusion → Down-regulation of renin release.

Answer: (A)[Go Back to Question 15](#)

Q16.

Solution**Concept:**

The induction of classic N-methyl-D-aspartate (NMDA) receptor-dependent Long-Term Potentiation (LTP) in hippocampal neurons requires both glutamate binding and postsynaptic depolarization to unblock the channel pore.

Solution:

Let's trace the sequence of molecular events that removes the magnesium block:

- (a) At resting membrane potentials, the pore of the NMDA receptor is physically blocked by an extracellular magnesium ion (Mg^{2+}), preventing ion flow even when glutamate is bound.
- (b) High-frequency (100 Hz) tetanic stimulation causes a rapid, sustained release of glutamate that strongly activates adjacent postsynaptic α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors.
- (c) This **sustained activation of AMPA receptors drives a strong localized dendritic depolarization**. This positive intracellular charge electrostatically repels the divalent Mg^{2+} ion out of the NMDA channel pore, allowing calcium (Ca^{2+}) influx to initiate downstream LTP signaling cascades.

Final Answer:

Sustained activation of AMPA receptors leads to sufficient localized dendritic depolarization to electrostatically repel Mg^{2+} ions out of the NMDA channel pore.

Answer: (A)[Go Back to Question 16](#)

Q17.

Solution**Concept:**

The basal ganglia circuit modulates voluntary motor movement via two balanced pathways: the direct pathway (which facilitates movement) and the indirect pathway (which inhibits movement). Dopaminergic projections from the substantia nigra pars compacta (SNc) modulate these pathways by exciting the direct pathway (via D₁ receptors) and inhibiting the indirect pathway (via D₂ receptors).

Solution:

Let's analyze the structural consequences of losing dopaminergic projections in Parkinson's disease:

- (a) The loss of nigrostriatal dopamine reduces the normal inhibition of the indirect pathway, leading to hyperactivation of striatal projections to the globus pallidus externus (GPe).
- (b) This increased inhibitory output silences the GPe, removing its tonic inhibition from the subthalamic nucleus (STN).
- (c) Uninhibited, the **Subthalamic Nucleus (STN) shifts into an abnormally hyperactive state**, sending excessive excitatory glutamatergic signals (+) to the globus pallidus internus and substantia nigra pars reticulata (GPi/SNr) complex. This hyperactive GPi/SNr output strongly suppresses thalamocortical drive, causing bradykinesia and rigidity.

Final Answer:

Tonic inhibitory output from the Subthalamic Nucleus (STN) to the GPi/SNr complex.

Answer: (A)

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Answer Key

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

