NEET PG Physiology Sample Paper-10

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 male athlete participates in an endurance time trial under extreme heat. His plasma membrane ion channel dynamics are studied via patch-clamp analysis. A novel mutation in his skeletal muscle voltage-gated sodium channels alters the inactivation gate kinetics, prolonging the absolute refractory period. Which of the following parameters represents the mathematical equivalent of the cell membrane acting as a simple RC electrical circuit to determine the rate of passive membrane potential decay?

(A) $\tau = R_m \cdot C_m$

(B) $\lambda = \sqrt{\frac{R_m}{R_i}}$

(C) $I = \frac{V}{R}$

(D) $E_m = \frac{RT}{F} \ln \frac{P_k[K^+]_o}{P_k[K^+]_i}$

Q2. An experimental drug blocks the active transport of primary solutes by binding directly to the intracellular phosphorylation domain of P-type ATPases. Assuming this drug selectively paralyzes the Na^+—K^+ ATPase pump across systemic vascular endothelial cells, what is the immediate, direct consequence on the intracellular concentrations of sodium ($[Na^+]_i$), potassium ($[K^+]_i$), and the overall cell volume?

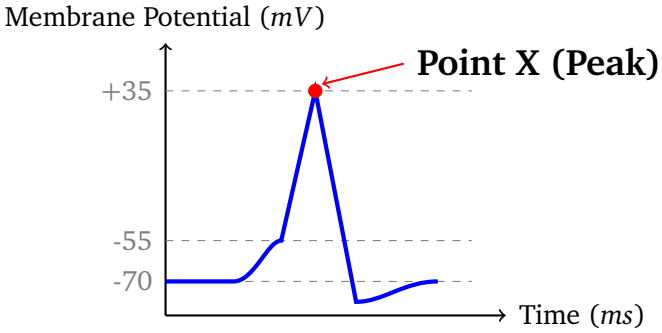
(A) Increased $[Na^+]_i$, Decreased $[K^+]_i$, Cell Swelling

(B) Decreased $[Na^+]_i$, Increased $[K^+]_i$, Cell Shrinkage

(C) Increased $[Na^+]_i$, Increased $[K^+]_i$, Cell Swelling

(D) Decreased $[Na^+]_i$, Decreased $[K^+]_i$, No Change

Q3. A cell physiologist uses an optical biosensor to track the thermodynamic potential shifts across a synthetic lipid bilayer. Analyze the standard action potential waveform generated in the experimental model below. Identify the exact phase indicated by the specific point marked **X** where net inward positive current completely equilibrates with net outward positive current before reversing polarity:



(A) Resting Membrane Potential

(B) Threshold Potential

(C) Overshoot Peak

(D) Hyperpolarizing Afterpotential

Q4. During an invasive cardiac catheterization procedure on a 62-year-old female with calcific aortic stenosis, a continuous real-time left ventricular pressure-volume loop is displayed on the surgical monitor. If the patient's myocardial contractility is abruptly enhanced via an acute intravenous infusion of a selective β_1-adrenergic agonist, how will the end-systolic pressure-volume relationship (ESPVR) slope and the stroke volume (SV) change on the loop?

(A) ESPVR slope decreases; SV decreases

(B) ESPVR slope increases; SV increases

(C) ESPVR slope remains flat; SV increases

(D) ESPVR slope shifts right; SV remains static

Q5. A 54-year-old male with a history of chronic untreated hypertension presents with signs of left ventricular hypertrophy. Microvascular analysis reveals a dramatic decrease in compliance of the systemic large arteries. Which of the following physiologic consequences perfectly describes the direct effect of this decreased arterial compliance on the central aortic pulse pressure and the velocity of the pressure transmission wave?

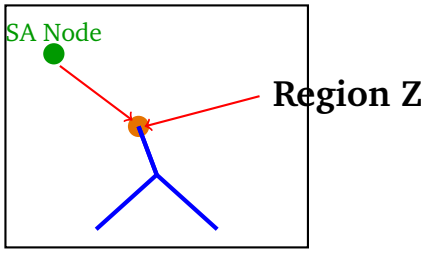
(A) Decreased pulse pressure, decreased pulse wave velocity

(B) Increased pulse pressure, increased pulse wave velocity

(C) Increased pulse pressure, decreased pulse wave velocity

(D) Decreased pulse pressure, increased pulse wave velocity

Q6. A clinical research fellow reviews a high-resolution electrical tracing tracking conduction intervals through specialized myocardial tissue nodes. Observe the specific vector progression pattern illustrated in the cardiac conduction schematic below. Identify the region marked **Z** which represents the slowest conduction velocity zone responsible for the physiological delay essential for adequate ventricular filling:



Conduction Vector Pathway

(A) Sinoatrial (SA) Node

(B) Atrioventricular (AV) Node

(C) Right Bundle Branch

(D) Purkinje Fibers

Q7. An experimental animal model is prepared where the bilateral carotid sinuses and aortic arch baroreceptors are surgically denervated. Following complete recovery, the animal's mean arterial pressure (MAP) is recorded continuously over a 48-hour period. Compared to a healthy control animal, what characteristic feature will be observed in the MAP recordings of the denervated animal?

(A) Permanent profound hypotension with zero variability

(B) Markedly increased variability in MAP without a major change in the true 24-hour average MAP

(C) Sustained, fixed malignant hypertension with zero variability

(D) Permanent isolated diastolic hypotension with preserved systolic peaks

Q8. A 28-year-old female climber ascends rapidly to an altitude of 4,500 meters without supplemental oxygen. Within hours, her central and peripheral chemoreceptors trigger marked hyperventilation. As a direct result of this respiratory adaptation, what are the anticipated primary shifts in her arterial blood gas parameters regarding pH, PCO_2, and plasma bicarbonate concentration ($[HCO_3^-]$) before renal compensation begins?

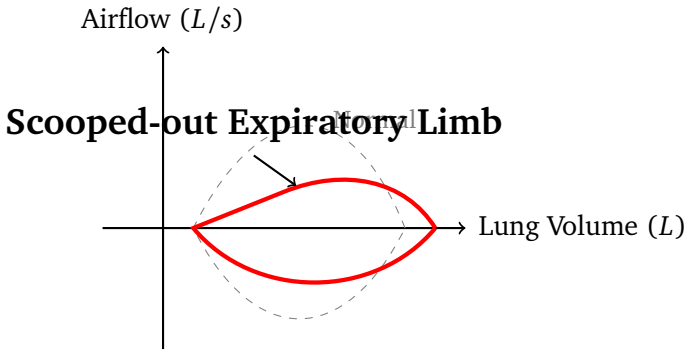
(A) Decreased pH, Increased PCO_2, Increased $[HCO_3^-]$

(B) Increased pH, Decreased PCO_2, Normal to minimally decreased $[HCO_3^-]$

(C) Increased pH, Increased PCO_2, Decreased $[HCO_3^-]$

(D) Decreased pH, Decreased PCO_2, Increased $[HCO_3^-]$

Q9. A pulmonary physiology laboratory evaluates a patient complaining of exertional dyspnea using dynamic spirometry graphs. Examine the dynamic loop shown in the diagram below. Based on the configuration of the expiratory limb plateau and the inspiratory limb morphology, identify the specific category of mechanical ventilatory impairment illustrated:



(A) Pure Restrictive Lung Disease

(B) Obstructive Lung Disease

(C) Fixed Upper Airway Obstruction

(D) Variable Extrathoracic Obstruction

Q10. A 33-year-old male is treated in the ICU for acute respiratory distress syndrome (ARDS). Due to widespread alveolar collapse and inflammation, his functional residual capacity (FRC) is severely compromised. In terms of respiratory mechanics, a decrease in FRC typically shifts the dynamic operating point of the lung compliance curve to which specific direction, and how does it alter the total work of breathing?

(A) Shifts to a flatter, non-compliant portion; increases work of breathing

(B) Shifts to a steeper, highly compliant portion; decreases work of breathing

(C) Shifts to a flatter portion; decreases work of breathing

(D) No shift occurs; work of breathing increases solely due to airway resistance

Q11. An experimental drug molecule selectively constricts the efferent arteriole of the renal cortical nephrons while leaving the afferent arteriole completely unaffected. Assuming renal auto-regulatory mechanisms are experimentally clamped and unable to intervene, what simultaneous changes will occur in the Glomerular Filtration Rate (GFR) and the Renal Plasma Flow (RPF)?

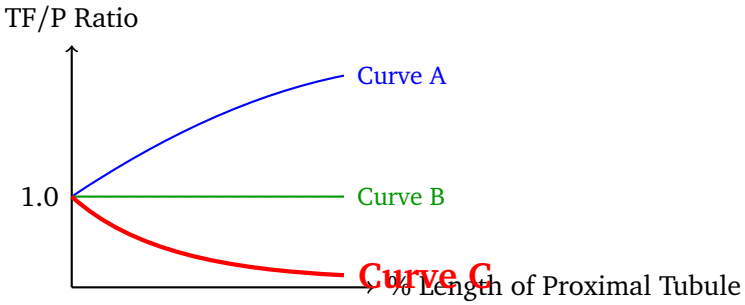
(A) GFR decreases; RPF increases

(B) GFR increases; RPF decreases

(C) GFR increases; RPF increases

(D) GFR decreases; RPF decreases

Q12. A clinical scientist maps the tubular fluid-to-plasma concentration ratio (TF/P) of various solutes along the different anatomic zones of a functional nephron segment. Look closely at the graphical curves plotted below. Identify the specific curve that correctly represents the transport profile of **bicarbonate ions (HCO_3^-)** along the course of the Proximal Convoluted Tubule (PCT):



(A) Curve A

(B) Curve B

(C) Curve C

(D) None of the curves

Q13. A 72-year-old woman with chronic kidney disease presents with profound metabolic acidosis. Her renal clearance parameters are evaluated. Which physiological mechanism serves as the definitive, adaptive renal response to synthesize and add *new* bicarbonate (HCO_3^-) ions back into the systemic circulation to combat chronic metabolic acidosis?

(A) Increased filtration fraction at the glomerulus

(B) Down-regulation of apical Na-H exchangers (NHE3)

(C) Glutamine metabolism and ammonium (NH_4^+) excretion in the proximal tubule

(D) Decreased H-ATPase pump activity in Type A intercalated cells

Q14. A patient presents with a rare ACTH-secreting pituitary microadenoma (Cushing's Disease). Chronically elevated circulating cortisol levels exert profound metabolic actions. Which of the following pathways perfectly highlights the direct cellular action of excess cortisol on protein and carbohydrate metabolism in peripheral skeletal muscle versus hepatic tissue?

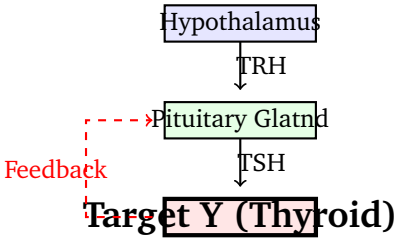
(A) Muscle: Stimulates protein synthesis; Liver: Inhibits gluconeogenesis

(B) Muscle: Stimulates glucose uptake; Liver: Stimulates glycogenolysis only

(C) Muscle: Promotes protein catabolism; Liver: Stimulates gluconeogenesis

(D) Muscle: Inhibits protein breakdown; Liver: Decreases glucose output

Q15. A molecular endocrinology panel visualizes the feedback loop kinetics controlling hormone secretion in thyroid disorders. Examine the classic multi-tier axis schematic below. If a patient possesses circulating autoantibodies that act as long-acting thyroid stimulators by directly binding to and activating the receptor labeled **Target Y**, what will be the resulting status of hormone secretion from the pituitary gland?



(A) Elevated TSH secretion via positive feedback

(B) Suppressed TSH secretion via negative feedback

(C) Normal TSH secretion due to receptor bypassing

(D) Isolated elevation of TRH with suppressed TSH

Q16. A neurophysiology study investigates the gating properties of sensory pathways. A mechanical noxious stimulus applied to the right lower limb triggers a rapid reflex response. Concurrently, high-frequency electrical stimulation of larger, myelinated tactile A-beta (Aβ) afferent fibers from the same cutaneous region significantly attenuates the transmission of pain signals through

the spinothalamic tract. This clinical scenario represents the physiological basis of which classic neurophysiological concept?

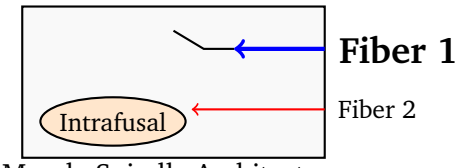
(A) Müller's Doctrine of Specific Nerve Energies

(B) Gate Control Theory of Pain

(C) Weber-Fechner Law of Sensation

(D) Inverse Myotatic Reflex Breakdown

Q17. A neuro-rehabilitation unit evaluates a patient recovering from a cerebrovascular accident affecting the motor cortex. The specialized clinician tests stretch reflex feedback loops by monitoring muscle spindle afferent tracking. Observe the structural functional arrangement of the muscle spindle loop illustrated below. Identify the specific nerve fiber marked **Alpha (α) Motor Neuron** responsible for extrafusal muscle fiber contraction:



Muscle Spindle Architecture

(A) Fiber 1 (Innervates Extrafusal Fibers)

(B) Fiber 2 (Innervates Intrafusal Ends)

(C) Both Fiber 1 and Fiber 2 combined

(D) Neither Fiber

Detailed Solutions

Q1.

Solution

Concept: A biological cell membrane behaves electrically like a parallel RC circuit, where the lipid bilayer acts as a capacitor (C_m) that stores charge, and the embedded ion channels act as a resistor (R_m) that resists ionic current flow. The passive decay of membrane potential over time following a stimulus is determined by the membrane time constant (τ).

Solution:

Let's analyze the mathematical properties of passive membrane potential electrical circuits:

(a) The membrane time constant (τ) is defined as the product of the membrane resistance and the membrane capacitance:

$$\tau = R_m \cdot C_m$$

(b) Physically, τ represents the time required for the membrane potential to fall to approximately 37% ($1/e$) of its initial value during passive decay, or to rise to 63% ($1-1/e$) of its maximum value during passive charging.

(c) The alternative parameter $\lambda = \sqrt{R_m/R_i}$ represents the length constant, which dictates how a passive potential decays over *distance* along an axon or muscle fiber rather than over time.

Final Answer: $\boxed{\tau = R_m \cdot C_m}$

Answer: (A)

Go Back to Question 1

Q2.

Solution

Concept: The primary active Na^+-K^+ ATPase pump utilizes the energy derived from ATP hydrolysis to transport $3\,Na^+$ ions out of the cell in exchange for $2\,K^+$ ions moving into the cell. This electrogenic cycle maintains the chemical concentration gradients and osmotic balance across mammalian cell membranes.

Solution:

Let's analyze the direct physiological consequences of paralyzing the Na^+-K^+ ATPase pump:

(a) When the pump is selectively blocked, the primary efflux pathway for sodium stops. Passive leak pathways continue to allow sodium to enter the cell down its electrochemical gradient, leading to an **increased intracellular sodium concentration ($[Na^+]_i$)**.

(b) Conversely, potassium can no longer be actively pumped into the cell. It continuously leaks out through background potassium channels down its chemical gradient, causing a **decreased intracellular potassium concentration ($[K^+]_i$)**.

(c) Because the pump exports 3 positive charges for every 2 it imports, its blockade leads to a net accumulation of intracellular solutes. This accumulation increases intracellular osmolarity, drawing water into the cell down an osmotic gradient and causing **cell swelling**.

Final Answer: $\boxed{\text{Increased } [Na^+]_i,\ \text{Decreased } [K^+]_i,\ \text{Cell Swelling}}$

Answer: (A)

Go Back to Question 2

Q3.

Solution

Concept: An action potential waveform represents sequential variations in ion permeability across a cell membrane. The peak or overshoot phase occurs at the transition point where the rapid depolarizing phase ends and the repolarizing phase begins.

Solution:

Let's look at the electrical and kinetic parameters at the point marked **X**:

(a) During the Phase 0 upstroke, the activation of voltage-gated sodium channels causes a massive influx of positive charges (I_{Na}), driving the membrane potential past 0 mV toward the sodium equilibrium potential.

(b) At **Point X (the overshoot peak)**, the membrane potential reaches its maximum positive voltage (approximately +35 mV). At this exact instant, the net inward depolarizing sodium current slows down due to channel inactivation and perfectly matches the rising outward repolarizing potassium current (I_K).

(c) Because the net ionic current ($I_{net} = I_{in} - I_{out}$) equals zero at this apex, the rate of voltage change (dV/dt) momentarily drops to zero before the polarity reverses and repolarization begins.

Final Answer: Overshoot Peak

Answer: (C)

Go Back to Question 3

Q4.

Solution

Concept: In a left ventricular pressure-volume (PV) loop, the slope of the End-Systolic Pressure-Volume Relationship (ESPVR) serves as a reliable, load-independent index of myocardial contractility (inotropy).

Solution:

Let's trace the mechanical changes that occur when contractility is enhanced by a β_1-adrenergic agonist:

(a) Infusing a β_1-agonist increases intracellular calcium availability and accelerates cross-bridge cycling within cardiac myocytes, **increasing the slope of the ESPVR** and shifting it to the left.

(b) A steeper ESPVR slope means the ventricle can contract to a smaller End-Systolic Volume (ESV) at any given systolic pressure.

(c) Because the ventricle empties more completely while End-Diastolic Volume (EDV) remains stable or increases slightly, the total width of the PV loop expands, resulting in an **increased stroke volume (SV)** ($SV = EDV - ESV$).

Final Answer: ESPVR slope increases; SV increases

Answer: (B)

Go Back to Question 4

Q5.

Solution

Concept: Large elastic arteries (such as the aorta) store a portion of the stroke volume ejected during systole and release that stored energy during diastole via elastic recoil. A reduction in compliance ($C = \Delta V / \Delta P$) makes these vessel walls more rigid.

Solution:

Let's break down the hemodynamic consequences of decreased compliance in the large central arteries:

(a) **Pulse Pressure:** During ventricular ejection, a rigid, non-compliant aorta cannot expand to accommodate the stroke volume, causing a sharp rise in systolic pressure. During diastole, the lack of elastic recoil causes pressure to drop rapidly. This combination of an elevated systolic and a reduced diastolic baseline significantly **increases central arterial pulse pressure**.

(b) **Pulse Wave Velocity (PWV):** The speed at which the pressure wave travels down the arterial tree is inversely proportional to vascular compliance, as described by the Moens-Korteweg equation:

$$\text{PWV} = \sqrt{\frac{E \cdot h}{2 \cdot R \cdot \rho}}$$

As compliance falls and arterial stiffness (E) rises, the resistance to vessel wall expansion increases, which **increases the pulse wave velocity**.

Final Answer: Increased pulse pressure, increased pulse wave velocity

Answer: (B)

Go Back to Question 5

Q6.

Solution

Concept: The electrical impulse that coordinates cardiac contraction travels through specialized pathways at varying speeds. A localized delay in conduction is essential to allow the atria to contract fully and empty their blood into the ventricles before ventricular contraction begins.

Solution:

Let's evaluate the conduction velocity across the different regions of the heart:

(a) The impulse originates in the SA node and travels across the atrial myocardium to the **Atrioventricular (AV) Node (Region Z)**.

(b) The AV node possesses the **slowest conduction velocity** in the entire cardiac electrical system (approximately 0.02 to 0.05 m/s). This slow conduction is caused by the small diameter of the nodal cells and a lower density of gap junctions between them.

(c) This slow conduction produces the normal physiological delay seen as the PR interval on an ECG. Once past the AV node, the impulse enters the bundle branches and Purkinje fibers, where conduction velocities increase significantly (up to 4.0 m/s) to ensure rapid, coordinated ventricular contraction.

Final Answer: Atrioventricular (AV) Node

Answer: (B)

Go Back to Question 6

Q7.

Solution

Concept: The arterial baroreceptor reflex is a short-term negative feedback system that stabilizes systemic blood pressure against acute changes caused by posture, exercise, or stress.

Solution:

Let's analyze the long-term hemodynamic adjustments that occur following complete baroreceptor denervation:

(a) Afferent signals from the carotid sinuses and aortic arch normally inhibit central sympathetic outflow. Denervating these receptors removes this inhibitory brake, causing an initial surge in sympathetic activity and acute hypertension.

(b) Over a 48-hour period, renal and secondary fluid-volume mechanisms adjust to this loss of neural input, bringing the **true 24-hour average Mean Arterial Pressure (MAP) back close to normal lines**.

(c) However, because the short-term stabilizing feedback loop is gone, the animal can no longer counter daily disturbances. This results in **markedly increased variability in MAP**, characterized by extreme blood pressure spikes during movement or excitement and deep drops during rest.

Final Answer: Markedly increased variability in MAP without a major change in the true 24-hour average MAP

Answer: (B)

Go Back to Question 7

Q8.

Solution

Concept: Ascending to high altitude decreases ambient barometric pressure, reducing the partial pressure of inspired oxygen (P_IO_2). The resulting hypoxia is sensed by peripheral chemoreceptors, which drive a compensatory increase in ventilation.

Solution:

Let's trace the acute changes in arterial blood gas parameters caused by hyperventilation:

(a) Hyperventilation clears carbon dioxide (CO_2) from the alveoli faster than metabolic processes can produce it, causing an acute drop in arterial carbon dioxide tension (**decreased P_aCO_2**).

(b) According to the Henderson-Hasselbalch relationship, a reduction in dissolved CO_2 moves the carbonic acid equilibrium equation to the left, reducing hydrogen ion concentration and **increasing arterial pH** (acute respiratory alkalosis).

(c) Because renal compensation (the excretion of excess bicarbonate ions) takes 24 to 72 hours to develop, the **plasma bicarbonate concentration ($[HCO_3^-]$) remains normal to minimally decreased** during the initial hours of this respiratory shift.

Final Answer: Increased pH, Decreased PCO_2, Normal to minimally decreased $[HCO_3^-]$

Answer: (B)

Go Back to Question 8

Q9.

Solution

Concept: A flow-volume loop plots respiratory airflow against lung volume during maximum forced expiration and inspiration. The shape of these curves reflects the mechanical properties and resistance profiles of the respiratory system.

Solution:

Let's analyze the structural features of the provided flow-volume loop:

(a) The solid loop shows a distinct change during the forced expiratory phase, where airflow drops rapidly after reaching peak expiratory flow. This produces a characteristic **"scooped-out" appearance**.

(b) This "scooping" is a classic diagnostic sign of **Obstructive Lung Disease** (such as asthma or COPD). It is caused by dynamic airway compression and increased airway resistance, which limit airflow at lower lung volumes.

(c) In contrast, restrictive lung diseases produce a smaller, symmetrically narrowed loop with preserved flow rates relative to volume, while fixed upper airway obstructions flatten both the inspiratory and expiratory curves.

Final Answer: Obstructive Lung Disease

Answer: (B)

Go Back to Question 9

Q10.

Solution

Concept: The compliance curve of the lung is non-linear and S-shaped. At very low or very high lung volumes, compliance drops, meaning greater changes in transpulmonary pressure are required to produce a change in volume.

Solution:

Let's analyze how a drop in Functional Residual Capacity (FRC) alters lung mechanics in ARDS:

(a) In ARDS, widespread alveolar collapse (atelectasis) and inflammation significantly reduce the volume of gas remaining in the lungs at the end of a normal expiration, lowering the FRC.

(b) This loss of volume **shifts the dynamic operating point downward onto the lower, flatter portion of the compliance curve**.

(c) On this non-compliant segment, the stiffness of the lung tissue is increased. The respiratory muscles must work harder to reopen collapsed alveoli during inspiration, significantly **increasing the total work of breathing**.

Final Answer: Shifts to a flatter, non-compliant portion; increases work of breathing

Answer: (A)

Go Back to Question 10

Q11.

Solution

Concept: Glomerular filtration rate (GFR) and renal plasma flow (RPF) are regulated by the relative resistances of the afferent and efferent arterioles. Constricting these vessels alters the hydrostatic pressure profile within the glomerular capillaries (P_G).

Solution:

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

(a) Constricting the efferent arteriole creates a constriction downstream of the glomerulus. This restricts blood outflow from the capillary bed, which backs up blood and **increases glomerular capillary hydrostatic pressure (P_G)**, driving an **increase in GFR**.

(b) This constriction increases total renal vascular resistance, which reduces the total volume of blood flowing through the kidney, resulting in a **decrease in Renal Plasma Flow (RPF)**.

(c) This combination of increased GFR and decreased RPF elevates the overall filtration fraction (FF = GFR/RPF).

Final Answer: GFR increases; RPF decreases

Answer: (B)

Go Back to Question 11

Q12.

Solution

Concept: The tubular fluid-to-plasma ratio (TF/P) measures how the concentration of a solute changes along the nephron relative to its concentration in plasma. A ratio of 1.0 indicates that the solute is reabsorbed at the same rate as water, while a ratio below 1.0 indicates that solute reabsorption exceeds water reabsorption.

Solution:

Let's analyze the transport curves along the proximal convoluted tubule (PCT):

(a) In the PCT, approximately 67% of filtered water is reabsorbed. For solutes like sodium, reabsorption matches water reabsorption, keeping their TF/P ratio constant at 1.0 (Curve B). For poorly reabsorbed substances like inulin or creatinine, water removal causes their concentration to rise above 1.0 (Curve A).

(b) For **bicarbonate ions (HCO_3^-)**, the PCT actively reabsorbs the vast majority (about 80 to 85%) of the filtered load via apical carbonic anhydrase mechanisms.

(c) Because bicarbonate is reabsorbed much faster than water along the PCT, its concentration in the remaining tubular fluid drops rapidly, which is represented by **Curve C**, where the TF/P ratio falls well below 1.0.

Final Answer: $\boxed{CurveC}$

Answer: (C)

Go Back to Question 12

Q13.

Solution

Concept: The kidneys counter chronic metabolic acidosis through two primary mechanisms: reclaiming filtered bicarbonate and synthesizing *new* bicarbonate ions. Reclaiming filtered bicarbonate simply prevents the loss of existing base, whereas generating *new* bicarbonate adds net base to the systemic circulation to replace what was consumed by metabolic acids.

Solution:

Let's examine the cellular pathways involved in generating new bicarbonate ions:

(a) Generating new bicarbonate requires excreting hydrogen ions bound to non-bicarbonate urinary buffers, primarily ammonium (NH_4^+) and titratable acids (such as phosphate).

(b) In response to chronic acidosis, proximal tubule cells increase **glutamine metabolism**. Each molecule of glutamine is metabolized into two ammonium ions (NH_4^+) and two alpha-ketoglutarate molecules.

(c) The alpha-ketoglutarate is further metabolized to produce **new bicarbonate ions (HCO_3^-)**, which are transported across the basolateral membrane into the blood. Concurrently, the NH_4^+ ions are secreted into the lumen and excreted in urine, resulting in a net gain of systemic base.

Final Answer: Glutamine metabolism and ammonium (NH_4^+) excretion in the proximal tubule.

Answer: (C)

Go Back to Question 13

Q14.

Solution

Concept: Cortisol is a primary glucocorticoid that regulates carbohydrate, protein, and lipid metabolism. Its primary metabolic function is to maintain adequate blood glucose levels, particularly during periods of stress or fasting, by altering metabolic pathways in a tissue-specific manner.

Solution:

Let's analyze the distinct actions of cortisol on skeletal muscle versus hepatic tissue:

(a) **Skeletal Muscle:** Cortisol acts as a catabolic hormone. It **promotes protein catabolism** (breakdown) into free amino acids and inhibits protein synthesis. It also reduces glucose uptake by decreasing the translocation of GLUT4 transporters to the cell membrane, conserving glucose for central tissues.

(b) **Hepatic Tissue:** Cortisol acts as an anabolic hormone for glucose production. It enters the hepatocyte nucleus and increases the transcription of key gluconeogenic enzymes (such as phosphoenolpyruvate carboxykinase). This **stimulates gluconeogenesis**, utilizing the amino acids released from skeletal muscle breakdown to synthesize new glucose, while also promoting hepatic glycogen synthesis.

Final Answer: Muscle: Promotes protein catabolism; Liver: Stimulates gluconeogenesis

Answer: (C)

Go Back to Question 14

Q15.

Solution

Concept: The hypothalamic-pituitary-thyroid axis is regulated by a negative feedback loop. The hypothalamus secretes Thyrotropin-Releasing Hormone (TRH), which stimulates the anterior pituitary to release Thyroid-Stimulating Hormone (TSH). TSH then binds to TSH receptors on the thyroid gland, driving the production and release of thyroid hormones (T_4 and T_3).

Solution:

Let's trace the feedback loop alterations caused by long-acting thyroid stimulator autoantibodies (such as those in Graves' disease):

(a) These autoantibodies bind directly to and activate the TSH receptor (**Target Y**) on thyroid follicular cells, mimicking the action of TSH.

(b) This continuous, unregulated activation drives excessive synthesis and secretion of thyroid hormones (T_4 and T_3), independent of pituitary control.

(c) These elevated levels of free T_4 and T_3 travel via the bloodstream to the anterior pituitary and hypothalamus, where they activate thyroid hormone receptors to trigger strong **negative feedback**. This feedback suppresses the transcription and secretion of TSH, dropping plasma TSH levels to near zero.

Final Answer: Suppressed TSH secretion via negative feedback

Answer: (B)

Go Back to Question 15

Q16.

Solution

Concept: The perception of pain can be modified by concurrent mechanical stimulation. The structural framework for this interaction is defined by the Gate Control Theory of Pain, which describes how different types of nerve fibers interact within the dorsal horn of the spinal cord.

Solution:

Let's analyze the synaptic interactions within the spinal cord dorsal horn:

(a) Noxious stimuli are carried by slow, unmyelinated C fibers and thinly myelinated A-delta fibers, which synapse on transmission cells within the spinothalamic tract to send pain signals to the brain.

(b) High-frequency stimulation of large, heavily myelinated **tactile A-beta ($A\beta$) afferent fibers** (which carry touch and vibration signals) activates inhibitory interneurons located within the substantia gelatinosa of the dorsal horn.

(c) These activated interneurons release inhibitory neurotransmitters (such as GABA or glycine) that presynaptically inhibit the pain fibers, closing the spinal "gate" and preventing pain signals from traveling up the spinothalamic tract. This mechanism provides the physiological basis for treatments like transcutaneous electrical nerve stimulation (TENS) and rubbing a stubbed toe.

Final Answer: Gate Control Theory of Pain

Answer: (B)

Go Back to Question 16

Q17.

Solution

Concept: Muscle spindles are specialized sensory receptors embedded within skeletal muscle that detect changes in muscle length and the rate of change in length, regulating the stretch reflex loop. They consist of internal intrafusal fibers surrounded by standard, force-producing extrafusal muscle fibers.

Solution:

Let's identify the functional roles of the motor neurons shown in the muscle spindle architecture:

(a) **Alpha (α) motor neurons (Fiber 1)** are large, heavily myelinated lower motor neurons that originate in the anterior horn of the spinal cord and **innervate extrafusal muscle fibers**. Activating these neurons generates the tension and force required for muscle contraction.

(b) **Gamma (γ) motor neurons (Fiber 2)** are smaller motor neurons that selectively **innervate the contractile ends of intrafusal muscle fibers**. Activating gamma motor neurons adjusts the tension and sensitivity of the muscle spindle receptor, preventing it from going slack during extrafusal muscle contraction (alpha-gamma coactivation).

Final Answer: Fiber 1 (Innervates Extrafusal Fibers)

Answer: (A)

Go Back to Question 17

Answer Key

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