NEET PG Physiology Sample Paper-6

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 participating in a wilderness survival course loses access to fresh water. After 36 hours of acute water deprivation, which of the following changes in the cellular dynamics of her principal cells in the late distal tubule and collecting duct is most accurate?

(A) Increased basolateral insertion of Aquaporin-3 and Aquaporin-4 via a cAMP-independent pathway

(B) Translocation of Aquaporin-2 vesicles to the apical membrane mediated by $G\alpha_s$ protein coupled receptor activation

(C) Decreased phosphorylation of Aquaporin-2 by protein kinase A due to receptor desensitization

(D) Direct activation of protein kinase C leading to endocytosis of luminal Aquaporin-2 channels

Q2. A 45-year-old male with long-standing uncontrolled hypertension undergoes an echocardiogram, which demonstrates concentric left ventricular hypertrophy. At the cellular level, this adaptation is primarily driven by a shift in gene expression. Which of the following best describes the physiological basis of this hypertrophic response?

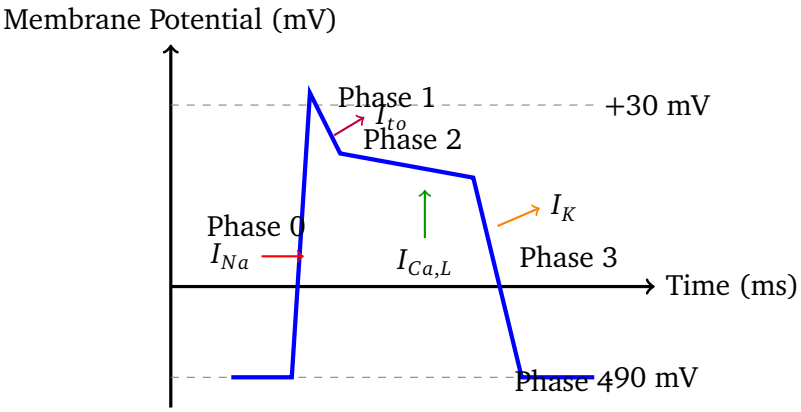
(A) Upregulation of fast α-myosin heavy chain (α-MHC) to increase the velocity of contraction under high afterload

(B) Re-expression of the fetal gene program including β-myosin heavy chain (β-MHC) and atrial natriuretic peptide (ANP)

(C) Downregulation of angiotensin II type 1 (AT_1) receptors to prevent further mechanical stretch transduction

(D) Selective activation of the calcineurin-NFAT pathway resulting exclusively in eccentric replication of sarcomeres in series

Q3. An experimental physiologist isolates a single mammalian cardiac ventricular myocyte to map its action potential. The schematic diagram below demonstrates the major ionic currents corresponding to specific phases of the cardiac action potential:



During Phase 2 (the plateau phase), which of the following accurately describes the net balance of charge movement across the sarcolemma?

(A) Inward I_{Na} through fast voltage-gated sodium channels exactly balances outward I_{to} through transient outward potassium channels

(B) Inward $I_{Ca,L}$ through L-type calcium channels matches the outward potassium efflux primarily via I_{Kr} and I_{Ks} channels

(C) Rapid outward calcium extrusion via the NCX exchanger balances the passive inward leak of chloride ions

(D) Inward rectifier potassium current (I_{K1}) reaches its peak conductance, overpowering all depolarizing currents

Q4. A 28-year-old female presents with acute respiratory distress due to a severe exacerbation of bronchial asthma. Arterial blood gas analysis reveals significant hypoxemia. In response to regional alveolar hypoxia, her pulmonary

vasculature undergoes localized vasoconstriction. What is the fundamental cellular mechanism behind this physiological phenomenon?

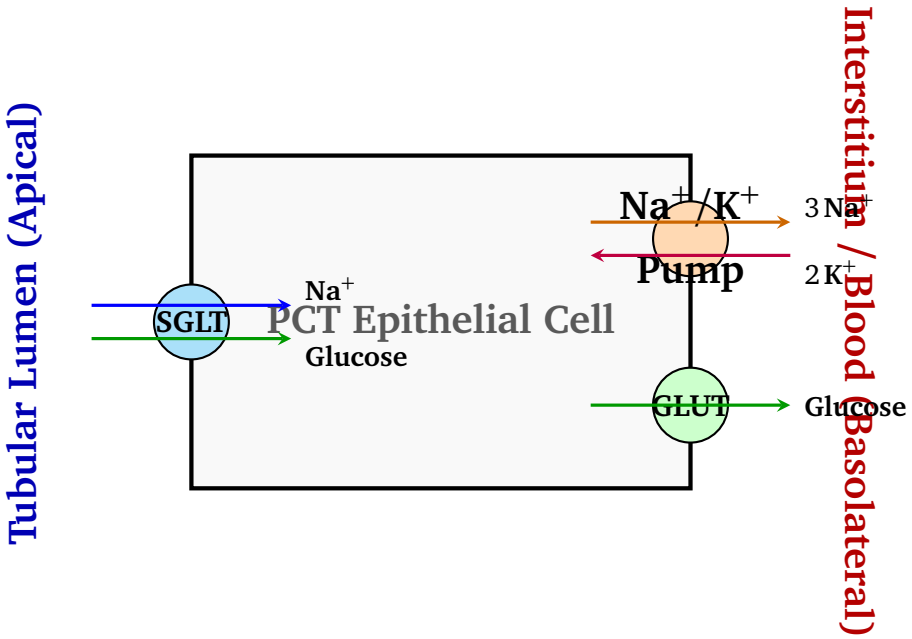
(A) Hypoxia inhibits voltage-gated potassium channels (K_V) in pulmonary artery smooth muscle cells, causing depolarization and calcium influx via L-type calcium channels

(B) Hypoxia stimulates endothelial nitric oxide synthase (eNOS), causing a paradoxically localized depletion of cyclic GMP within smooth muscle cells

(C) Hypoxia hyperpolarizes the smooth muscle membrane by opening ATP-sensitive potassium channels (K_{ATP}), triggering store-operated calcium entry

(D) Hypoxia induces immediate down-regulation of endothelin-1 receptors, switching the vascular tone from dilation to structural constriction

Q5. A researcher utilizes a specialized microelectrode setup to measure transport across the basolateral and apical membranes of the proximal convoluted tubule (PCT). The diagram below illustrates the active transport mechanisms involved in handling filtered sodium and glucose:



If a pharmacological inhibitor completely blocks the basolateral Na^+/K^+-

ATPase pump shown above, what will be the immediate downstream consequence on the luminal transport of glucose?

(A) Luminal glucose transport will increase due to the compensatory upregulation of apical GLUT-2 transporters

(B) Luminal glucose transport will stop because the intracellular sodium gradient required to drive SGLT is dissipated

(C) Luminal glucose transport will remain unaffected since SGLT operates purely via primary active transport utilizing luminal ATP hydrolysis

(D) Luminal glucose transport will reverse direction, actively secreting glucose from the intracellular compartment into the lumen via SGLT

Q6. A 62-year-old postmenopausal female presents with severe proximal muscle weakness, central obesity, and purple striae on her abdomen. Laboratory analysis reveals highly elevated serum cortisol levels and low plasma ACTH levels. Which of the following cellular changes is directly responsible for the muscle weakness seen in this patient?

(A) Cortisol-induced inhibition of glucose uptake in skeletal muscle via downregulation of GLUT-4 transporters along with accelerated myofibrillar protein degradation

(B) Cortisol-mediated hyperstabilization of lysosomal membranes, preventing the normal turnover of structural sarcomeric components

(C) Upregulation of muscle-specific glycogen synthase leading to pathological glycogen storage myopathy

(D) Competitive inhibition of acetylcholine binding at the postsynaptic nicotinic receptors of the neuromuscular junction

Q7. A patient presents with a resting heart rate of 45 beats per minute. An electrocardiogram (ECG) reveals regular P waves and regular QRS complexes, but there is complete dissociation between them, confirming a third-degree (complete) AV block. If the ventricular rate is driven by an idioventricular pacemaker site located in the Purkinje fibers, which of the following

ionic currents is primarily responsible for the phase 4 spontaneous diastolic depolarization in these pacemaker cells?

(A) The rapid inward calcium current ($I_{Ca,L}$) activated at highly positive potentials

(B) The hyperpolarization-activated cyclic nucleotide-gated (HCN) funny current (I_f)

(C) The outward delayed rectifier potassium current (I_{Kr}) running in reverse

(D) The sodium-calcium exchanger (I_{NCX}) operating exclusively in its reverse-mode configuration

Q8. A healthy 25-year-old male ascends rapidly from sea level to an altitude of 4,000 meters. Within hours, his peripheral chemoreceptors detect changes in his arterial blood, resulting in a marked increase in alveolar ventilation. Which of the following profiles accurately describes his arterial blood gas and acid-base status shortly after arrival at high altitude?

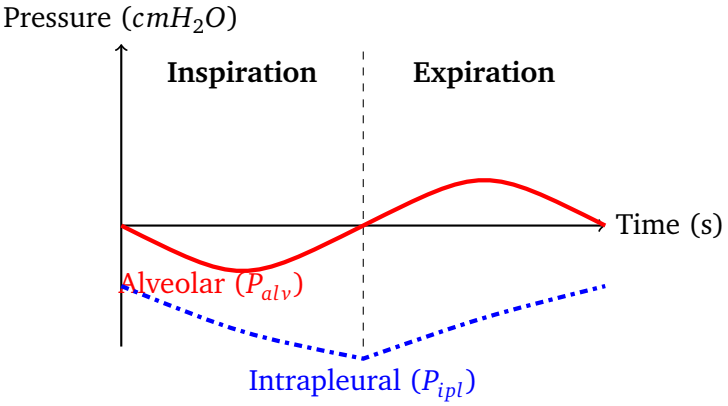
(A) Decreased PaO_2, Increased $PaCO_2$, Decreased arterial pH

(B) Decreased PaO_2, Decreased $PaCO_2$, Increased arterial pH

(C) Normal PaO_2, Decreased $PaCO_2$, Decreased arterial pH

(D) Decreased PaO_2, Decreased $PaCO_2$, Decreased arterial pH due to rapid renal bicarbonate excretion

Q9. An experimental setup measures the changes in intrapleural pressure, alveolar pressure, and lung volume during a normal, quiet respiratory cycle. The diagram below represents these parameters synchronized across inspiration and expiration:



Based on the mechanics illustrated in the diagram, at what point in the respiratory cycle is the transpulmonary pressure ($P_{tp} = P_{alv} - P_{ipl}$) at its maximum value?

(A) At the exact midpoint of active inspiration

(B) At the end of a quiet inspiration, just before expiration begins

(C) At the exact midpoint of passive expiration

(D) At the very beginning of inspiration, when alveolar pressure is zero

Q10. A 70-year-old male with severe emphysema undergoes pulmonary function testing. Due to the destruction of alveolar septa and loss of elastic recoil, his pressure-volume curves deviate significantly from normal. Which of the following physiological parameters is characteristically decreased in this patient compared to a healthy age-matched individual?

(A) Static lung compliance (C_L)

(B) Residual Volume (RV)

(C) Diffusing capacity of the lung for carbon monoxide (DL_{CO})

(D) Functional Residual Capacity (FRC)

Q11. A 58-year-old male presenting with a suspected retroperitoneal tumor undergoes a comprehensive metabolic and renal evaluation. His 24-hour urine collection and blood samples yield the following parameters: Plasma concentration of Substance X (P_x) = 2.0 mg/L Urine concentration of Substance X (U_x) = 100 mg/L Urine flow rate (V) = 2.0 mL/min If Substance X is known to be freely filtered at the glomerulus, with 30% of the filtered load being actively reabsorbed in the proximal tubules, what is the calculated Glomerular Filtration Rate (GFR) for this patient?

(A) 70 mL/min

(B) 100 mL/min

(C) 142.8 mL/min

(D) 200 mL/min

Q12. A 34-year-old female experiences severe postpartum hemorrhage, losing approximately 1.5 liters of blood before localized hemostasis is achieved. Her mean arterial pressure drops to 65 mmHg. Which of the following immediate neuroendocrine reflexes is activated to restore systemic blood pressure?

(A) Increased firing rate of afferents from the carotid sinus and aortic arch baroreceptors

(B) Decreased sympathetic outflow to the afferent arterioles of the kidney, maximizing GFR

(C) Decreased firing rate of high-pressure baroreceptors leading to disinhibition of the medullary vasomotor center

(D) Enhanced secretion of atrial natriuretic peptide (ANP) from the cardiac atria to induce systemic venoconstriction

Q13. A 54-year-old male presents with a two-month history of worsening polyuria, polydipsia, and unexplained weight loss. Fasting plasma glucose is 185 mg/dL, confirming a diagnosis of Type 2 Diabetes Mellitus. When his blood glucose concentration spikes to 320 mg/dL postprandially, glucose appears in his urine. The physiological mechanism underlying this renal glucosuria is:

(A) Downregulation of the sodium-glucose cotransporter 2 (SGLT2) gene expression in response to high insulin levels

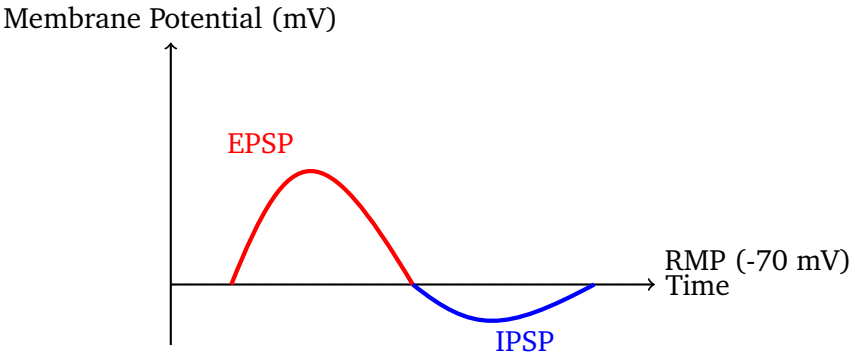
(B) Complete saturation of the apical sodium-glucose cotransporters (T_m) along the proximal convoluted tubule

(C) An increase in the tight junction permeability of the thin descending limb to glucose molecules

(D) Direct competitive inhibition of SGLT1 transporters by elevated ketone bodies in the glomerular filtrate

Q14. A neurophysiology laboratory examines the synaptic transmission characteristics of the central nervous system. The diagram below visualizes the

generation of an Excitatory Postsynaptic Potential (EPSP) versus an Inhibitory Postsynaptic Potential (IPSP) at a postsynaptic membrane:



Which of the following ionic shifts across the postsynaptic membrane is correctly paired with the generation of the potentials depicted above?

(A) EPSP: Net influx of Cl^- ions; IPSP: Net influx of Na^+ ions

(B) EPSP: Net efflux of K^+ ions; IPSP: Net influx of Ca^{2+} ions

(C) EPSP: Net influx of Na^+ or Ca^{2+} ions; IPSP: Net influx of Cl^- ions or net efflux of K^+ ions

(D) EPSP: Selective closing of ligand-gated cation channels; IPSP: Opening of voltage-gated sodium channels

Q15. A 23-year-old male is brought to the emergency department after sustaining a deep stab wound to his mid-back. Neurological examination reveals a complete hemisection of the left half of the spinal cord at the T8 vertebral level (Brown-Séquard syndrome). Which of the following sensory or motor deficits will be observed below the level of the lesion?

(A) Loss of voluntary motor function on the right side, and loss of pain and temperature sensation on the left side

(B) Loss of voluntary motor function on the left side, and loss of proprioception and vibratory sensation on the right side

(C) Loss of voluntary motor function on the left side, and loss of pain and temperature sensation on the right side

(D) Loss of proprioception on the right side, and loss of pain and temperature sensation on the left side

Q16. A 30-year-old female presents with a distinct cluster of symptoms including cold intolerance, bradycardia, weight gain despite poor appetite, and a generalized slowing of deep tendon reflexes. A diagnosis of primary hypothyroidism is made. Which of the following changes in specific cellular metabolic indicators is expected in this patient's tissues?

(A) Increased transcription of the gene encoding the Na^+/K^+-ATPase pump

(B) Decreased systemic basal oxygen consumption (VO_2) and down-regulation of beta-1 adrenergic receptors in cardiac tissue

(C) Increased systemic uncoupling protein-1 (UCP-1) expression in brown adipose tissue

(D) Accelerated hepatic cholesterol clearance via upregulation of low-density lipoprotein (LDL) receptors

Q17. An investigator tests the vascular responsiveness of isolated canine coronary artery rings in an organ bath. The addition of acetylcholine (ACh) to an intact vessel ring induces rapid vasodilation. However, when the inner endothelial lining of the vessel ring is mechanically removed prior to the experiment, the addition of ACh causes vasoconstriction instead. This finding underscores that endothelial-dependent relaxation requires the synthesis of a gaseous signaling molecule. What is the intracellular downstream target of this endothelial-derived gaseous molecule within the underlying vascular smooth muscle cells?

(A) Membrane-bound Adenlyl Cyclase, which elevates intracellular cAMP levels

(B) Soluble Guanylyl Cyclase (sGC), which increases intracellular cGMP levels and activates Protein Kinase G (PKG)

(C) Phospholipase C (PLC), which generates inositol trisphosphate (IP_3) and diacylglycerol (DAG)

(D) Sarcoplasmic Reticulum Ca^{2+}-ATPase (SERCA), via direct inhibitory phosphorylation

Detailed Solutions

Q1.

Solution

Concept:

The primary mechanism regulating water reabsorption in the renal collecting system relies on antidiuretic hormone (ADH), also called arginine vasopressin (AVP). During severe water deprivation, high plasma osmolality triggers ADH release from the posterior pituitary. ADH travels via the bloodstream to bind specifically to basolateral V2 receptors on the principal cells of the late distal tubule and collecting duct, initiating an intracellular signaling cascade.

Solution:

(a) The binding of ADH to the V2 receptor activates a heterotrimeric G-protein, specifically the $G\alpha_s$ subunit, which directly stimulates the membrane-bound enzyme adenylyl cyclase.

(b) Adenylyl cyclase catalyzes the conversion of intracellular ATP into cyclic adenosine monophosphate (cAMP), acting as a potent second messenger.

(c) Elevated cAMP levels activate Protein Kinase A (PKA). Active PKA phosphorylates specific serine residues on intracellular vesicles containing Aquaporin-2 (AQP2) water channels.

(d) This phosphorylation promotes the exocytosis and translocation of AQP2-bearing vesicles, leading to their direct insertion into the apical (luminal) membrane of the principal cells.

(e) This massive increase in apical water permeability allows water to flow passively along the osmotic gradient from the tubule lumen into the hypertonic medullary interstitium, conserving water.

(f) In contrast, Aquaporin-3 and Aquaporin-4 channels are constitutively expressed on the basolateral membrane and do not undergo acute ADH-dependent regulated translocation.

Final Answer: Translocation of Aquaporin-2 vesicles to the apical membrane mediated by $G\alpha_s$ protein coupled receptor activation.

Answer: (B) Go Back to Question 1

Q2.

Solution

Concept:

Myocardial hypertrophy is the adaptive response of the heart to a chronic increase in hemodynamic workload. Chronic arterial hypertension creates a persistent pressure overload (increased afterload), forcing the left ventricle to develop higher wall stress to eject blood. To minimize this wall stress, myocytes undergo parallel addition of sarcomeres, leading to concentric left ventricular hypertrophy characterized by wall thickening without chamber dilation.

Solution:

(a) Chronic mechanical stretch and neurohumoral factors like angiotensin II activate intracellular hypertrophic signaling cascades in adult ventricular myocytes.

(b) This signaling induces a qualitative shift in gene expression known as the re-expression of the fetal gene program, which alters structural and physiological proteins.

(c) In adult rodents and human ventricles, this includes a prominent shift from the fast α-myosin heavy chain (α-MHC) isoform to the slower β-myosin heavy chain (β-MHC) isoform.

(d) The β-MHC isoform has lower ATPase activity, which decreases the velocity of shortening but significantly improves thermodynamic efficiency, letting the wall sustain stress with less oxygen consumption.

(e) Along with structural alterations, adult myocytes re-express fetal secretory proteins, notably atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP), to mitigate overload.

(f) Eccentric hypertrophy involves adding sarcomeres in series rather than in parallel, which expands chamber volume under volume overload, distinct from the concentric model.

Final Answer: Re-expression of the fetal gene program including β-myosin heavy chain (β-MHC) and atrial natriuretic peptide (ANP).

Answer: (B) Go Back to Question 2

Q3.

Solution

Concept:

The ventricular action potential possesses a prolonged plateau phase (Phase 2) that lasts several hundred milliseconds, distinguishing it from skeletal muscle potentials. This prolonged depolarization prevents tetanic contraction and ensures adequate time for ventricular emptying. Electrophysiologically, the plateau represents a delicate, dynamic equilibrium where inward depolarizing currents perfectly counter outward repolarizing currents.

Solution:

(a) Phase 0 is initiated by the rapid influx of Na^+ through voltage-gated sodium channels (I_{Na}), followed by brief transient repolarization in Phase 1 driven by outward potassium current (I_{to}).

(b) As Phase 2 establishes, voltage-gated L-type calcium channels (Ca_V 1.2) open in response to depolarization, creating a sustained inward calcium current ($I_{Ca,L}$).

(c) Simultaneously, delayed rectifier potassium channels open, generating outward potassium currents classified into rapid (I_{Kr}) and slow (I_{Ks}) components.

(d) The net movement of positive charges entering the cell via L-type calcium channels balances the positive charges leaving via potassium channels, holding the membrane potential steady near 0 mV.

(e) Calcium entering during Phase 2 plays a pivotal role in triggering calcium-induced calcium release (CICR) from the sarcoplasmic reticulum via ryanodine receptors, initiating contraction.

(f) Phase 2 terminates as L-type calcium channels slowly inactivate while delayed rectifier potassium currents remain active, shifting the net balance toward repolarization (Phase 3).

Final Answer: Inward $I_{Ca,L}$ through L-type calcium channels matches the outward potassium efflux primarily via I_{Kr} and I_{Ks} channels.

Answer: (B) Go Back to Question 3

Q4.

Solution

Concept:

Hypoxic pulmonary vasoconstriction (HPV) is a highly specialized intrinsic physiological mechanism unique to the pulmonary vasculature. Unlike systemic blood vessels, which dilate in response to hypoxia to increase local perfusion, pulmonary arterioles constrict. This adaptive response shunts blood away from poorly ventilated or hypoxic alveolar regions toward well-ventilated segments, optimizing ventilation-perfusion matching.

Solution:

(a) The primary sensors for alveolar hypoxia reside within the pulmonary artery smooth muscle cells (PASMCs) and involve mitochondrial redox state alterations.

(b) Under hypoxic conditions, a decrease in alveolar oxygen tension inhibits specific voltage-gated potassium channels (K_V), particularly $K_V1.5$ and $K_V2.1$, present on the PASMC sarcolemma.

(c) Inhibition of these potassium channels decreases the outward potassium efflux, leading to accumulation of positive charge internally and depolarization of the smooth muscle membrane.

(d) This membrane depolarization activates voltage-dependent L-type calcium channels, causing an influx of extracellular calcium ions down their electrochemical gradient into the cytosol.

(e) The elevated intracellular calcium binds to calmodulin, activating myosin light chain kinase (MLCK), which phosphorylates myosin cross-bridges and induces sustained smooth muscle contraction.

(f) This direct, localized, endothelium-independent mechanism limits shunting of de-oxygenated blood through unventilated areas of the lung during acute respiratory pathologies like asthma.

Final Answer: Hypoxia inhibits voltage-gated potassium channels (K_V) in pulmonary artery smooth muscle cells, causing depolarization and calcium influx via L-type calcium channels.

Answer: (A) Go Back to Question 4

Q5.

Solution

Concept:

The reabsorption of filtered solute across epithelial cells in the proximal convoluted tubule utilizes secondary active transport systems. This transport couples the movement of a solute against its concentration gradient to the passive downstream movement of sodium ions. Maintaining a steep sodium gradient across the apical membrane is essential to power these secondary transporters.

Solution:

(a) The primary engine for proximal tubular reabsorption is the Na^+/K^+-ATPase pump located exclusively on the basolateral membrane of the epithelial cell.

(b) This pump actively extrudes three sodium ions out of the cell into the interstitium while pumping two potassium ions inward, expending ATP.

(c) The constant extrusion keeps intracellular sodium concentrations low (around 15 mEq/L) compared to the tubular lumen (around 140 mEq/L), creating a chemical gradient.

(d) It also generates a negative intracellular electrical potential, establishing a strong electrochemical gradient that drives sodium across the apical membrane.

(e) Apical sodium-glucose cotransporters (SGLT) exploit this electrochemical gradient, carrying glucose into the cell against its chemical gradient as sodium enters down its own.

(f) Inhibiting the basolateral Na^+/K^+-ATPase allows intracellular sodium to rise rapidly, abolishing the electrochemical gradient and stopping all secondary active transport of glucose.

Final Answer: Luminal glucose transport will stop because the intracellular sodium gradient required to drive SGLT is dissipated.

Answer: (B) Go Back to Question 5

Q6.

Solution

Concept:

Glucocorticoids like cortisol play a foundational role in maintaining metabolic homeostasis, but chronic pathological excess leads to Cushing's syndrome. Cortisol exerts prominent catabolic effects on skeletal muscle, bone, and connective tissue to mobilize substrates for gluconeogenesis. Prolonged high levels cause muscle wasting and proximal myopathy, presenting as difficulty climbing stairs or rising from a chair.

Solution:

(a) Cortisol acts on target cells by binding to intracellular glucocorticoid receptors, which translocate to the nucleus to regulate specific gene transcription.

(b) In skeletal muscle, cortisol directly downregulates the transcription and translocation of the insulin-responsive glucose transporter, GLUT-4, to the sarcolemma.

(c) This induces local insulin resistance, decreasing glucose uptake and limiting energy substrate availability within the myocyte for metabolic functions.

(d) Simultaneously, cortisol activates the ubiquitin-proteasome pathway by upregulating muscle-specific ubiquitin ligases, including MuRF1 and atrogin-1, which target myofibrillar proteins for degradation.

(e) The resulting breakdown of structural proteins like actin and myosin leads to progressive atrophy of fast-twitch type II muscle fibers.

(f) This combination of restricted glucose uptake and accelerated structural protein breakdown drives the proximal muscle weakness seen in hypercortisolemic patients.

Final Answer: Cortisol-induced inhibition of glucose uptake in skeletal muscle via downregulation of GLUT-4 transporters along with accelerated myofibrillar protein degradation.

Answer: (A) Go Back to Question 6

Q7.

Solution

Concept:

The electrical conduction system of the heart possesses intrinsic automaticity, which is the capacity to generate spontaneous action potentials without external neural stimulation. This automaticity is driven by a slow, progressive depolarization of the membrane potential during Phase 4 of the action potential cycle. While the sinoatrial node is the primary pacemaker, latent pacemakers exist in the AV node and Purkinje system.

Solution:

(a) In a third-degree AV block, atrial impulses cannot pass to the ventricles, prompting a latent ventricular site like the Purkinje fibers to establish an escape rhythm.

(b) The spontaneous diastolic depolarization of Phase 4 in these pacemaker tissues relies heavily on the funny current, designated as I_f.

(c) The I_f current is carried through hyperpolarization-activated cyclic nucleotide-gated (HCN) channels on the cell membrane.

(d) Unlike most voltage-gated channels that open upon depolarization, HCN channels open when the membrane hyperpolarizes at the end of Phase 3 repolarization.

(e) Once open at negative potentials (around -60 mV), HCN channels conduct a mixed inward current dominated by sodium influx (Na^+).

(f) This slow inward leak of positive charge depolarizes the membrane toward the threshold required to activate T-type and L-type calcium channels, triggering the next action potential.

Final Answer: The hyperpolarization-activated cyclic nucleotide-gated (HCN) funny current (I_f).

Answer: (B) Go Back to Question 7

Q8.

Solution

Concept:

Ascending to high altitude exposes the body to a fall in barometric pressure, which decreases the ambient partial pressure of oxygen (PO_2) while maintaining the normal fractional concentration of oxygen. This change lowers the driving pressure for oxygen diffusion across the alveolar-capillary membrane, leading to arterial hypoxemia. This drops the arterial partial pressure of oxygen (PaO_2) and activates peripheral chemoreceptors.

Solution:

(a) The decrease in PaO_2 is sensed by glomus cells in the carotid and aortic bodies, which signal the medullary respiratory center to increase alveolar ventilation.

(b) This hyperventilation increases the clearance of carbon dioxide (CO_2) from the alveoli, causing a drop in the arterial partial pressure of carbon dioxide ($PaCO_2$).

(c) According to the Henderson-Hasselbalch relationship, a reduction in $PaCO_2$ shifts the carbonic acid-bicarbonate equilibrium, lowering hydrogen ion levels and increasing arterial pH above 7.45.

(d) This initial systemic acid-base status is termed uncompensated acute respiratory alkalosis, which develops within hours of ascending to high altitude.

(e) This respiratory alkalosis shifts the oxygen-hemoglobin dissociation curve to the left, increasing hemoglobin's affinity for oxygen to enhance pulmonary oxygen loading.

(f) Renal compensation via bicarbonate excretion takes days to normalize pH, meaning the acute profile shows low PaO_2, low $PaCO_2$, and elevated pH.

Final Answer: Decreased PaO_2, Decreased $PaCO_2$, Increased arterial pH.

Answer: (B) Go Back to Question 8

Q9.

Solution

Concept:

Ventilation relies on cyclic variations in pulmonary pressures relative to atmospheric pressure, which are driven by alterations in thoracic cavity volume. Transpulmonary pressure (P_{tp}) represents the transmural pressure gradient across the lung wall, defined mathematically as alveolar pressure (P_{alv}) minus intrapleural pressure (P_{ipl}). This pressure acts as the distending force holding the lungs open against their natural elastic recoil.

Solution:

(a) At rest before inspiration, P_{alv} is 0 cmH_2O and P_{ipl} is roughly -5 cmH_2O, yielding an initial static transpulmonary pressure of +5 cmH_2O.

(b) Contraction of the diaphragm expands the thoracic cavity, causing intrapleural pressure to drop toward its most negative value (around -8 cmH_2O) at the end of inspiration.

(c) This expansion lowers alveolar pressure below atmospheric level, creating a transient negative pressure that draws air into the lungs.

(d) At the end of a quiet inspiration, airflow ceases momentarily as the lung volume reaches its peak, causing alveolar pressure to return to 0 cmH_2O.

(e) Calculating P_{tp} at this end-inspiratory point ($P_{alv} - P_{ipl} = 0 - (-8) = +8\ cmH_2O$) reveals that transpulmonary pressure reaches its maximum value here.

(f) This maximum transpulmonary pressure corresponds to the peak lung volume and reflects the maximum structural distortion and elastic recoil energy stored within the lung tissue.

Final Answer: At the end of a quiet inspiration, just before expiration begins.

Answer: (B) Go Back to Question 9

Q10.

Solution

Concept:

Pulmonary emphysema is a destructive obstructive lung disease characterized by the permanent enlargement of airspaces distal to the terminal bronchioles. Inflammatory proteases break down elastin fibers within the alveolar septa, damaging the structural framework of the lungs. This loss of elastic tissue increases static lung compliance (C_L), meaning the lungs distend easily but lack elastic recoil.

Solution:

(a) The loss of elastic recoil permits the lungs to expand to larger volumes at a given pressure, causing increases in Residual Volume (RV) and Functional Residual Capacity (FRC).

(b) Due to diminished radial traction, the poorly supported conducting airways tend to collapse during expiration, trapping air and worsening hyperinflation.

(c) Alveolar wall destruction decreases the total surface area available for gas exchange across the blood-air barrier.

(d) This loss of functional alveolar-capillary membrane area decreases the diffusing capacity of the lung for carbon monoxide (DL_{CO}).

(e) DL_{CO} quantification tracks gas transfer efficiency across the alveolar membrane, making a reduced value a hallmark indicator of parenchymal destruction.

(f) Thus, while compliance and volumes increase due to loss of recoil, gas exchange capacity (DL_{CO}) falls significantly in emphysema patients.

Final Answer: Diffusing capacity of the lung for carbon monoxide (DL_{CO}).

Answer: (C) Go Back to Question 10

Q11.

Solution

Concept:

Renal clearance principles dictate the rate at which a substance is removed from the plasma by the kidneys. For any substance that is freely filtered at the glomerulus, the filtered load is calculated as the product of Glomerular Filtration Rate (GFR) and its plasma concentration (P_x). If the substance undergoes subsequent tubular modification, its urinary excretion rate ($U_x \times V$) reflects the net outcome of filtration, reabsorption, and secretion.

Solution:

(a) The problem states that Substance X is freely filtered at the glomerulus, meaning its concentration in the initial Bowman's space filtrate equals its concentration in the plasma ($P_x = 2.0$ mg/L).

(b) The urinary excretion rate of Substance X can be calculated using the given parameters: $U_x = 100$ mg/L and the urine flow rate $V = 2.0$ mL/min.

(c) Multiplying these values yields an excretion rate ($U_x \times V$) of 100 mg/L $\times$ 2.0 mL/min = 200 mg/min.

(d) According to the problem, 30

(e) Expressing this relationship mathematically gives the equation: Excretion Rate = 0.70 $\times$ Filtered Load, which expands to $U_x \times V = 0.70 \times (\text{GFR} \times P_x)$.

(f) Substituting the known values into the equation produces: 200 mg/min = 0.70 $\times$ (GFR $\times$ 2.0 mg/L). Solving for GFR yields 200/1.4 = 142.85 mL/min, which rounds directly to 142.8 mL/min.

Final Answer: 142.8 mL/min

Answer: (C) Go Back to Question 11

Q12.

Solution

Concept:

Systemic arterial blood pressure is continuously monitored and regulated by specialized mechanoreceptors known as high-pressure baroreceptors. These nerve endings are strategically embedded within the adventitia of the carotid sinuses and the aortic arch. They respond directly to structural stretch caused by changes in transmural pressure, maintaining cardiovascular homeostasis through a rapid, negative-feedback neural reflex loop.

Solution:

(a) Acute postpartum hemorrhage induces a severe reduction in total blood volume, leading to a profound drop in venous return, cardiac output, and systemic mean arterial blood pressure.

(b) The reduction in arterial pressure causes a decrease in the distending transmural force within the carotid sinuses and aortic arch, reducing mechanical stretch on the baroreceptor membranes.

(c) Consequently, the baseline firing rate of action potentials transmitted along the afferent nerve pathways (the glossopharyngeal and vagus nerves) to the medullary cardiovascular center is drastically diminished.

(d) Under normal conditions, these afferent signals stimulate the nucleus tractus solitarius (NTS) to inhibit the caudal ventrolateral medulla, thereby suppressing sympathetic outflow from the rostral ventrolateral medulla.

(e) A decreased firing rate of these high-pressure baroreceptors leads to disinhibition of the medullary vasomotor center, lifting the normal tonic suppression on the sympathetic nervous system.

(f) This reflex increases sympathetic outflow, causing systemic vasoconstriction, venoconstriction, and positive inotropic and chronotropic cardiac responses to preserve perfusion to vital organs.

Final Answer: Decreased firing rate of high-pressure baroreceptors leading to disinhibition of the medullary vasomotor center.

Answer: (C) Go Back to Question 12

Q13.

Solution

Concept:

The renal handling of filtered glucose relies on secondary active transport mechanisms located in the early segments of the proximal convoluted tubule. Under normal physiological conditions, the amount of glucose filtered by the glomeruli increases linearly with plasma glucose concentration because glucose is freely filtered. The proximal tubule reabsorbs all filtered glucose, preventing its excretion in urine.

Solution:

(a) The apical reabsorption of glucose is mediated by sodium-glucose cotransporters (primarily SGLT2 in the early proximal tubule and SGLT1 in the late proximal tubule), which carry glucose against its gradient.

(b) Because these transport systems rely on a fixed number of functional membrane proteins, they exhibit a strict saturation threshold known as the transport maximum (T_m).

(c) In a healthy individual, the renal transport maximum for glucose is approximately 375 mg/min in men and 300 mg/min in women, which corresponds to a plasma glucose threshold of roughly 180 to 200 mg/dL.

(d) When a patient with uncontrolled Type 2 Diabetes Mellitus experiences severe postprandial hyperglycemia up to 320 mg/dL, the total filtered load of glucose outpaces the capacity of these transporters.

(e) Once all available SGLT binding sites are occupied, the transport mechanism reaches complete saturation, meaning any additional filtered glucose cannot be reabsorbed.

(f) This excess, unreabsorbed glucose remains trapped within the tubular fluid and passes through the nephron to be excreted, causing glucosuria and driving osmotic diuresis.

Final Answer: Complete saturation of the apical sodium-glucose cotransporters (T_m) along the proximal convoluted tubule.

Answer: (B) Go Back to Question 13

Q14.

Solution

Concept:

Synaptic transmission within the central nervous system occurs through the binding of neurotransmitters to specific postsynaptic receptors. This interaction triggers the opening or closing of ligand-gated ion channels, altering the ion permeability of the postsynaptic membrane. The direction of the resulting ion flux determines whether the membrane potential depolarizes or hyperpolarizes, generating local graded potentials.

Solution:

(a) An Excitatory Postsynaptic Potential (EPSP) is a local, graded depolarization that shifts the resting membrane potential closer to the threshold required to trigger an action potential.

(b) This depolarization is caused by the binding of excitatory neurotransmitters like glutamate to receptors that open non-selective cation channels, facilitating a net influx of Na^+ or Ca^{2+} ions.

(c) Conversely, an Inhibitory Postsynaptic Potential (IPSP) is a local hyperpolarization that drives the postsynaptic membrane potential further away from the firing threshold, stabilizing the cell.

(d) IPSPs are generated when inhibitory neurotransmitters like GABA or glycine bind to their receptors, promoting an influx of anions or an efflux of cations.

(e) This binding opens ligand-gated chloride channels, driving a net influx of Cl^- ions down their concentration gradient, or opens potassium channels, driving a net efflux of K^+ ions.

(f) The balance of these ionic shifts across the postsynaptic membrane determines the generation of the potentials depicted, reflecting the summation of concurrent synaptic inputs.

Final Answer: EPSP: Net influx of Na^+ or Ca^{2+} ions; IPSP: Net influx of Cl^- ions or net efflux of K^+ ions.

Answer: (C)

Go Back to Question 14

Q15.

Solution

Concept:

A spinal cord hemisection results in a distinct neurological presentation known as Brown-Séquard syndrome. This condition highlights the anatomical organization and decussation patterns of the major ascending sensory pathways and descending motor tracts. Understanding where these pathways cross the midline is essential for localizing clinical deficits relative to the site of a structural lesion.

Solution:

(a) Voluntary motor control is mediated by the descending corticospinal tract. The fibers of the lateral corticospinal tract cross the midline at the pyramidal decussation in the medulla.

(b) Because these motor fibers descend ipsilaterally within the spinal cord, a lesion at the T8 level on the left side causes upper motor neuron signs and voluntary motor loss on the left side below the injury.

(c) Fine touch, vibration, and conscious proprioception travel via the dorsal column-medial lemniscal pathway, which ascends ipsilaterally within the spinal cord before crossing in the medulla.

(d) Damage to the left dorsal columns at the T8 level interrupts these signals from the same side, causing a loss of proprioceptive and vibratory sensations on the left side below the lesion.

(e) Pain and temperature sensations are carried by the spinothalamic tract. These primary afferent fibers enter the spinal cord and cross the midline via the anterior white commissure within one to two spinal segments.

(f) Because the spinothalamic pathway ascends contralaterally, a structural lesion of the left half of the spinal cord at T8 interrupts fibers originating from the right side, causing a loss of pain and temperature on the right side.

Final Answer: Loss of voluntary motor function on the left side, and loss of pain and temperature sensation on the right side.

Answer: (C)

Go Back to Question 15

Q16.

Solution

Concept:

Thyroid hormones, including thyroxine (T_4) and triiodothyronine (T_3), serve as primary systemic regulators of cellular metabolism and gene transcription. They diffuse into target cells, bind to nuclear thyroid hormone receptors, and stimulate the expression of proteins involved in energy expenditure. A deficiency in these hormones, as seen in primary hypothyroidism, decreases the metabolic rate across body tissues.

Solution:

(a) In primary hypothyroidism, the lack of active thyroid hormone compromises gene transcription, reducing the expression of the Na^+/K^+-ATPase pump, which accounts for a large share of baseline cellular energy use.

(b) This reduction in active transport proteins lowers the baseline metabolic rate, causing a prominent decrease in systemic basal oxygen consumption (VO_2) across peripheral tissues.

(c) Thyroid hormones also exert permissive effects on the cardiovascular system by upregulating the transcription of beta-1 adrenergic receptors within myocardial tissue.

(d) The loss of this permissive signaling leads to a marked downregulation of cardiac beta-1 receptors, causing a blunted response to catecholamines that presents clinically as bradycardia.

(e) Reduced metabolic activity decreases heat production, driving clinical manifestations such as marked cold intolerance, weight gain, sluggish deep tendon reflexes, and generalized lethargy.

(f) In addition, the expression of hepatic low-density lipoprotein (LDL) receptors is decreased, slowing cholesterol clearance and leading to hypercholesterolemia in hypothyroid patients.

Final Answer: Decreased systemic basal oxygen consumption (VO_2) and down-regulation of beta-1 adrenergic receptors in cardiac tissue.

Answer: (B)

Go Back to Question 16

Q17.

Solution

Concept:

Vascular tone regulation involves a dynamic interplay between endothelial cells and underlying vascular smooth muscle cells (VSMCs). Endothelial cells respond to chemical stimuli like acetylcholine (ACh) by synthesizing vasoactive factors. This endothelium-dependent relaxation depends on the production and diffusion of nitric oxide (NO), a short-lived gaseous signaling molecule that regulates smooth muscle tone.

Solution:

(a) When acetylcholine binds to muscarinic M_3 receptors on intact endothelial cells, it triggers an increase in intracellular calcium that activates endothelial nitric oxide synthase (eNOS).

(b) Active eNOS converts L-arginine into nitric oxide, which diffuses across the internal elastic lamina into the adjacent vascular smooth muscle cells.

(c) Within the cytoplasm of smooth muscle cells, nitric oxide binds with high affinity to the heme moiety of soluble guanylyl cyclase (sGC), activating the enzyme.

(d) Activated sGC catalyzes the conversion of intracellular GTP into cyclic GMP (cGMP), which acts as an essential second messenger within the smooth muscle architecture.

(e) Accumulating cGMP activates Protein Kinase G (PKG), which phosphorylates proteins to decrease intracellular calcium levels and reduce the calcium sensitivity of the contractile machinery.

(f) If the endothelium is mechanically removed, ACh cannot stimulate NO production; instead, it binds directly to M_3 receptors on smooth muscle cells, causing paradoxical vasoconstriction.

Final Answer: Soluble Guanylyl Cyclase (sGC), which increases intracellular cGMP levels and activates Protein Kinase G (PKG).

Answer: (B)

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Answer Key

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