NEET PG Physiology Sample Paper-4

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 study undergoes acute severe dehydration. On a molecular level, her supraoptic neurons must upregulate the transcription and vesicular trafficking of Aquaporin-2 (AQP2) to the apical membrane of renal principal cells. Which of the following secondary messenger cascades and specific motor proteins is primarily responsible for the rapid exocytosis and targeted apical translocation of these AQP2-bearing vesicles from the endosomal compartment?

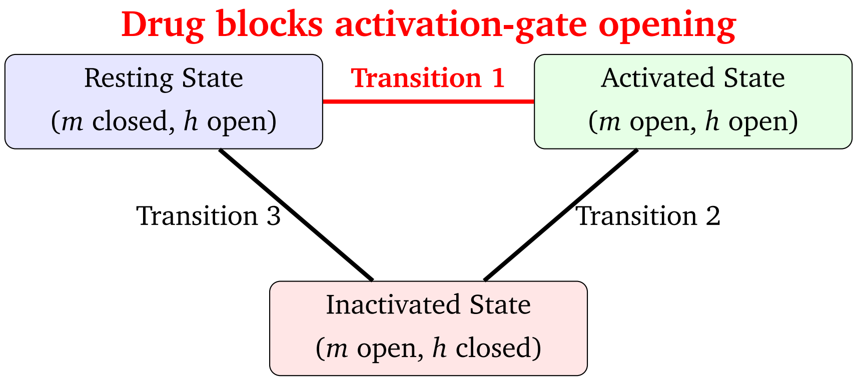
(A) cAMP-dependent Protein Kinase A activation leading to Dynein-mediated retrograde transport.

(B) cGMP-dependent Protein Kinase G activation leading to Kinesin-1-mediated anterograde transport.

(C) cAMP-dependent Protein Kinase A activation leading to Kinesin-mediated anterograde transport along microtubules.

(D) PLC-β activation causing intracellular Ca^{2+} spikes leading to Myosin-V activation on actin filaments.

Q2. An experimental drug molecule, Compound X-99, is applied to an isolated patch of skeletal muscle sarcolemma. Researchers note that while the cell maintains a normal resting membrane potential of −90 mV, a threshold electrical stimulus fails to initiate a propagated action potential. Further analysis reveals that the drug locks the inner activation gate (*m*-gate) of voltage-gated Na^+ channels in the closed position without affecting the inactivation gate (*h*-gate). Based on the channel-state diagram below, which transition is directly blocked by Compound X-99?



(A) State Transition 1

(B) State Transition 2

(C) State Transition 3

(D) None of the transitions are altered; the resting potential is what prevents activation.

Q3. During a laboratory evaluation of cell membrane transport kinetics, the intracellular accumulation rate of a solute S is plotted against its extracellular concentration. It is observed that when metabolic poisons like dinitrophenol (DNP) are added, the maximal transport rate (V_{max}) drops to zero, and transport ceases entirely. However, when the extracellular sodium concentration ($[Na^+]_o$) is altered to match the intracellular concentration ($[Na^+]_i$), the influx of solute S also completely halts despite abundant ATP. This transport mechanism is best categorized as:

(A) Primary Active Transport utilizing a P-type ATPase.

(B) Secondary Active Co-transport (Symport) driven by an electrochemical gradient.

(C) Simple Passive Diffusion via an open ion channel.

(D) Facilitated Diffusion utilizing a stereospecific carrier protein.

Q4. A 62-year-old male with a history of long-standing uncontrolled systemic hypertension presents to the cardiology clinic. Echocardiography demonstrates concentric left ventricular hypertrophy. Which of the following parameters regarding ventricular wall stress (σ), wall thickness (h), intraventricular pressure (P), and internal radius (r) best describes the physiological compensatory adaptation according to the Law of Laplace during early-stage hypertrophy?

(A) An increase in r minimizes σ despite an increased P.

(B) An increase in h acts to normalize σ in the setting of elevated P.

(C) A decrease in h increases σ to augment stroke volume output.

(D) An increase in P directly decreases h to maintain stable ejection fractions.

Q5. A clinical trial participant is administered an experimental class-III antiarrhythmic agent that causes highly selective, dose-dependent prolongation of Phase 2 (the plateau phase) of the ventricular action potential. Which of the following combinations of ion channel modifications best explains this specific electrocardiographic lengthening of the QT interval without altering the resting membrane potential?

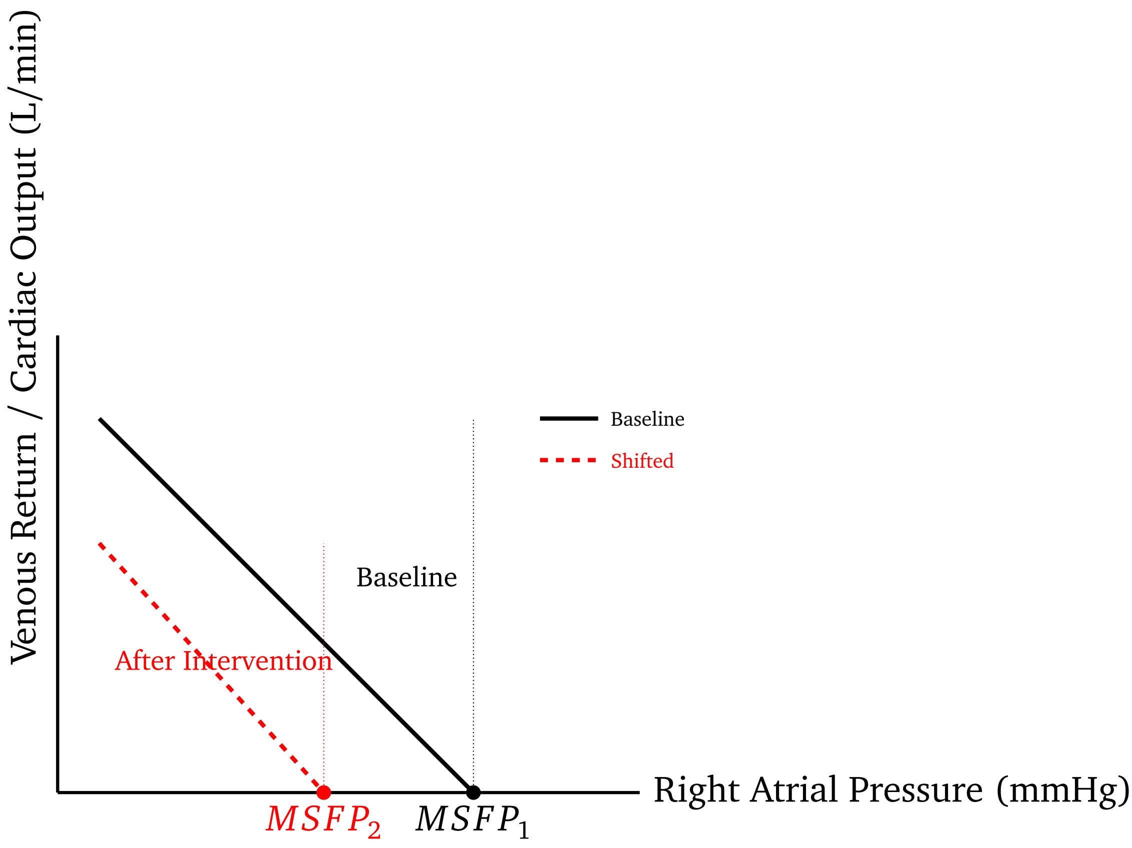
(A) Blockade of I_{K1} channels coupled with early activation of I_{to} channels.

(B) Prolongation of L-type Ca^{2+} channel ($I_{Ca,L}$) opening and/or inhibition of delayed rectifier K^+ channels (I_{Kr}/I_{Ks}).

(C) Upregulation of I_{Na} inward current during Phase 0 depolarization.

(D) Acceleration of the Na^+/K^+ ATPase pump electrogenic exchange rate.

Q6. An intensive care patient is being monitored using a pulmonary artery (Swan–Ganz) catheter. Following a therapeutic intervention, the systemic vascular function curve shifts from the solid line to the dashed line as shown below. Which intervention best explains this hemodynamic change?



(A) Acute intravenous infusion of 2 liters of normal saline.

(B) Administration of a potent venodilator (e.g., Nitroglycerin).

(C) Administration of a pure positive inotrope (e.g., Dobutamine).

(D) Isolated increase in total peripheral resistance (TPR) due to arteriolar constriction.

Q7. During a routine exercise tolerance test, a healthy 28-year-old female athlete increases her workload from rest to maximal exertion. Despite a massive 4-fold increase in her total cardiac output, her mean arterial pressure (MAP) only rises moderately (by roughly 15–20%). This relatively stable blood pressure profile during strenuous physiological exertion is explained by which concurrent systemic mechanism?

(A) A proportional increase in parasympathetic tone to the peripheral vascular beds.

(B) Marked metabolic vasodilation in active skeletal muscles causing a profound drop in total peripheral resistance (TPR).

(C) Baroreceptor resetting that completely shuts off sympathetic output to the heart.

(D) Passive mechanical collapse of large elastic arteries under high flow states.

Q8. A team of high-altitude physiologists studies an unacclimatized lowlander who is rapidly transported to an altitude of 5,000 meters above sea level. Within hours, her arterial blood gas profile reveals severe hypoxemia accompanied by respiratory alkalosis. Which of the following central or peripheral sensing loops is primarily responsible for triggering the compensatory hyperventilation observed in this acute stage?

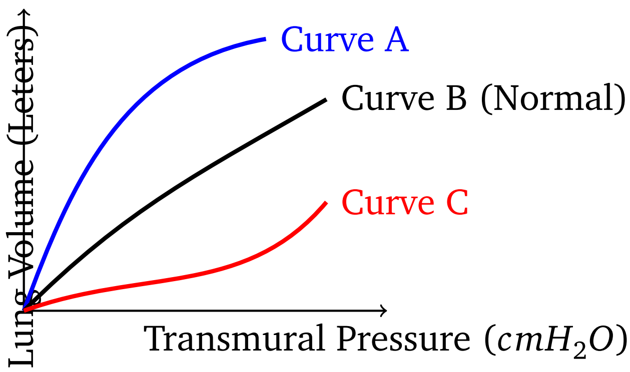
(A) Central chemoreceptors sensing a local drop in cerebrospinal fluid pH.

(B) Glomus cells of the carotid bodies sensing a reduction in dissolved arterial PO_2 below 60 mmHg.

(C) Pulmonary stretch receptors triggering the Hering-Breuer deflation reflex.

(D) Aortic body chemoreceptors sensing an elevated carboxyhemoglobin level.

Q9. A clinical pulmonologist evaluates the lung volumes and static compliance loops of four different patients. The diagram below illustrates the pressure-volume relationship curves of normal lung tissue versus various pathological pulmonary states. Which curve corresponds to a patient suffering from severe idiopathic pulmonary fibrosis (IPF)?



(A) Curve A

(B) Curve B (Normal)

(C) Curve C

(D) None of the curves correlate with parenchymal disease states.

Q10. An intensive care physician manages a mechanical ventilation protocol for a patient with acute respiratory distress syndrome (ARDS). The patient is ventilated with a tidal volume (V_T) of 450 mL, and a respiratory rate of 12 breaths/min. Dead space volume (V_D) measured via Bohr's method is 150 mL. If the ventilator settings are adjusted to a V_T of 300 mL and a rate of 18 breaths/min, what change occurs in the calculated alveolar ventilation ($\dot{V}_A$)?

(A) Alveolar ventilation remains perfectly unchanged at 3600 mL/min.

(B) Alveolar ventilation decreases from 3600 mL/min to 2700 mL/min.

(C) Alveolar ventilation increases from 2400 mL/min to 3600 mL/min.

(D) Alveolar ventilation decreases from 3600 mL/min to 1800 mL/min.

Q11. A pharmacologist tests a new loop diuretic compound that inhibits the $Na^+-K^+-2Cl^-$ cotransporter (NKCC2) in the thick ascending limb of Henle. Beyond blocking active sodium chloride reabsorption, this agent completely abolishes the lumen-positive transepithelial potential difference ($\approx +10$ mV) typically found in this segment. As a direct consequence, the renal clearance of which of the following pairs of ions will be most significantly increased?

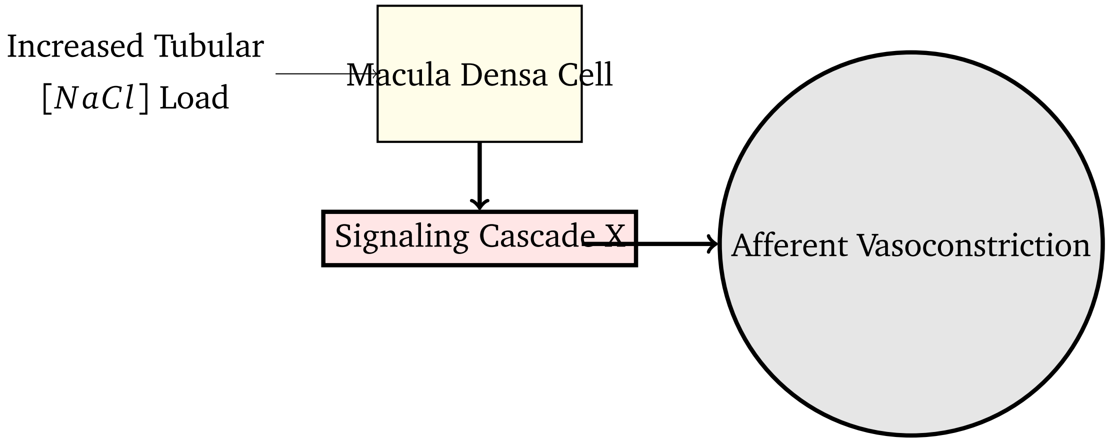
(A) H^+ and HCO_3^-

(B) Ca^{2+} and Mg^{2+}

(C) Glucose and Amino Acids

(D) Phosphate and Silicate

Q12. An experimental animal model is evaluated to understand tubuloglomerular feedback (TGF) mechanisms. The delivery rate of fluid to the macula densa is artificially titrated upwards. The schematic below depicts the juxtaglomerular apparatus (JGA) feedback loop components. Which physiological response sequence is triggered inside the macula densa cells to cause constriction of the adjacent afferent arteriole?



(A) Decreased Na^+ entry → Intracellular swelling → Nitric Oxide release.

(B) Hyperpolarization via K^+ efflux → Prostaglandin E_2 exocytosis.

(C) Increased $Na^+ - K^+ - 2Cl^-$ entry → ATP consumption → Intracellular Adenosine generation and release via basolateral membranes.

(D) Inactivation of stretch-activated Ca^{2+} channels → Reduced renin cleavage.

Q13. A 45-year-old woman is evaluated for a complex acid-base disturbance. Laboratory parameters reveal an arterial blood pH of 7.22, a plasma bicarbonate concentration ($[HCO_3^-]$) of 12 mEq/L, and a partial pressure of carbon dioxide (PCO_2) of 30 mmHg. Serum electrolyte panel reveals: $Na^+ = 140$ mEq/L, $Cl^- = 118$ mEq/L. Which clinical scenario matches this metabolic profile?

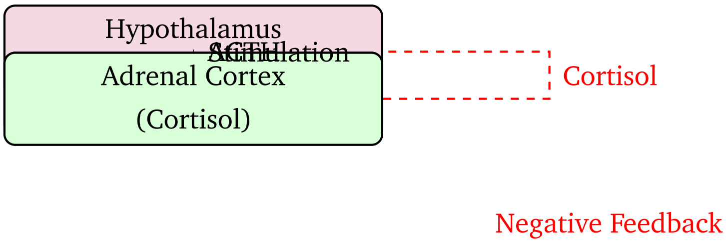
(A) Severe diabetic ketoacidosis (DKA) with accumulation of acetoacetate.

(B) Severe chronic diarrhea resulting in substantial gastrointestinal base bicarbonate loss.

(C) Persistent pernicious vomiting leading to high-volume loss of gastric hydrochloric acid.

(D) Acute respiratory failure secondary to central nervous system depression.

Q14. A 34-year-old female develops severe postpartum hemorrhage requiring multiple blood transfusions. Several months later, she presents with lethargy, cold intolerance, amenorrhea, and failure of lactation. A hormonal axis evaluation is illustrated below. Based on the underlying pathology, which structure represented by Node X is primarily damaged?



(A) Hypothalamus

(B) Node X (Anterior Pituitary)

(C) Adrenal Cortex

(D) Intermediate Zone of the Neurohypophysis

Q15. A molecular biologist isolates pancreatic islet cells to study insulin secretion kinetics. She observes that when glucose concentrations in the perfusion medium are raised, there is an immediate first phase of insulin release, followed by a prolonged secondary phase. If an experimental toxin that selectively blocks the enzyme Glucokinase is added to the system, what happens to the ATP-sensitive K^+ channels (K_{ATP}) and insulin release?

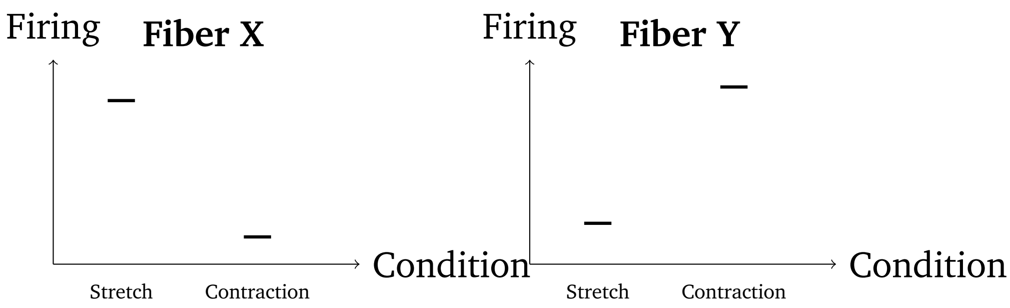
(A) K_{ATP} channels remain open, preventing cell depolarization and completely blocking insulin secretion.

(B) K_{ATP} channels close immediately, triggering massive unregulated insulin release.

(C) K_{ATP} channels undergo hyperpolarization, accelerating the first phase only.

(D) Intracellular calcium drops, forcing immediate exocytosis of remaining zinc-insulin hexamers.

Q16. A clinical neurophysiology study records the discharge frequency of two proprioceptive afferent fibers during passive stretch and active isometric contraction of the biceps brachii muscle. The firing patterns are shown below. Which statement correctly identifies Fiber Type Y?



(A) Fiber Y is a Ia afferent from a muscle spindle tracking changes in dynamic muscle length.

(B) Fiber Y is a Ib afferent from a Golgi tendon organ sensing active muscle tension.

(C) Fiber Y is a dynamic gamma motor axon providing fusimotor biasing.

(D) Fiber Y is an alpha motor neuron recording recurrent inhibition.

Q17. A 29-year-old male sustains a traumatic spinal cord injury resulting in complete hemisection of the left half of the spinal cord at the level of the T10 thoracic dermatome (Brown-Séquard syndrome). Neurophysiological examination 4 weeks post-injury will reveal which of the following precise patterns of sensory and motor deficits below the level of the lesion?

(A) Ipsilateral loss of pain/temperature sensation; contralateral loss of voluntary motor control and proprioception.

(B) Contralateral loss of pain/temperature sensation; ipsilateral loss of voluntary motor control, fine touch, and proprioception.

(C) Bilateral loss of pain and temperature sensation with total preservation of voluntary motor pathways.

(D) Ipsilateral loss of all modalities (pain, temperature, touch, motor) with zero contralateral abnormalities.

Detailed Solutions

Q1.

Solution

Concept: Antidiuretic hormone (ADH/vasopressin) regulates water reabsorption in the renal collecting duct principal cells by binding to basolateral V_2 receptors. This G-protein coupled receptor ($G_{\alpha s}$) activates adenylate cyclase, triggering intracellular signaling cascades that direct the exocytosis of **Aquaporin-2 (AQP2)** water channels to the apical membrane.

Solution:

Let's analyze the intracellular signaling mechanisms and cytoskeleton motor dynamics:

(a) Binding of ADH to the V_2 receptor drives an increase in intracellular **cyclic adenosine monophosphate (cAMP)**, which directly activates **Protein Kinase A (PKA)**.

(b) Active PKA phosphorylates specific residues (such as Serine-256) on the cytoplasmic tail of AQP2 molecules located within endosomal vesicles.

(c) To reach the apical membrane, these AQP2-bearing storage vesicles must undergo anterograde (forward) transport along organized **microtubule tracks**. This outward movement towards the cell periphery is powered exclusively by the molecular motor protein **kinesin**, making option C the correct secondary messenger and motor pairing.

Final Answer: cAMP-dependent Protein Kinase A activation leading to Kinesin-mediated anterograde transport along microtubules.

Answer: (C)

Go Back to Question 1

Q2.

Solution

Concept: Voltage-gated Na^+ channels regulate the initiation and rapid upstroke (Phase 0) of action potentials in excitable membranes. These channels possess a voltage-dependent inner activation gate (**m-gate**) and a time-dependent outer inactivation gate (**h-gate**).

Solution:

Let's evaluate the functional channel state transitions indicated by the diagram:

(a) At a normal baseline resting membrane potential of −90 mV, the channel resides in the **Resting State** (m-gate closed, h-gate open).

(b) When a threshold stimulus is applied, the change in membrane voltage forces the activation gate (m-gate) to open rapidly while the inactivation gate (h-gate) remains open momentarily, allowing Na^+ influx. This conversion from the resting state to the **Activated State** is represented by **State Transition 1**.

(c) Because Compound X-99 locks the inner activation gate (m-gate) closed, it prevents the channel from opening in response to depolarization. Consequently, the channel cannot progress from its resting configuration to the open, conducting configuration, directly blocking **State Transition 1**.

Final Answer: State Transition 1

Answer: (A)

Go Back to Question 2

Q3.

Solution

Concept: Active membrane transport processes are divided into primary and secondary modes. Primary active transport couples the breakdown of ATP directly to solute movement against a gradient, whereas secondary active transport uses the energy stored in an electrochemical gradient generated by an upstream primary pump.

Solution:

Let's analyze the kinetics and behavioral dependencies of the transport system:

(a) The total cessation of transport following the introduction of the metabolic poison dinitrophenol (DNP, which halts ATP synthesis) confirms that the accumulation of solute S is an active, energy-requiring process, ruling out passive or facilitated diffusion (options C and D).

(b) When the extracellular sodium concentration ($[Na^+]_o$) is altered to equal the intracellular sodium concentration ($[Na^+]_i$), the sodium chemical gradient ($\Delta\mu_{Na^+}$) is reduced to zero.

(c) The observation that transport of solute S ceases completely when this sodium gradient is abolished—even when ATP is artificially abundant—confirms that the carrier does not hydrolyze ATP directly. Instead, it is a **Secondary Active Co-transport (Symport)** mechanism powered by the inward potential energy of the sodium electrochemical gradient.

Final Answer: Secondary Active Co-transport (Symport) driven by an electrochemical gradient.

Answer: (B)

Go Back to Question 3

Q4.

Solution

Concept: The **Law of Laplace** defines the mechanical relationships governing wall stress (σ) in a hollow spherical or cylindrical organ, such as the left ventricle of the heart, via the formula:

$$\sigma = \frac{P \cdot r}{2h}$$

where P is the intraventricular pressure, r is the internal chamber radius, and h is the myocardial wall thickness.

Solution:

Let's evaluate the geometric and physical modifications occurring during concentric left ventricular hypertrophy:

(a) Long-standing uncontrolled systemic hypertension forces the left ventricle to pump against a chronically elevated afterload, resulting in a persistent increase in intraventricular pressure (P).

(b) According to the Laplace relationship, an increase in P directly elevates myocardial wall stress (σ), which increases myocardial oxygen demand and reduces efficiency.

(c) To minimize wall stress and normalize cardiac load, individual cardiomyocytes add sarcomeres in parallel, causing an **increase in wall thickness (h)**. By thickening the muscular wall, the denominator of the equation increases, effectively neutralizing the elevated pressure (P) and acting to stabilize σ during early-stage compensation.

Final Answer: An increase in h acts to normalize σ in the setting of elevated P.

Answer: (B)

Go Back to Question 4

Q5.

Solution

Concept: The ventricular action potential plateau phase (**Phase 2**) is maintained by a delicate balance between inward depolarizing currents, principally via L-type calcium channels ($I_{Ca,L}$), and outward repolarizing currents, mediated by delayed rectifier potassium channels (I_{Kr} and I_{Ks}).

Solution:

Let's evaluate how ion channel modifications prolong the plateau phase:

(a) Class III antiarrhythmic agents target the repolarization process. To selectively prolong Phase 2 of the action potential without modifying the resting membrane potential (which is maintained by inward rectifying I_{K1} channels during Phase 4), an agent must delay the net rate of repolarization or prolong inward currents during the plateau.

(b) **Inhibition of the delayed rectifier potassium channels** (I_{Kr} or I_{Ks}) slows the efflux of potassium ions during the plateau, extending its duration.

(c) Alternatively, **prolonging the opening of L-type calcium channels** ($I_{Ca,L}$) provides a sustained inward positive charge that opposes repolarization. Either mechanism shifts the balance to lengthen Phase 2, which manifests on a surface electrocardiogram as a prolongation of the QT interval.

Final Answer: Prolongation of L-type Ca^{2+} channel ($I_{Ca,L}$) opening and/or inhibition of delayed rectifier K^+ channels (I_{Kr}/I_{Ks}).

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: A systemic vascular function curve graphs the relationship between right atrial pressure and venous return. The point where the venous return curve intercepts the horizontal pressure axis (where flow drops to zero) represents the **Mean Systemic Filling Pressure (MSFP)**, which reflects the total volume of blood within the systemic vasculature relative to its capacitance.

Solution:

Let's analyze the directional shift of the vascular function curve profile:

(a) The baseline solid curve has an initial mean systemic filling pressure labeled as $MSFP_1$ (at 4 mmHg). Following the clinical intervention, the dashed curve exhibits a parallel shift downward and to the left, establishing a lower mean systemic filling pressure at $MSFP_2$ (at 2.5 mmHg).

(b) A decrease in MSFP is fundamentally caused by either a reduction in total blood volume (e.g., hemorrhage) or an increase in venous vascular capacitance (venodilator therapy).

(c) Administering a **potent venodilator** like nitroglycerin increases the compliance of the systemic venous capacitance vessels (the primary blood reservoir). This traps blood in the venous periphery, reducing the stressed volume and the MSFP, which drives a leftward parallel shift of the venous return profile.

Final Answer: Administration of a potent venodilator (e.g., Nitroglycerin).

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept: Mean arterial pressure (MAP) is dictated by Ohm's law applied to the cardiovascular system, expressed as:

$$MAP = CO \times TPR$$

where CO is the cardiac output and TPR is the total peripheral resistance.

Solution:

Let's analyze the hemodynamic balancing mechanisms occurring during strenuous exercise:

(a) During heavy physical exercise, cardiac output (CO) increases significantly (up to 4- to 5-fold in healthy individuals) driven by synchronous elevations in heart rate and stroke volume.

(b) If TPR remained unchanged, this massive surge in CO would cause a life-threatening elevation in blood pressure. However, exercising skeletal muscles accumulate local metabolic vasodilators (such as adenosine, K^+, H^+, lactate, and CO_2).

(c) This local accumulation causes extensive arteriolar smooth muscle relaxation and **marked metabolic vasodilation within the active skeletal muscle beds**. Because the skeletal muscle vascular bed is large, this local vasodilation drives a profound decrease in overall TPR, balancing the rise in CO and maintaining a stable blood pressure profile.

Final Answer: Marked metabolic vasodilation in active skeletal muscles causing a profound drop in total peripheral resistance (TPR).

Answer: (B)

Go Back to Question 7

Q8.

Solution

Concept: The respiratory system utilizes distinct chemical monitoring zones to manage blood gas homeostasis. Central chemoreceptors, located on the ventrolateral surface of the medulla, respond exclusively to changes in hydrogen ion (H^+) concentration within the cerebrospinal fluid (CSF), which is driven by dissolved arterial PCO_2. Peripheral chemoreceptors, located in the carotid and aortic bodies, respond to changes in arterial PO_2, PCO_2, and pH.

Solution:

Let's evaluate the sensory mechanisms activated during acute high-altitude exposure:

(a) Rapid ascent to 5,000 meters exposes an unacclimatized individual to a lower atmospheric pressure, decreasing inspired oxygen and causing severe arterial hypoxemia.

(b) Central chemoreceptors cannot sense changes in oxygen tension directly. Furthermore, hyperventilation causes hypocapnia, which increases CSF pH and acts to inhibit the central respiratory drive.

(c) Instead, the acute hyperventilatory response is triggered by **glomus cells within the carotid bodies**. These peripheral chemoreceptors are sensitive to dissolved arterial oxygen and increase their afferent firing rate when arterial **PO_2 drops below 60 mmHg**, overriding the inhibitory central signaling to drive ventilation.

Final Answer: Glomus cells of the carotid bodies sensing a reduction in dissolved arterial $\text{PO}_2 below 60 mmHg$.

Answer: (B)

Go Back to Question 8

Q9.

Solution

Concept: Lung compliance represents the change in lung volume per unit change in transmural pressure ($\Delta V/\Delta P$). On a static compliance pressure-volume loop, restrictive parenchymal disorders reduce compliance due to structural scarring, whereas obstructive disorders (like emphysema) increase compliance due to loss of elastic recoil.

Solution:

Let's analyze the compliance curves shown in the diagram:

(a) Curve B represents normal lung compliance and tissue architecture.

(b) Curve A exhibits a steeper slope, shifting upward and to the left. This represents an increase in lung compliance, characteristic of destructive diseases like emphysema.

(c) **Curve C** features a flattened slope, shifting downward and to the right. This represents a significant reduction in lung compliance. Because **idiopathic pulmonary fibrosis (IPF)** causes extensive deposition of rigid collagen matrices in the alveolar interstitium, the lungs become stiff and resistant to expansion, matching the low-compliance profile of Curve C.

Final Answer: Curve C

Answer: (C)

Go Back to Question 9

Q10.

Solution

Concept: Alveolar ventilation ($\dot{V}_A$) represents the actual volume of fresh atmospheric gas entering the respiratory zones per minute. It is calculated by subtracting the dead space volume (V_D) from the total tidal volume (V_T), and multiplying by the respiratory rate (f):

$$\dot{V}_A = (V_T - V_D) \times f$$

Solution:
Let's calculate and compare the initial and modified alveolar ventilation volumes:

(a) **Initial Settings:** Tidal volume $V_{T1} = 450$ mL, dead space $V_D = 150$ mL, and rate $f_1 = 12$ breaths/min.

$$\dot{V}_{A1} = (450 \text{ mL} - 150 \text{ mL}) \times 12 = 300 \text{ mL} \times 12 = 3600 \text{ mL/min}$$

(b) **Modified Settings:** Tidal volume is decreased to $V_{T2} = 300$ mL, dead space remains $V_D = 150$ mL, and rate is increased to $f_2 = 18$ breaths/min.

$$\dot{V}_{A2} = (300 \text{ mL} - 150 \text{ mL}) \times 18 = 150 \text{ mL} \times 18 = 2700 \text{ mL/min}$$

(c) This comparison shows that shifting to a smaller tidal volume—even with a compensatory increase in respiratory rate—causes a larger proportion of each breath to be wasted in dead space, decreasing net alveolar ventilation from 3600 mL/min to 2700 mL/min.

Final Answer: Alveolar ventilation decreases from 3600 mL/min to 2700 mL/min.

Answer: (B)

Go Back to Question 10

Q11.

Solution

Concept: In the **thick ascending limb** of Henle's loop, active reabsorption of sodium, potassium, and chloride ions by the NKCC2 cotransporter is accompanied by back-leakage of potassium ions into the tubular lumen via ROMK channels. This selective back-leak generates a positive transepithelial electrical gradient ($\approx +10$ mV) in the lumen relative to the interstitium.

Solution:

Let's trace how the loss of this electrical gradient alters divalent cation handling:

(a) This lumen-positive potential difference provides the primary electrical driving force that repels positively charged ions, driving their reabsorption through low-resistance paracellular pathways.

(b) The principal ions dependent on this paracellular, charge-driven reabsorption mechanism are the divalent cations **calcium (Ca^{2+})** and **magnesium (Mg^{2+})**.

(c) When a loop diuretic blocks NKCC2 and abolishes the lumen-positive potential difference, this electrical driving force is lost. As a result, the paracellular reabsorption of these cations ceases, leading to an increase in the renal clearance of Ca^{2+} and Mg^{2+}.

Final Answer: $\boxed{\mathrm{Ca}^{2+} and Mg^{2+} \mathrm{Ca}^{2+} and Mg^{2+} \mathrm{Ca}^{2+} and Mg^{2+} \mathrm{Ca}^{2+} and Mg^{2+}}$

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept: **Tubuloglomerular feedback (TGF)** is an autoregulatory mechanism matching renal perfusion with nephron filtration rates. It is mediated by the specialized **macula densa** cells located in the thick ascending limb adjacent to the afferent and efferent arterioles.

Solution:

Let's trace the intracellular cascade activated by increased tubular solute delivery:

(a) When the glomerular filtration rate (GFR) or fluid delivery increases, an elevated load of Na^+, K^+, and Cl^- enters the macula densa cells via the apical **NKCC2 cotransporter**.

(b) The rapid entry of these ions increases intracellular sodium concentrations, forcing the basolateral Na^+/K^+ ATPase pump to work at an accelerated rate. This rapid pump activity causes high **ATP consumption**, generating adenosine breakdown products.

(c) **Adenosine** is subsequently released across the basolateral membrane into the juxtaglomerular interstitium. It binds to local A_1 adenosine receptors on nearby vascular smooth muscle cells, triggering an influx of calcium that induces constriction of the adjacent afferent arteriole to normalize GFR.

Final Answer: Increased Na^+-K^+-$2Cl^-$ entry → ATP consumption → intracellular adenosine generation and release via basolateral membranes.

Answer: (C)

Go Back to Question 12

Q13.

Solution

Concept: Evaluating a complex acid-base disturbance requires calculating the **serum anion gap (AG)** to differentiate between types of metabolic acidosis. The formula used is:

$$AG = [Na^+] - ([Cl^-] + [HCO_3^-])$$

Solution:
Let's analyze the blood gas metrics and calculate the anion gap value:

(a) The patient has an arterial pH of 7.22, confirming a primary metabolic acidosis ($[HCO_3^-] = 12$ mEq/L). The PCO_2 of 30 mmHg reflects a normal compensatory respiratory alkalosis.

(b) Substituting the electrolyte values into the formula:

$$AG = 140 - (118 + 12) = 140 - 130 = 10 \text{ mEq/L}$$

(c) A value of 10 mEq/L falls entirely within the normal reference range (8 to 12 mEq/L), establishing a diagnosis of **normal anion gap metabolic acidosis** (hyperchloremic metabolic acidosis).

(d) This condition is caused by a direct loss of bicarbonate ions, rather than the accumulation of unmeasured organic acids (ruling out DKA). Severe **chronic diarrhea** causes significant bicarbonate loss in stool, which is compensated for by renal retention of chloride ions ($Cl^- = 118$ mEq/L), matching this laboratory profile.

Final Answer: Severe chronic diarrhea resulting in substantial gastrointestinal base bicarbonate loss.

Answer: (B)

Go Back to Question 13

Q14.

Solution

Concept: **Sheehan syndrome** (postpartum pituitary necrosis) results from severe systemic hypotension or hemorrhage during childbirth. During pregnancy, the anterior pituitary gland hypertrophies due to increased prolactin demand, making it highly susceptible to ischemic infarction if systemic perfusion drops sharply.

Solution:

Let's identify the functional component corresponding to Node X:

(a) The diagram traces the hypothalamic-pituitary-adrenal axis, where the hypothalamus releases corticotropin-releasing hormone (CRH) to stimulate **Node X**, which then releases adrenocorticotropic hormone (ACTH) to drive cortisol secretion from the adrenal cortex. This identifies **Node X** as the **anterior pituitary gland (adenohypophysis)**.

(b) The patient's presentation—lethargy (loss of TSH), cold intolerance, and failure of lactation (loss of prolactin) following a severe postpartum hemorrhage—is pathognomonic for Sheehan syndrome.

(c) This ischemic event causes necrosis of the **anterior pituitary (Node X)**, rendering it nonfunctional and breaking the downstream communication loop.

Final Answer: Node X (Anterior Pituitary)

Answer: (B)

Go Back to Question 14

Q15.

Solution

Concept: Insulin secretion from pancreatic beta cells is tightly coupled to cellular glucose metabolism. Glucose enters the cell via GLUT transporters and is immediately phosphorylated by the rate-limiting enzyme **glucokinase**, which acts as the cell's metabolic glucose sensor.

Solution:

Let's analyze the downstream effects of blocking glucokinase:

(a) Phosphorylation of glucose by glucokinase initiates glycolysis and mitochondrial respiration, which increases the intracellular **ATP/ADP ratio**.

(b) A high ATP/ADP ratio binds to and closes the **ATP-sensitive K^+ (K_{ATP}) channels** in the plasma membrane.

(c) Closure of these channels prevents potassium efflux, inducing cell depolarization. This depolarization activates voltage-gated Ca^{2+} channels, triggering the exocytosis of insulin-containing vesicles.

(d) If an experimental toxin blocks glucokinase, glucose cannot be metabolized, and the ATP/ADP ratio fails to rise. Consequently, the **K_{ATP} channels remain open**, preventing membrane depolarization and completely blocking insulin secretion.

Final Answer: K_{ATP} channels remain open, preventing cell depolarization and completely blocking insulin secretion.

Answer: (A)

Go Back to Question 15

Q16.

Solution

Concept: Skeletal muscle contains two types of proprioceptive mechanoreceptors that monitor mechanical state: muscle spindles (arranged in parallel with extrafusal fibers) and Golgi tendon organs (arranged in series at the musculo-tendinous junctions).

Solution:

Let's analyze the firing responses of the fibers under passive and active conditions:

(a) **Passive Stretch:** Both the muscle spindle and the tendon are elongated. The muscle spindle fires rapidly to track changes in length (Fiber X), while the Golgi tendon organ experiences lower tension and fires minimally (Fiber Y).

(b) **Active Isometric Contraction:** Extrafusal muscle fibers contract and shorten. This shortens the parallel muscle spindle, causing it to go slack and silencing its sensory afferent (**Type Ia / Fiber X**).

(c) However, active muscle contraction puts significant mechanical tension on the musculo-tendinous junction. This structural stress stimulates the in-series **Golgi tendon organ**, causing its afferent (**Type Ib / Fiber Y**) to fire at its maximum rate. This confirms that Fiber Y is a Ib afferent sensing changes in active muscle tension.

Final Answer: Fiber Y is a Ib afferent from a Golgi tendon organ sensing changes in active muscle tension.

Answer: (B)

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Q17.

Solution

Concept: A complete hemisection of the left side of the spinal cord at the T10 level (**Brown-Séquard syndrome**) disrupts three primary neural pathways: the descending corticospinal tract, the ascending dorsal column-medial lemniscus system, and the ascending spinothalamic tract.

Solution:

Let's trace the sensory and motor deficits based on where these tracts decussate:

(a) **Corticospinal Tract Dorsal Columns:** Both pathways carry information that decussates superiorly in the brainstem or medulla. Therefore, a lesion on the left side of the spinal cord disrupts these pathways before they cross, causing **ipsilateral (left-sided) loss of voluntary motor control, fine touch, and proprioception** below T10.

(b) **Spinothalamic Tract:** Conveys pain and temperature sensations. These fibers decussate within the spinal cord (1 to 2 segments above entry) and ascend on the opposite side.

(c) A left-sided lesion cuts the crossed fibers originating from the right side of the body, causing a **contralateral (right-sided) loss of pain and temperature sensation** below the level of the injury, matching option B.

Final Answer: Contralateral loss of pain/temperature sensation; ipsilateral loss of voluntary motor control, fine touch, and proprioception.

Answer: (B)

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Answer Key

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