

NEET PG Physiology Sample Paper-8

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. An experimental biophysicist measures the membrane potential (V_m) of a mutated mammalian cell line. The intracellular concentration of a synthetic monovalent anion (X^-) is 10 times higher than its extracellular concentration. If the cell membrane is made exclusively permeable to X^- and the temperature is held at 37°C ($2.303\frac{RT}{F} \approx 61.5\text{ mV}$), calculate the equilibrium membrane potential (E_X) dictated by this gradient.

- (A) -61.5 mV
- (B) $+61.5\text{ mV}$
- (C) -30.7 mV
- (D) 0 mV

Q2. During a patch-clamp analysis of a novel epithelial cell lining, a researcher discovers a channel that exhibits a reflection coefficient (σ) of exactly 0.00 for solute alpha, and 1.00 for solute beta. Which of the following statements accurately characterizes the osmotic water flux across this membrane?

- (A) Solute alpha will exert maximum effective osmotic pressure.
- (B) Solute beta will induce no hydrostatic pressure driving forces.
- (C) Solute alpha will pass through without generating an effective osmotic gradient.

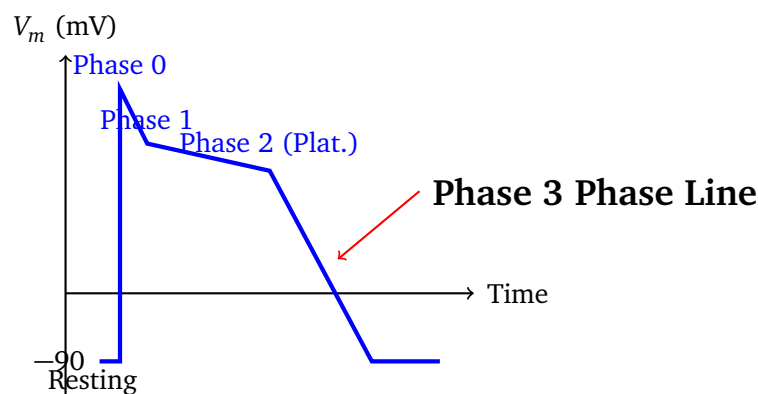


(D) Both solutes will contribute equally to the total effective osmotic pressure.

Q3. A patient presents with a severe genetic mutation disrupting the normal functioning of the clathrin-coated pit accessory protein, AP2. A biopsy of the hepatic tissue is taken, and cellular uptake rates are measured. Which of the following cellular transport mechanisms will be selectively eliminated or severely down-regulated in this patient's hepatocytes?

- (A) Fluid-phase pinocytosis
- (B) Receptor-mediated endocytosis of LDL
- (C) Caveolae-dependent pinocytosis
- (D) Macropinocytosis of extracellular fluid

Q4. A clinical researcher analyzes an advanced microfluidic model of a ventricular myocardial action potential recording under the influence of an experimental Class III antiarrhythmic agent. Identify the specific phase configuration labeled in the active voltage-clamp trace below where the net outward repolarizing potassium current (I_{Kr} and I_{Ks}) reaches its peak absolute activation kinetics before shutting down at hyperpolarized baselines:



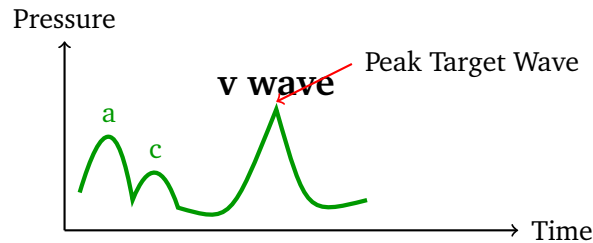
- (A) Phase 0 Upstroke
- (B) Phase 1 Rapid Repolarization
- (C) Phase 2 Plateau Phase
- (D) Phase 3 Phase Line



- Q5.** An invasive cardiologist measures hemodynamic shifts in an animal model experiencing severe acute aortic regurgitation. Which of the following sets of physiological changes best reflects the rapid compensatory left ventricular responses to maintain stroke volume before myocardial failure sets in?
- (A) Decreased End-Diastolic Volume, Increased End-Systolic Volume
 - (B) Increased End-Diastolic Volume, Decreased End-Systolic Volume
 - (C) Increased End-Diastolic Volume, Increased Stroke Volume
 - (D) Decreased End-Diastolic Volume, Decreased Stroke Volume
- Q6.** A 62-year-old male with chronic hypertension undergoes a cardiac catheterization procedure. The data indicates an isolated, significant decrease in the compliance of the large central elastic arteries. Which of the following alterations in his central aortic pressure pulse waveform profile will be observed?
- (A) Decreased Systolic Pressure, Increased Diastolic Pressure
 - (B) Increased Systolic Pressure, Decreased Diastolic Pressure
 - (C) Unchanged Pulse Pressure with elevated Mean Arterial Pressure
 - (D) Decreased Systolic Pressure, Decreased Pulse Pressure
- Q7.** During a laboratory demonstration of cardiac electrophysiology, the sinoatrial (SA) node of a canine heart is perfused with a highly selective blocker of the hyperpolarization-activated cyclic nucleotide-gated (HCN) channels (I_f). What is the primary direct consequence of this blocker on the pacemaking action potential?
- (A) Hyperpolarization of the peak overshoot potential
 - (B) Marked reduction in the rate of Phase 4 spontaneous diastolic depolarization
 - (C) Complete blockade of the Phase 0 upstroke depolarization velocity
 - (D) Acceleration of the Phase 3 repolarization rate via I_{K1} activation



- Q8.** A critical care specialist examines a central venous pressure (CVP) tracing obtained from a patient in the intensive care unit. Identify the mechanical cardiac event corresponding directly to the apex of the physiological wave labeled as the “v wave” in the diagram provided below:

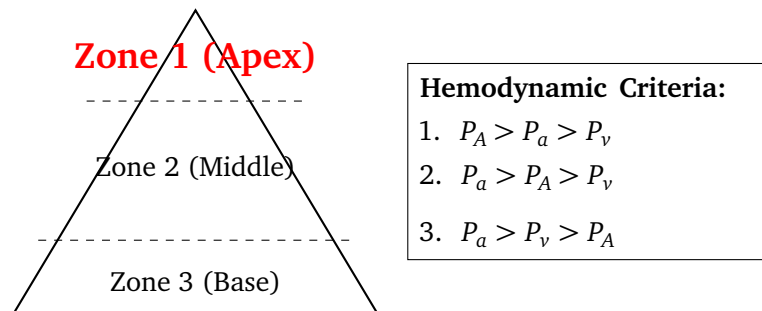


- (A) Isovolumetric ventricular contraction phase
 (B) Atrial systole leading to backward blood propulsion
 (C) Venous return filling the atrium against a closed tricuspid valve
 (D) Rapid ventricular ejection causing atrial floor dropping
- Q9.** An elite high-altitude climber ascends rapidly to a peak without supplemental oxygen. Arterial blood gas parameters reveal an acute drop in P_aO_2 to 48 mmHg, accompanied by an arterial pH of 7.55. Which of the following changes in the cerebrospinal fluid (CSF) microenvironment occurs next to limit further compensatory hyperventilation?
- (A) Local accumulation of H^+ ions in the CSF
 (B) Active pumping of HCO_3^- out of the CSF into the bloodstream
 (C) Respiratory acidosis within the interstitial fluid of the medulla
 (D) Passive accumulation of metabolic CO_2 in the lateral ventricles
- Q10.** A patient suffering from progressive pulmonary fibrosis undergoes pulmonary function testing. The laboratory reports a dramatic reduction in lung compliance, along with a mismatching of ventilation and perfusion. Which of the following changes in the functional residual capacity (FRC) and the inward elastic recoil of the lung is true?
- (A) Increased FRC, Increased Lung Recoil Force



- (B) Decreased FRC, Increased Lung Recoil Force
- (C) Increased FRC, Decreased Lung Recoil Force
- (D) Decreased FRC, Decreased Lung Recoil Force

Q11. The physiological diagram below depicts a standard West Zone model of pulmonary blood flow distribution in an upright human lung. Identify the exact hemodynamic relationship and limitation that defines the boundaries of **Zone 1** near the apex of the lung structure:



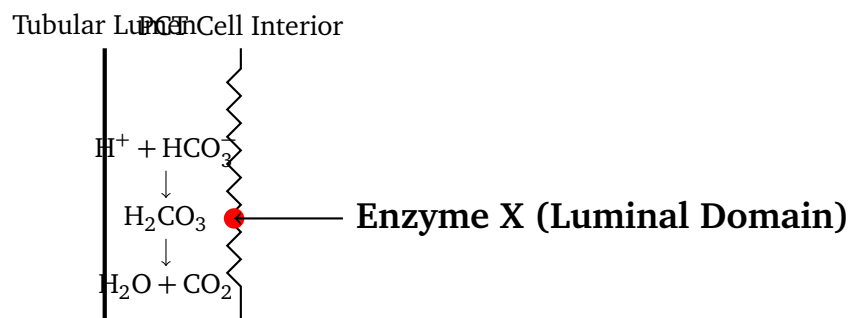
- (A) Arterial pressure exceeds alveolar pressure ($P_a > P_A$)
 - (B) Alveolar pressure exceeds arterial pressure ($P_A > P_a > P_v$)
 - (C) Venous pressure exceeds alveolar pressure ($P_v > P_A$)
 - (D) Venous pressure equals arterial pressure ($P_v = P_a$)
- Q12.** An experimental drug blocks the apical $Na^+ - K^+ - 2Cl^-$ (NKCC2) cotransporter in the thick ascending limb of Henle's loop. Aside from direct natriuresis, what is the impact of this drug on the corticopapillary osmotic gradient and urinary concentrating ability?
- (A) Gradient is preserved; maximum concentrated urine is produced.
 - (B) Gradient is obliterated; maximum urine concentrating ability is lost.
 - (C) Gradient is enhanced due to increased distal water reabsorption.
 - (D) Gradient shifts to the outer cortex; urea recycling stops completely.
- Q13.** A patient with severe renal artery stenosis develops systemic hypertension. Micropuncture evaluation reveals an altered single-nephron glomerular filtration rate (SNGFR). If the efferent arteriolar resistance (R_e) is selectively



increased via local Angiotensin II production while renal blood flow drops, what is the net effect on glomerular capillary hydrostatic pressure (P_G) and Filtration Fraction (FF)?

- (A) Decreased P_G , Decreased FF
- (B) Increased P_G , Increased FF
- (C) Increased P_G , Decreased FF
- (D) Decreased P_G , Increased FF

Q14. A researcher maps the titration profile of acid-base buffer mechanisms across the length of a nephron. Observe the schematic of the proximal convoluted tubule (PCT) cell below. Identify the specialized brush border enzyme labeled as **Enzyme X** that catalyzes the rapid interconversion of carbonic acid into water and carbon dioxide within the luminal space:



- (A) Cytosolic Carbonic Anhydrase II
- (B) Membrane-bound Carbonic Anhydrase IV
- (C) Na^+/H^+ Exchanger isoform 3 (NHE3)
- (D) Apical H^+ -ATPase pump

Q15. A patient is diagnosed with a pituitary adenoma secreting excess adrenocorticotrophic hormone (ACTH). Laboratory testing confirms bilateral adrenal hyperplasia. At the molecular level within the zona fasciculata cells, binding of ACTH to its melanocortin 2 (MC2R) receptor initiates steroidogenesis primarily through which immediate downstream intracellular signaling cascade?

- (A) Activation of $G_{\alpha q}$ followed by Phospholipase C activation

- (B) Activation of $G_{\alpha s}$ followed by Adenylyl Cyclase and cAMP generation
- (C) Tyrosine kinase autophosphorylation and STAT5 recruitment
- (D) Opening of L-type voltage-gated calcium channels

Q16. A clinical trial investigates a novel antagonist of the Calcium-Sensing Receptor (CaSR) located on the parathyroid chief cells. When this antagonist binds, it blocks the receptor from detecting ionized calcium, mimicking a state of constant hypocalcemia. What is the expected physiological result regarding parathyroid hormone (PTH) secretion and renal calcium handling?

- (A) Suppression of PTH secretion, increased renal calcium clearance
- (B) Stimulation of PTH secretion, increased renal calcium reabsorption
- (C) Suppression of PTH secretion, decreased renal phosphate excretion
- (D) Stimulation of PTH secretion, increased renal phosphate reabsorption

Q17. A neurophysiology laboratory induces long-term potentiation (LTP) in a hippocampal slice preparation. High-frequency tetanic stimulation of the Schaffer collateral pathway leads to persistent synaptic strengthening at the CA1 pyramidal neurons. This classical form of LTP requires the displacement of which blocking ion from the channel pore of the NMDA receptor complex to permit calcium influx?

- (A) Zinc ion (Zn^{2+})
- (B) Magnesium ion (Mg^{2+})
- (C) Sodium ion (Na^+)
- (D) Chloride ion (Cl^-)



Detailed Solutions

Q1.

Solution

Concept:

The equilibrium potential (E_X) for any net ionic species moving down an isolation gradient across a selectively permeable lipid bilayer is dictated by the Nernst equation. This equation establishes the specific voltage baseline required to introduce an electrostatic repulsive force that perfectly balances the diffusion driving force of the chemical concentration gradient.

Solution:

Let's compute the equilibrium potential using the provided Nernst baseline constants:

$$E_X = \frac{2.303 RT}{zF} \log_{10} \left(\frac{[X^-]_{\text{outside}}}{[X^-]_{\text{inside}}} \right)$$

We are given that $2.303 \frac{RT}{F} \approx 61.5 \text{ mV}$. Let's evaluate the structural parameters given in the problem:

- The target ion is a monovalent anion (X^-), which sets the valence parameter $z = -1$.
- The intracellular concentration is exactly 10 times higher than the extracellular concentration, which means the ratio of external to internal concentration is:

$$\frac{[X^-]_{\text{outside}}}{[X^-]_{\text{inside}}} = \frac{1}{10} = 10^{-1}$$

- Substituting these parameters into the log formula gives:

$$E_X = \frac{61.5 \text{ mV}}{-1} \log_{10}(10^{-1}) = (-61.5 \text{ mV}) \times (-1) = +61.5 \text{ mV}$$

Because the negative anion is more concentrated inside the cell, it naturally tends to diffuse down its chemical gradient toward the outside. To halt this net flux completely, the cell interior must possess a positive electrical charge (+61.5 mV) to electrostatically attract and retain the anion inside.

Final Answer: +61.5 mV

Answer: (B)

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Q2.

Solution

Concept: The reflection coefficient (σ) describes the permeability of a membrane to a given solute, ranging on a scale from 0.00 to 1.00. It directly alters the calculated effective osmotic pressure via the Staverman correction factor ($\pi_{\text{effective}} = \sigma \pi_{\text{theoretical}}$).

Solution:

Let's analyze the properties of both solutes according to their reflection coefficients:

- (a) Solute alpha exhibits a reflection coefficient (σ) of exactly 0.00. This indicates that the channel membrane is completely, freely permeable to alpha. Because solute alpha crosses the lipid bilayer at the same rate as water, it cannot exert a sustained concentration gradient across the barrier and will generate zero effective osmotic pressure ($\pi_{\text{effective}} = 0$).
- (b) Solute beta exhibits a reflection coefficient (σ) of exactly 1.00. This describes a completely impermeable solute that is fully "reflected" by the cell lining. Solute beta is trapped on one side of the membrane, creating a maximal effective osmotic gradient that drives water flux.

Final Answer:

Solute alpha will pass through without generating an effective osmotic gradient.

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

Q3.

Solution

Concept: Receptor-mediated endocytosis relies on specialized coat structures to package cargo molecules into intracellular vesicles. Adaptor Protein 2 (AP2) is an essential multi-subunit complex that acts as a structural bridge, linking the cytoplasmic tails of specific transmembrane cargo receptors to the outer clathrin coat lattice.

Solution:

Let's review the transport pathways affected by a loss of functional AP2 complexes:

- (a) The low-density lipoprotein (LDL) receptor requires its cytoplasmic sorting signal to bind to AP2. This binding concentrates the receptor-LDL complex within clathrin-coated pits before internalization.
- (b) A severe genetic mutation disrupting the AP2 protein selectively prevents clathrin from assembling around these cargo receptors, eliminating receptor-mediated endocytosis of LDL.
- (c) Other non-clathrin pathways—such as fluid-phase pinocytosis, caveolae-dependent potocytosis, and large-scale actin-driven macropinocytosis—rely on separate molecular machinery and remain operational.

Final Answer: Receptor-mediated endocytosis of LDL

Answer: (B)

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Q4.

Solution

Concept: The ventricular action potential waveform is shaped by sequential changes in the balance of inward and outward ionic currents. Phase 3 represents the rapid repolarization phase, where the cardiac membrane potential returns from the positive plateau level back down to the resting baseline value of approximately -90 mV.

Solution:

Let's analyze the activation kinetics of the delayed rectifier potassium currents (I_{Kr} and I_{Ks}):

- During Phase 2 (the plateau phase), inward L-type calcium current ($I_{Ca,L}$) balances the outward potassium currents, keeping the membrane potential relatively steady.
- As Phase 2 ends, the L-type calcium channels slowly inactivate, while the delayed rectifier potassium channels (I_{Kr} and I_{Ks}) open further due to their voltage-dependent activation kinetics.
- The rapid drop in voltage during Phase 3 is driven by these outward potassium currents, which reach their peak absolute activation during this phase. This driving force returns the cell to its resting membrane potential.

Final Answer:

Answer: (D)

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Q5.

Solution

Concept: Acute aortic regurgitation allows blood to flow backward from the aorta into the left ventricle (LV) during diastole. This retrograde volume combines with the normal venous return from the left atrium, placing an acute volume overload on the left ventricular chamber.

Solution:

Let's trace the acute mechanical compensatory mechanisms of the myocardium:

- (a) The added regurgitant volume rapidly increases the **End-Diastolic Volume (EDV)** of the left ventricle.
- (b) According to the Frank-Starling law of the heart, stretching the myocardial fibers increases their force of contraction, which enhances total stroke volume ($SV = EDV - ESV$).
- (c) This increased total stroke volume helps compensate for the retrograde leak, maintaining a sufficient forward stroke volume to sustain systemic perfusion before structural failure or long-term remodeling occurs.

Final Answer: Increased End-Diastolic Volume, Increased Stroke Volume

Answer: (C)

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Q6.

Solution

Concept: Arterial compliance ($C = \frac{\Delta V}{\Delta P}$) represents the capacity of large elastic vessels (like the aorta) to expand and cushion the volume of blood ejected during cardiac systole. Pulse pressure ($PP = \text{Systolic} - \text{Diastolic}$) is inversely proportional to this compliance.

Solution:

Let's evaluate the hemodynamic consequences of a isolated decrease in large vessel compliance:

- (a) During ventricular ejection (systole), a stiff, non-compliant aorta cannot expand to accommodate the stroke volume. This causes a steep rise in pressure, resulting in an ****elevated systolic blood pressure****.
- (b) During diastole, the rigid aorta lacks the elastic recoil required to sustain pressure while blood flows into the peripheral vasculature. As a result, aortic pressure drops rapidly, leading to a ****decreased diastolic blood pressure****.
- (c) This combination of elevated systolic and reduced diastolic pressures significantly increases the total pulse pressure wave profile.

Final Answer: Increased Systolic Pressure, Decreased Diastolic Pressure

Answer: (B)

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Q7.

Solution

Concept: Sinoatrial (SA) node pacemaker cells generate automaticity via a slow diastolic depolarization during Phase 4. The funny current (I_f), conducted through hyperpolarization-activated cyclic nucleotide-gated (HCN) channels, is a mixed sodium-potassium inward current activated at hyperpolarized potentials at the end of repolarization.

Solution:

Let's analyze the effects of blocking the HCN channels in pacemaking tissue:

- (a) The inward flow of positive ions via I_f normally depolarizes the cell membrane from its maximum diastolic potential toward the threshold required to fire an action potential.
- (b) Applying a selective I_f blocker slows this inward current, which significantly reduces the slope and rate of Phase 4 spontaneous diastolic depolarization.
- (c) Because it takes longer for the pacemaker cells to reach the firing threshold, the heart rate slows down (bradycardia), while the configuration of the active upstroke (Phase 0, mediated by L-type calcium channels) remains unchanged.

Final Answer: Marked reduction in the rate of Phase 4 spontaneous diastolic depolarization

Answer: (B)

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Q8.

Solution

Concept: A central venous pressure (CVP) or right atrial pressure waveform displays three positive waves (*a*, *c*, and *v*) and two negative descents (*x* and *y*) that correlate with specific mechanical events of the cardiac cycle.

Solution:

Let's trace the physiological origins of these waves:

- (a) The *a* wave corresponds to right atrial contraction, and the *c* wave reflects tricuspid valve bulging during early isovolumetric ventricular contraction.
- (b) The subsequent *x* descent represents atrial relaxation and the downward displacement of the tricuspid valve floor during ventricular ejection.
- (c) The *v* wave reflects the steady accumulation of systemic venous return filling the right atrium while the tricuspid valve remains completely closed during ventricular systole. The peak of the *v* wave marks the point of maximum atrial filling just before the tricuspid valve opens to begin ventricular diastole.

Final Answer: Venous return filling the atrium against a closed tricuspid valve

Answer: (C)

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Q9.

Solution

Concept: Ascending to high altitude decreases ambient barometric pressure, lowering arterial oxygen tension (P_aO_2) and triggering peripheral chemoreceptors to drive compensatory hyperventilation. This hyperventilation clears CO_2 , causing systemic respiratory alkalosis.

Solution:

Let's track the compensatory adjustments within the cerebrospinal fluid (CSF):

- (a) Carbon dioxide (CO_2) readily diffuses across the blood-brain barrier. The drop in arterial P_aCO_2 causes CO_2 to leave the brain tissue and CSF, creating a parallel respiratory alkalosis inside the medulla.
- (b) This alkaline shift inhibits central chemoreceptors, acting as an electrical brake that limits further compensatory hyperventilation.
- (c) To adapt and allow hyperventilation to continue, choroid plexus epithelial cells actively transport bicarbonate (HCO_3^-) out of the CSF into the blood. This reduction in CSF bicarbonate helps restore local pH toward normal, removing the inhibitory brake on ventilation.

Final Answer: Active pumping of HCO_3^- out of the CSF into the bloodstream

Answer: (B)

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Q10.

Solution

Concept: Functional Residual Capacity (FRC) represents the volume of air remaining in the lungs at the end of a normal tidal expiration. FRC is determined by the balance between two opposing forces: the outward elastic recoil of the chest wall and the inward elastic recoil of the lung parenchyma.

Solution:

Let's analyze the mechanical changes caused by progressive pulmonary fibrosis:

- (a) Pulmonary fibrosis deposits excess collagen fibers throughout the alveolar interstitium, which significantly reduces lung compliance and increases the lung's inward elastic recoil force.
- (b) Because the inward recoil force of the lung is enhanced, it pulls more strongly against the outward recoil of the chest wall.
- (c) This shift in mechanical equilibrium pulls the system to a lower resting volume, resulting in a ****decreased Functional Residual Capacity (FRC)****.

Final Answer:

Answer: (B)

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Q11.

Solution

Concept: The West zones model describes regional variations in pulmonary blood flow caused by the interaction of gravity, pulmonary arterial pressure (P_a), pulmonary venous pressure (P_v), and alveolar air pressure (P_A).

Solution:

Let's define the hemodynamic relationships across the zones:

- (a) In an upright lung, hydrostatic pressure drops moving toward the apex. In **Zone 1** (near the apex), pulmonary arterial pressure is at its lowest point.
- (b) This creates a relationship where alveolar air pressure exceeds the intravascular pressures ($P_A > P_a > P_v$).
- (c) As a result, the high alveolar pressure compresses the delicate pulmonary capillaries, collapsing the vessels and creating a functional dead space with minimal perfusion. Under normal physiological conditions, Zone 1 is small, but it can expand during severe hypotension or positive pressure ventilation.

Final Answer: Alveolar pressure exceeds arterial pressure ($P_A > P_a > P_v$)

Answer: (B)

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Q12.

Solution

Concept: The corticopapillary osmotic gradient is established by the countercurrent multiplier system of the loop of Henle and urea recycling, allowing the kidneys to excrete concentrated urine under the influence of antidiuretic hormone (ADH).

Solution:

Let's evaluate the consequences of blocking the apical NKCC2 cotransporter (e.g., via loop diuretics):

- (a) The NKCC2 cotransporter actively transports electrolytes (Na^+ , K^+ , and Cl^-) out of the lumen of the thick ascending limb into the medullary interstitium.
- (b) Inhibiting this transporter prevents solute accumulation in the medulla, which ****obliterates the hyperosmotic medullary gradient****.
- (c) Without this osmotic gradient, water cannot be drawn out of the collecting ducts via aquaporins, even in the presence of high ADH levels. Consequently, the kidneys lose their maximum concentrating ability, producing a large volume of dilute urine.

Final Answer: Gradient is obliterated; maximum urine concentrating ability is lost.

Answer: (B)

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Q13.

Solution

Concept: Glomerular capillary hydrostatic pressure (P_G) and single-nephron glomerular filtration rate (SNGFR) are regulated by the relative resistances of the afferent and efferent arterioles. The Filtration Fraction ($FF = \frac{GFR}{RPF}$) represents the proportion of renal plasma flow filtered across the glomerular barrier.

Solution:

Let's analyze the effects of selectively increasing efferent arteriolar resistance (R_e):

- Constricting the efferent arteriole creates a constriction downstream of the glomerulus. This restricts blood outflow from the capillary bed, which ****increases the glomerular capillary hydrostatic pressure (P_G)**** and maintains GFR despite a drop in renal blood flow.
- While GFR is supported or elevated by the higher pressure, total Renal Plasma Flow (RPF) decreases due to the overall increase in renal vascular resistance.
- Because GFR is preserved or increased while RPF falls, the ratio of filtration to plasma flow—the ****Filtration Fraction (FF)****—increases significantly.

Final Answer: Increased P_G , Increased FF

Answer: (B)

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Q14.

Solution

Concept: Proximal convoluted tubule (PCT) cells reclaim approximately 80% of filtered bicarbonate (HCO_3^-). Because the apical membrane is impermeable to bicarbonate ions, HCO_3^- must be converted into carbon dioxide (CO_2) within the tubular lumen to cross into the cell interior.

Solution:

Let's identify the enzymatic step that catalyzes this conversion in the lumen:

- Hydrogen ions (H^+) secreted by the Na^+/H^+ exchanger (NHE3) combine with filtered HCO_3^- to form carbonic acid (H_2CO_3) in the luminal fluid.
- Carbonic Anhydrase IV (CA-IV)** is a specialized, GPI-anchored membrane-bound enzyme attached to the apical brush border. It catalyzes the rapid breakdown of luminal H_2CO_3 into water and carbon dioxide ($\text{H}_2\text{O} + \text{CO}_2$).
- The resulting CO_2 gas diffuses across the cell membrane, where cytosolic Carbonic Anhydrase II (CA-II) converts it back into bicarbonate and a hydrogen ion, completing the reclamation cycle.

Final Answer: Membrane-bound Carbonic Anhydrase IV

Answer: (B)

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Q15.

Solution

Concept: Adrenocorticotrophic hormone (ACTH) regulates the synthesis and secretion of glucocorticoids (such as cortisol) within the zona fasciculata of the adrenal cortex by binding to highly specific cell-surface receptors.

Solution:

Let's trace the secondary intracellular signaling cascade activated by ACTH:

- (a) Binding of ACTH to the melanocortin 2 receptor (MC2R) induces a conformational change that activates a stimulatory G-protein subunit ($G_{\alpha s}$).
- (b) The activated $G_{\alpha s}$ subunit stimulates the membrane enzyme adenylyl cyclase, which converts ATP into cyclic adenosine monophosphate (cAMP).
- (c) Elevated intracellular cAMP activates Protein Kinase A (PKA). PKA then phosphorylates cholesterol ester hydrolase and increases the transcription of steroidogenic acute regulatory (StAR) protein, moving cholesterol into the mitochondria to initiate steroid hormone synthesis.

Final Answer: Activation of $G_{\alpha s}$ followed by Adenylyl Cyclase and cAMP generation

Answer: (B)

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Q16.

Solution

Concept: The Calcium-Sensing Receptor (CaSR) on parathyroid chief cells is a G-protein coupled receptor that monitors extracellular ionized calcium levels. Under normal conditions, calcium binding activates the receptor, inhibiting parathyroid hormone (PTH) synthesis and secretion.

Solution:

Let's analyze the effects of a CaSR antagonist (calcilytic agent):

- (a) The antagonist blocks calcium from binding to the CaSR, preventing receptor activation. This mimics a state of severe hypocalcemia, removing the inhibitory brake on the parathyroid gland.
- (b) Consequently, the parathyroid chief cells increase the synthesis and **secretion of parathyroid hormone (PTH)**.
- (c) Elevated systemic PTH acts on the distal convoluted tubules of the kidneys to **increase renal calcium reabsorption** (reducing urinary clearance) while simultaneously decreasing phosphate reabsorption in the proximal tubules to increase phosphate excretion.

Final Answer: Stimulation of PTH secretion, increased renal calcium reabsorption

Answer: (B)

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Q17.

Solution

Concept: Long-term potentiation (LTP) in the hippocampus is a cellular mechanism underlying learning and memory storage. It relies on the activation of N-methyl-D-aspartate (NMDA) receptor complexes on postsynaptic membranes, which act as coincidence detectors requiring both neurotransmitter binding and postsynaptic depolarization.

Solution:

Let's examine the ion gating mechanism of the NMDA receptor channel pore:

- (a) At resting membrane potentials, even when glutamate binds to the NMDA receptor, ions cannot flow through the channel pore because it is physically blocked by an extracellular **magnesium ion (Mg^{2+})**.
- (b) High-frequency tetanic stimulation of the Schaffer collaterals triggers a sustained release of glutamate that strongly activates neighboring AMPA receptors, causing a strong depolarization of the postsynaptic CA1 pyramidal membrane.
- (c) This positive intracellular charge electrostatically repels and expels the Mg^{2+} ion from the NMDA receptor channel pore. Once unblocked, calcium ions (Ca^{2+}) can enter the cell, activating downstream protein kinases that strengthen the synapse.

Final Answer: Magnesium ion (Mg^{2+})

Answer: (B)

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Answer Key

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

