

PREP XI RR

STUDY NOTES

PHYSIOLOGY

8

VIDEOS

135

PAGES

PREMIUM STUDY MATERIAL

PREP XI RR

Table of Contents follows on next page

#	Topic / Video Name	Pages
■ RR Reignite		
1	General Physiology	3 – 14
2	Nerve Muscle Physiology	15 – 32
3	CNS Physiology.	33 – 56
4	CVS Physiology	57 – 74
5	Respiratory Physiology	75 – 96
6	GIT Physiology	97 – 107
7	Renal Physiology	108 – 120
8	Endocrine and Reproductive Physiology	121 – 137

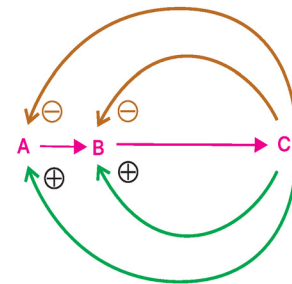
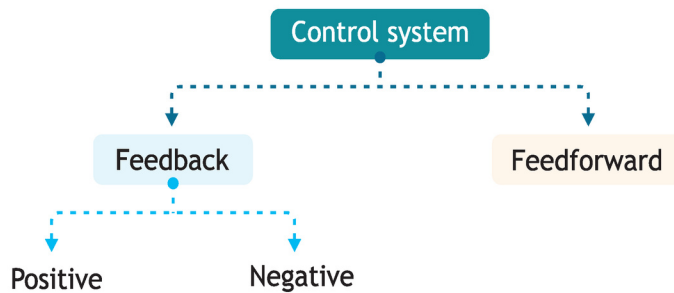


1. GENERAL PHYSIOLOGY

BIG QUESTION 1: What are the different types of control systems in the body?

Broad Answer: Control systems regulate body functions through feedback and anticipatory mechanisms.

PYQ: INICET 2020



DETAILED QUESTIONS

Q1.1: What is a positive feedback control system and when is it beneficial?

Answer:

- Output reinforces itself, forming a vicious cycle.
- Beneficial in:
 - o Blood clotting
 - o LH surge during ovulation
 - o Uterine contraction during childbirth (Ferguson reflex)
 - o Lactation (suckling of baby)
 - o Generation of nerve action potential
 - o Release of sarcoplasmic calcium during excitation-contraction coupling in cardiac muscle

Q1.2: What is a negative feedback control system?

Answer:

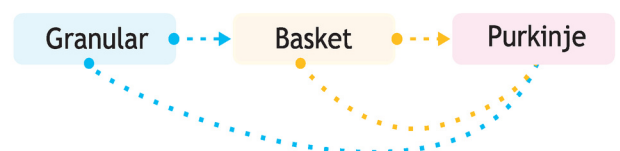
- Inhibitory input stabilises the system.
- Examples:
 - o Hypothalamic-Pituitary-Thyroid axis
 - o Baroreceptor reflex

Q1.3: What is a feedforward control system?

Answer:

PYQ: INICET 2021

- Anticipatory system where corrective actions occur before the change happens.
- Examples:
 - o Thermoregulation
 - o Increased heart rate and respiratory rate before exercise (psychic stimulation)
 - o Cephalic phase of gastric secretion



- o Receptive relaxation of stomach (Adaptive relaxation is not part of feedforward control)
- o Cerebellum's unidirectional output from granular to basket to Purkinje cells

Q1.4: What is meant by gain of a control system?

Answer:

- Gain = Correction / Error
- Indicates effectiveness of a control system.
- Example:
 - o BP at steady state = 100 mmHg
 - o BP after infusion = 160 mmHg
 - o Baroreceptor correction brings it to 120 mmHg
 - o Correction = 40 mmHg, Error = 20 mmHg
 - o Gain = $40 / 20 = 2$ (Negative direction)
- Infinite gain seen when correction is 100% (e.g., Kidney regulating BP and blood volume)



$$\text{Gain} = \frac{40}{20} = 2 \text{ (negative)}$$

$$\text{Gain} = -2$$

$$\frac{\text{Correction}}{\text{Error}} = \frac{\text{Correction}}{0} = \alpha$$

BIG QUESTION 2: How is body water distributed in the body?

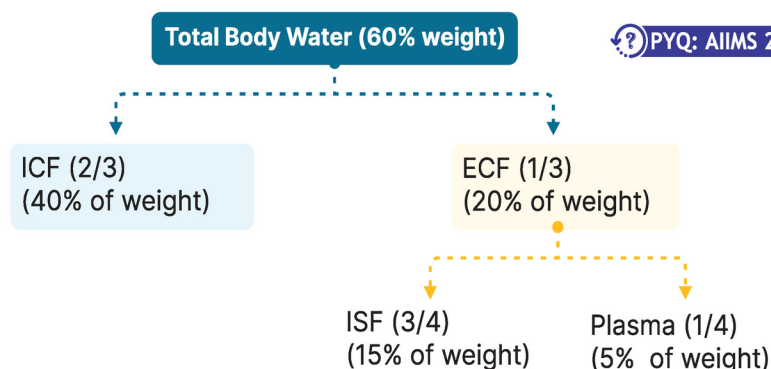
Broad Answer: Total body water is divided into intracellular and extracellular compartments.

DETAILED QUESTIONS

Q2.1: What is the distribution of body water?

Answer: Blood makes up to 8% of the body weight, 5% plasma, and 3% of total cell volume (RBC, WBC, and platelets)

Compartment	% of Body Weight	% of Total Body Water
Total Body Water (TBW)	60%	100%
Intracellular Fluid (ICF)	40%	66.6% (2/3 of TBW)
Extracellular Fluid (ECF)	20%	33.3% (1/3 of TBW)
Plasma	5%	25% of ECF
Interstitial Fluid (ISF)	15%	75% of ECF



🔍 PYQ: AIIMS 2020

🔍 PYQ: NEET PG 2021

Q2.2: What percentage of total body water is ECF?

Answer:

- 33.3% (1/3 of TBW)
- 20% of the body weight.

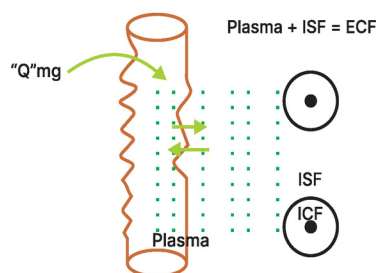
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Q2.3: What is transcellular fluid? Give examples.**Answer:**

- Fluid present in specific body cavities.
- Examples:
 - o CSF: 150 ml
 - o Intrapleural fluid: 10-20 ml
 - o Pericardial fluid: 50 ml
 - o Peritoneal fluid: 10 ml (males), 20 ml (females, post-ovulatory phase)
 - o Synovial fluid: ~ 1ml/large joint

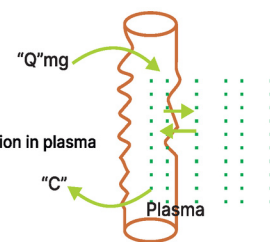
Q2.4: How is body water measured?**Answer:**

- Approximate: Based on body weight
- Exact: Using volume of distribution formula
 - o Volume of distribution = Q / C
 - Q = amount injected
 - C = plasma concentration
- If substance is metabolised/excreted:
 - o Modified formula: Volume = $(Q - e) / C$



$$\text{Volume of distribution} = \frac{Q}{C}$$

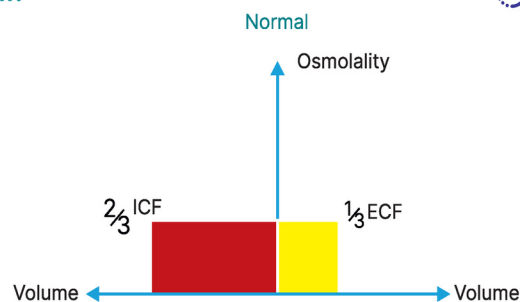
Q - total amount injected; C - concentration in plasma

**BIG QUESTION 3: What is the Darrow-Yannet (D-Y) Diagram and how does it explain fluid balance?****Broad Answer:**

D-Y diagram represents volume and osmolality changes in ICF and ECF compartments.

DETAILED QUESTIONS**Q3.1: What is the normal representation in a D-Y Diagram?****Answer:**

- Rectangles show ICF and ECF:
 - o Width = Volume
 - o Height = Osmolality



PYQ: FMGE 2023

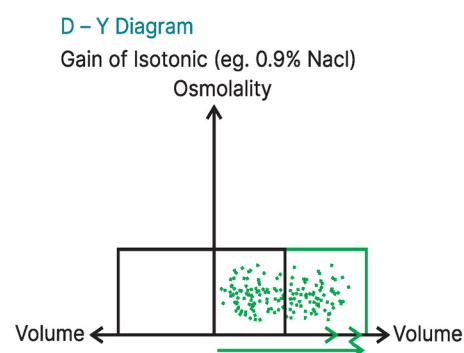
Q3.2: What indicators are used to measure various compartments?**Answer:**

Compartment	Indicator Used
Total Body Water	D ₂ O, Tritium oxide, Antipyrine
ECF Volume	Inulin, Sucrose, Na ⁺ , Iothalamate, Mannitol
ICF Volume	Measured indirectly as TBW - ECF
Plasma Volume	Evans' blue, I-albumin
ISF Volume	ECF - Plasma volume
RBC Volume	⁵¹ Cr, ⁵⁹ Fe tagged RBCs

Q3.3: What happens with a gain of isotonic fluid?

Answer:

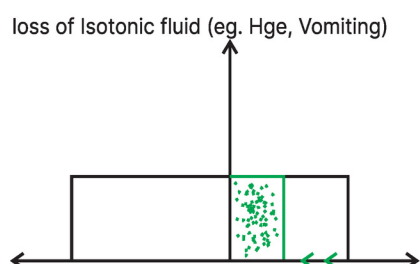
- ECF volume increases
- No change in osmolality
- Example: Isotonic saline (0.9% NaCl) infusion



Q3.4: What happens with a loss of isotonic fluid?

Answer:

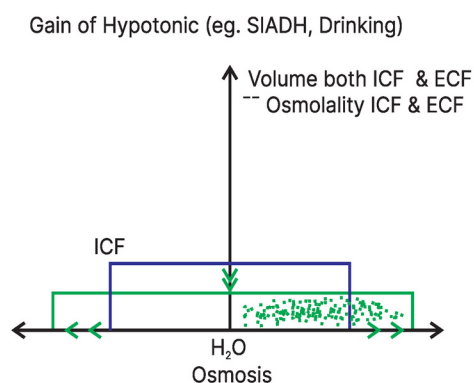
- ECF volume decreases
- No change in osmolality
- Examples: Haemorrhage, Vomiting



Q3.5: What happens with a gain of hypotonic fluid?

Answer:

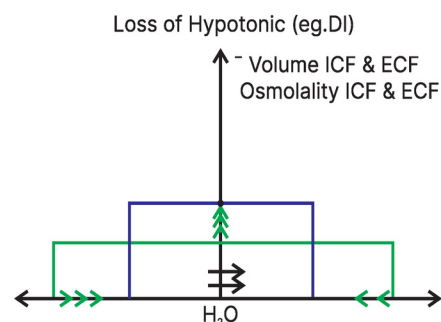
- ECF volume increases
- Osmolality of both ECF and ICF decreases
- Water moves into cells, increasing ICF volume
- Examples:
 - o SIADH
 - o Psychogenic polydipsia



Q3.6: What happens with a loss of hypotonic fluid?

Answer:

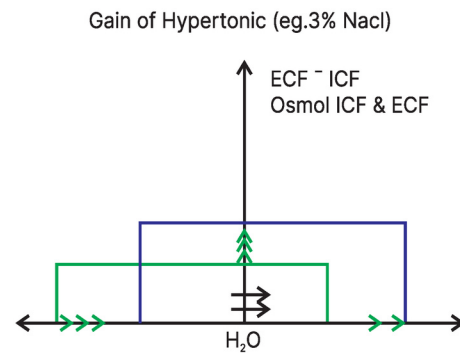
- ECF volume decreases
- ECF becomes hypertonic
- Water moves from ICF to ECF, decreasing ICF volume
- Example: Diabetes insipidus



Q3.7: What happens with a gain of hypertonic fluid?

Answer:

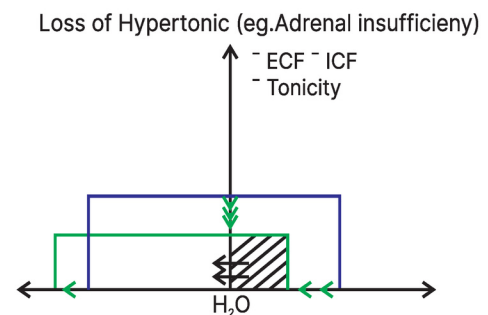
- ECF volume increases
- ECF osmolality increases
- Water moves out of cells, decreasing ICF volume
- Example: 3% NaCl infusion



Q3.8: What happens with a loss of hypertonic fluid?

Answer:

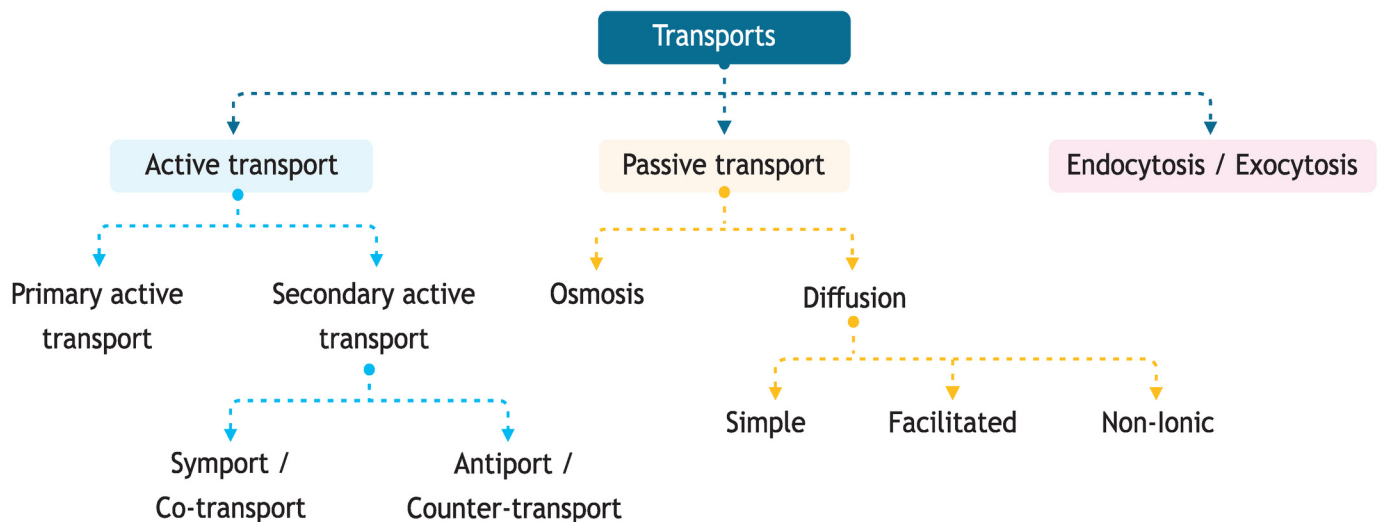
- Solute is lost more than water
- Water shifts from ECF to ICF
- ECF volume decreases, ICF volume increases
- Osmolality decreases in both compartments
- Example: Adrenal insufficiency



BIG QUESTION 4: What are the types and mechanisms of transport across the cell membrane?

Broad Answer:

Substances cross cell membranes via active or passive transport mechanisms, which may involve carrier or channel proteins.



DETAILED QUESTIONS

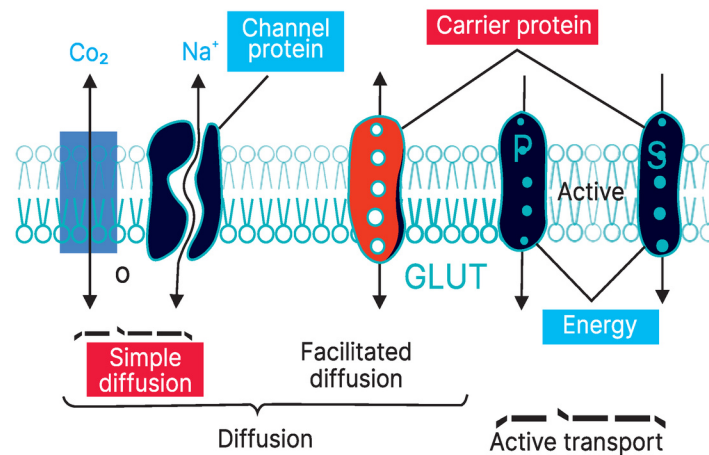
Q4.1: What are the types of transport across cell membranes?

Answer:

Type	Description	Example
Primary Active	Direct energy utilisation (ATP)	$\text{Na}^+ - \text{K}^+ - \text{ATPase}$

Secondary Active	Indirect energy utilisation via another ion gradient	SGLT (symport), NCX (antiport)
Passive Transport	No energy required; downhill transport	Simple and facilitated diffusion
Endocytosis/Exocytosis	Vesicular transport of large molecules	—

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Q4.2: What is the difference between active and passive transport?

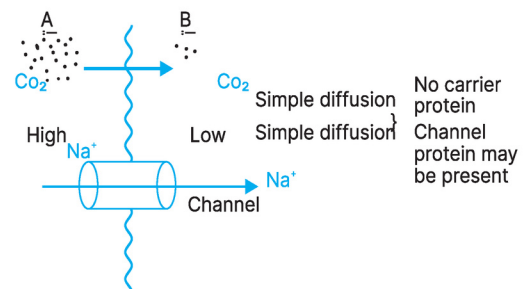
Answer:

Feature	Active Transport	Passive Transport
Energy Requirement	Requires ATP (direct or indirect)	No energy required
Direction	Against concentration gradient (uphill)	Along concentration gradient (downhill)

Q4.3: What is simple diffusion?

Answer:

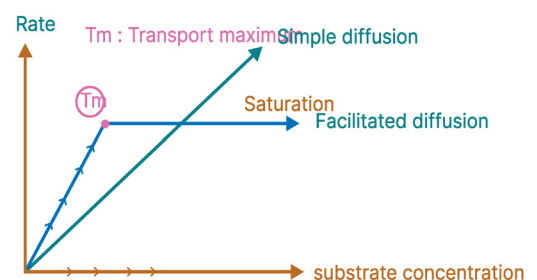
- Movement of non-polar or very small molecules from high to low concentration
- No energy or carrier protein required (channel protein may be present)
- Examples:
 - o CO_2 crosses without carrier or channel protein
 - o Na^+ crosses with the help of a channel protein



Q4.4: What is facilitated diffusion?

Answer:

- Movement from high to low concentration with a carrier protein
- Energy independent
- Example: Glucose transported via GLUT proteins



Q4.5: What is the role of channel proteins in transport?

Answer:

- Facilitate passive transport (diffusion)
- Types of channel proteins:

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- o Voltage-gated
- o Ligand-gated
- o Stretch-sensitive (Mechanosensitive)

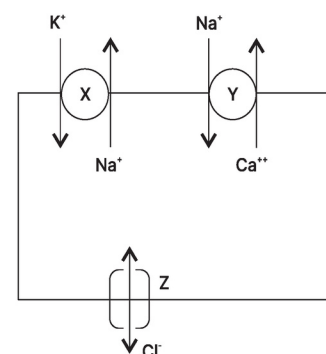
Q4.6: What is the role of carrier proteins?

Answer:

- Bind to substance → Conformational change → Release across membrane
- Involved in both active and facilitated diffusion

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Q4.7: A model cell with three different transporters (X, Y, Z) and an RMP of -75 mV is shown. Consider the intracellular and extracellular concentrations of all three ions to be typical of a normal cell. Which of the following best describes transporter Y?



- A. Facilitated diffusion
- B. Primary active transport
- C. Secondary active transport
- D. Simple diffusion

Answer: C. Secondary active transport

- Transporter Y requires ATP and moves ions against the concentration gradient.

BIG QUESTION 5: What are GLUT and SGLT transporters and their physiological relevance?

Broad Answer:

GLUT and SGLT transporters mediate glucose transport across membranes via facilitated diffusion or secondary active transport.

DETAILED QUESTIONS

Q5.1: What are the important GLUT transporters and their expression sites?

Answer:

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Transporter	Expression Site	Function/Notes
GLUT 1	RBC, BBB, Placenta	Universal GLUT, major GLUT in placenta/fetus
GLUT 2	β -cells of pancreas, Liver, Basolateral membrane of PCT & intestinal epithelial cells	Involved in glucose sensing & transport
GLUT 3	Neuronal membrane, WBC	Major neuronal GLUT
GLUT 4	Muscle & adipose tissue	Insulin-responsive GLUT
GLUT 5	Luminal membrane of intestine, sperm	Fructose transporter
GLUT 8	Blastocyst	Insulin-responsive GLUT in blastocyst
GLUT 11	—	Fructose transporter

Q5.2: What is the difference between GLUT and SGLT transporters?**Answer:**

- **GLUT:** Facilitated diffusion (energy independent)
- **SGLT:** Secondary active transport (energy dependent via Na^+ gradient)

Q5.3: What are the types and expression sites of SGLT transporters?**Answer:**

Transporter	Expression Site	Function/Notes
SGLT 1	GIT (intestinal luminal membrane), Kidney (Proximal Straight Tubule - PST)	Absorbs 100% glucose & Na^+ from lumen (GIT), 10% glucose reabsorption in kidney
SGLT 2	Kidney (Proximal Convoluted Tubule - PCT)	Absorbs 90% glucose & Na^+ , basolateral glucose transport via GLUT 2
SGLT 3	Skeletal muscle, Small intestine	Acts as glucose sensor

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Q5.4: Which of the following SGLT transporters is responsible for absorbing 90% of glucose in the kidney?

- A. SGLT 1
- B. SGLT 2
- C. SGLT 3
- D. GLUT 1

Answer: B. SGLT 2

- Located in PCT, absorbs 90% of glucose from kidney filtrate.

Q5.5: What is transport maximum (T_m) and how does it relate to facilitated diffusion?**Answer:**

- T_m is the maximum rate of transport when all carrier proteins are saturated.
- Facilitated diffusion exhibits T_m ; simple diffusion does not.

Q5.6: How does Fick's law apply to simple diffusion?**Answer:**

- Rate (J) = $D \times A \times \Delta C / x$
 - o D = Diffusion constant
 - o A = Surface area
 - o ΔC = Concentration difference
 - o x = Membrane thickness

BIG QUESTION 6: What happens to glucose transport when membrane permeability is altered?**Broad Answer:**

In absence of transporters and permeability, glucose cannot cross the membrane, leading to osmotic imbalance.

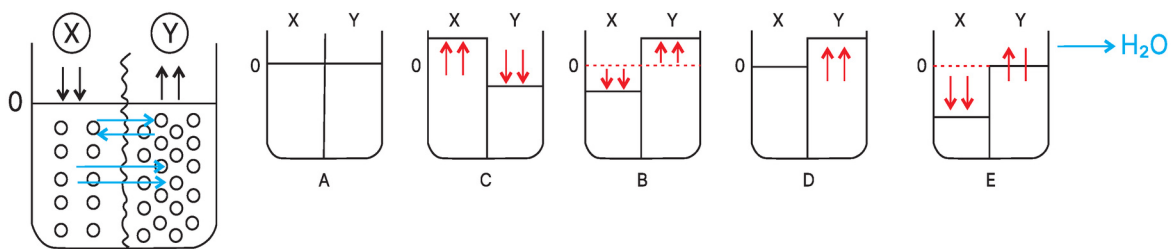
DETAILED QUESTIONS

Q6.1: What happens if the membrane is impermeable to glucose and no transporters are present?

Answer:

- Glucose cannot cross the membrane.
- Osmotic imbalance develops.
- Water shifts occur to achieve equilibrium.
- Compartment volumes change accordingly.

Q6.2: Two compartments (X and Y) are separated by a typical biological membrane. Glucose concentrations in compartments X and Y at time zero are shown. There are no transporters for glucose, and the membrane is impermeable to glucose. Which of the following best represents the compartment volumes after equilibrium?



- A. A
B. B
C. C
D. D

Answer: B. B

- Water moves to equalise osmotic pressure, altering compartment volumes, but glucose remains confined.

BIG QUESTION 7: What is the structure and function of the cell cytoskeleton?

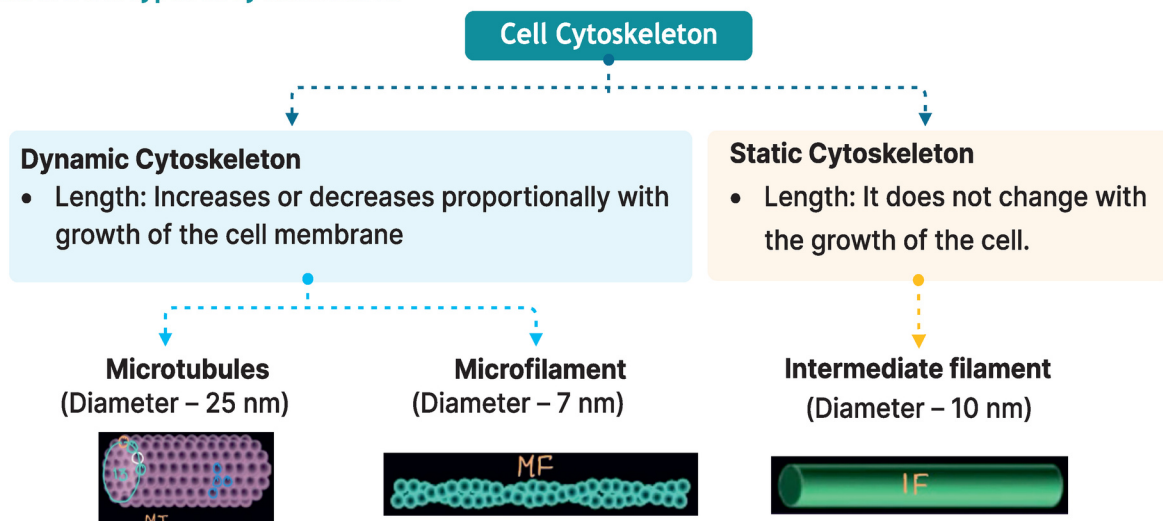
Broad Answer:

The cytoskeleton provides structural support, aids intracellular transport, and is involved in motility and cell division.

DETAILED QUESTIONS

Q7.1: What are the types of cytoskeleton?

Answer:

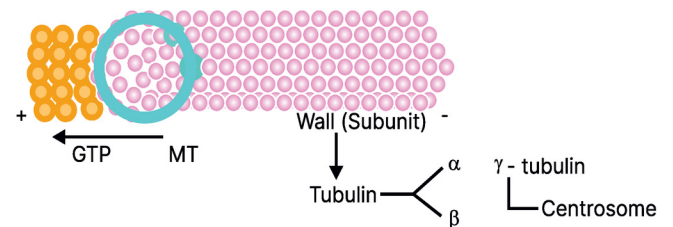


Type	Diameter	Key Features
Microtubules	25 nm	Dynamic, cell transport, division, motility
Microfilaments	7 nm	Dynamic, composed of actin, abundant in cells
Intermediate Filaments	10 nm	Static, provide structural stability, used as cell markers

Q7.2: What are the properties and functions of microtubules?

Answer:

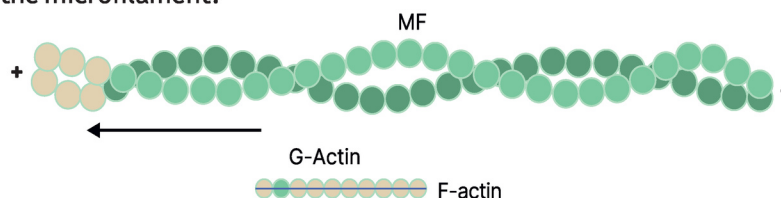
- Composed of α and β tubulin subunits.
- 13 protofilaments alternate to form microtubules.
- γ -tubulin forms a mitotic spindle at centrioles.
- Present in cilia and flagella for motility.
- Responsible for intracellular transport.
- Polymerisation occurs at + end (growth), depolymerisation at - end.
- GTP provides energy for polymerisation.



Q7.3: What are the properties and functions of microfilaments?

Answer:

- Composed of actin, the most abundant intracellular protein.
- G-actin (globular) polymerises to form F-actin (filamentous).
- Two F-actins intertwine to form one microfilament.
- Polymerisation occurs at + end during cell growth.
 - o The end remains non-growing.
 - o When the cell grows $\rightarrow$ Increase polymerization $\rightarrow$ +ve end The other end where the cell does not grow is known as the -ve end of the microfilament.



Q7.4: What are intermediate filaments and their significance?

Answer:

- Fixed structure, no subunits.
- Provide structural stability.
- Used as cell markers for different cell types.

Intermediate Filament	Cell Type Identified
Cytokeratin	Epithelial cells
Vimentin	Mesenchymal cells
Desmin	Skeletal muscle cells
GFAP	Astrocytes

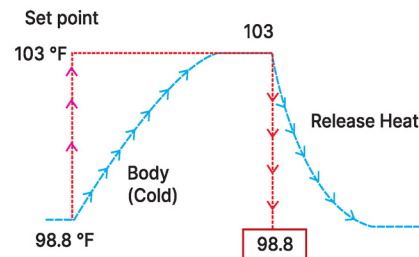
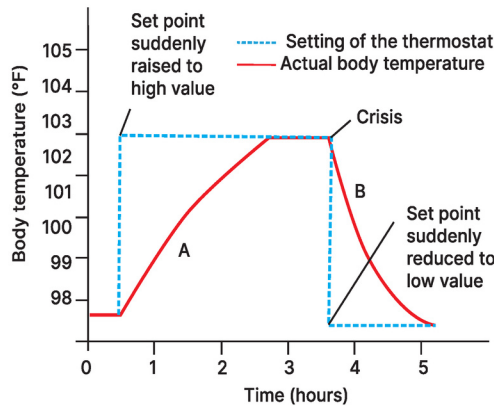
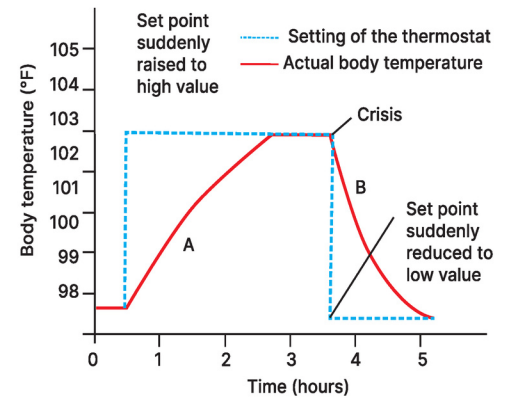
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Q7.5: The following diagram shows the change in hypothalamic set points to the body temperature, Which change will be seen at A as compared to B.

- A. Sweating
- B. Increased blood flow to the skin
- C. Shivering
- D. Shutdown of chemical thermoregulator.

Answer: C. Shivering

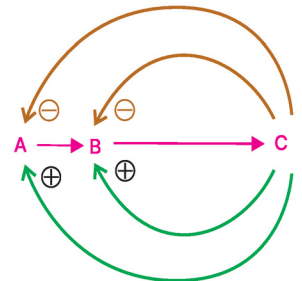
- A. Vasoconstriction, Piloerection, epinephrine secretion and shivering
- B. Vasodilation and sweating.



Q7.6: How do positive and negative feedback control systems differ?

Answer:

Positive feedback control system	Negative feedback control system
Input returned is of stimulatory type, So, if C is stimulating B and again B will produce C, the pathway will move in the same direction also known as the Vicious Cycle.	In the negative feedback control system, the C will give inhibitory input to B or A.
It destabilizes our control system	It stabilizes our control system

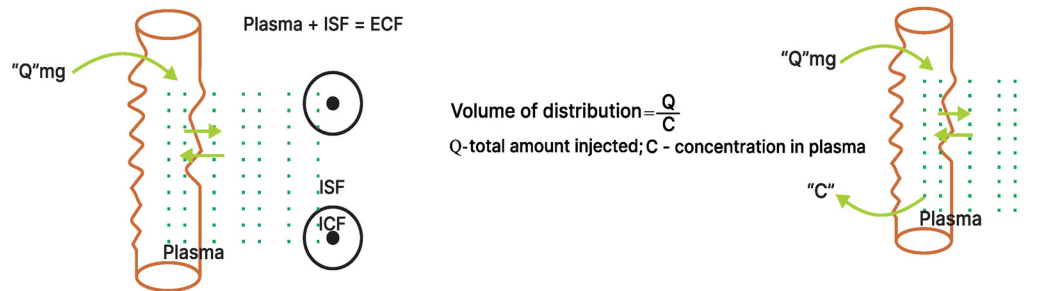


Q7.7: How is the concept of volume of distribution applied in pharmacology when using substances like inulin to measure body water, and how is the formula modified for substances that undergo metabolism and excretion?

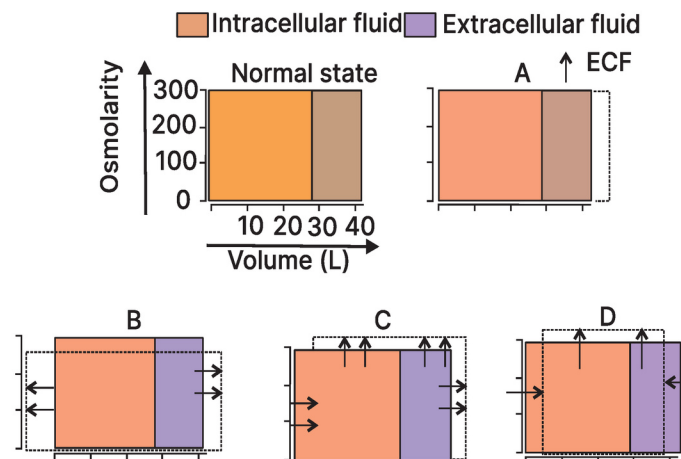
Answer:

- Inulin (Q mg) is injected into capillaries to measure body water.
- It distributes evenly in plasma and interstitial fluid (ISF).
- Once equilibrium is reached, plasma concentration is measured using a formula.
- Inulin is inert and not metabolized significantly.
- For metabolized substances, metabolism and excretion must be accounted for.
- In such cases, the formula is modified accordingly.

$$\text{Volume} = \frac{Q-e}{c} \quad e \rightarrow \text{excreted or metabolized}$$



Q7.8: Q. In each diagram, the normal state (solid line) is superimposed on the abnormal state (dashed lines) to illustrate the shifts in the volume (width of rectangles) and total osmolarity (height of rectangles) of the ECF and ICF. Which diagram represents the changes (after osmotic equilibrium) in ECF and ICF volumes and osmolarities after infusion of 3% NaCl?



- A. A
- B. B
- C. C
- D. D

Answer: C. C



2. NERVE MUSCLE PHYSIOLOGY

BIG QUESTION 1: What is the basic structure and functional organization of a single neuron?

Broad Answer: A neuron has three main structural parts: **dendrites**, **soma (cell body)**, and **axon**.

- Dendrites are **afferent** (receive signals).
- The soma (perikaryon) contains the nucleus and organelles.
- The axon is **efferent** and initiates action potentials. It originates from the **axon hillock**, continues through the **initial segment**, and ends in **axon terminals (telodendria)**.
- The axon may be **myelinated**, with **Nodes of Ranvier** in between the myelin segments.

DETAILED QUESTIONS

Q1.1: What is the function of dendrites in a neuron?

Answer: Dendrites receive signals from other neurons and are termed **afferents**.

Q1.2: What is the soma of a neuron also known as?

Answer: The soma is also known as the **perikaryon**.

Q1.3: Which neuron has the smallest cell body and what is its size?

Answer: The **granular cell of the cerebellum** has the smallest cell body, measuring **5 μm** .

Q1.4: Which neuron has the largest cell body and what is its size?

Answer: The **anterior horn cell of the spinal cord** has the largest cell body, measuring **135 μm** .

Q1.5: What is the axon termed as and what is its function?

Answer: The axon is termed an **efferent** structure and conducts impulses **away** from the soma.

Q1.6: What is the axon hillock and where is it located?

Answer: The **axon hillock** is the part of the cell body where the **membrane thickens** and connects to the axon.

Q1.7: What is the initial segment and what is its length?

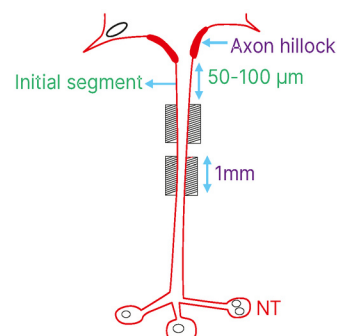
Answer: The **initial segment** follows the axon hillock and is **50-100 μm** in length.

Q1.8: What are axon knobs or telodendria?

Answer: They are **multiple small vesicular endings** at the terminal end of axons.

Q1.9: What is the length of a myelin sheath in a long neuron?

Answer: The myelin sheath can be **1 mm** in length.



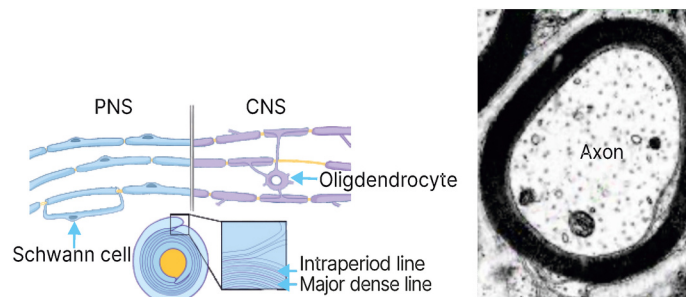
Q1.10: What is the name and length of the region between two myelin sheaths?

Answer: It is called the **Node of Ranvier** and is 1 μm in length.

BIG QUESTION 2: How does myelination differ between the CNS and PNS?

Broad Answer:

Characteristic	Peripheral Nervous System (PNS)	Central Nervous System (CNS)
Myelination	One Schwann cell per neuron.	One oligodendrocyte for multiple neurons (up to 30).
Demyelinating Disease	Localized disease, e.g., Guillain-Barre syndrome.	Diffuse disease, e.g., multiple sclerosis.

**DETAILED QUESTIONS****Q2.1: What cell forms the myelin sheath in the PNS?**

Answer: Schwann cell.

Q2.2: What cell forms the myelin sheath in the CNS?

Answer: Oligodendrocyte.

Q2.3: How many neurons can a single oligodendrocyte myelinate?

Answer: Up to 30 neurons.

Q2.4: What type of demyelinating disease affects the PNS?

Answer: Guillain-Barre Syndrome, a localized disease.

Q2.5: What type of demyelinating disease affects the CNS?

Answer: Multiple Sclerosis, a diffuse disease.

BIG QUESTION 3: What are the functional zones of a neuron and their electrophysiological roles?

Broad Answer:

Neurons are functionally divided into areas based on electrical activity:

- **Dendrites & Soma:** Generate **graded potentials** (EPSP/IPSP).
- **Initial Segment** (motor neurons) or **Nodes of Ranvier** (sensory neurons): Site of **action potential** initiation.
- Voltage-gated Na^+ channels are maximally concentrated at the **Nodes of Ranvier**, followed by the **initial segment**.

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DETAILED QUESTIONS

Q3.1: Where are EPSPs and IPSPs generated in the neuron?

Answer: In the dendrites and soma.

Q3.2: Where is the action potential initiated in a sensory neuron?

Answer: At the Nodes of Ranvier.

Q3.3: Where is the action potential initiated in a motor neuron?

Answer: At the initial segment of the axon.

Q3.4: Rank the density of voltage-gated Na^+ channels in different neuron regions.

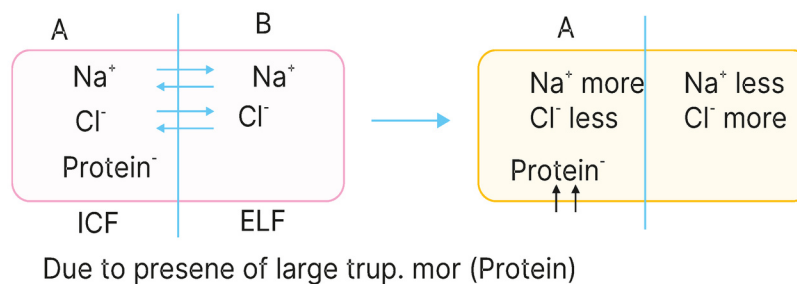
Answer:

Region	Na^+ Channel Density (channels/ μm^2)
Nodes of Ranvier	2000-12000
Initial Segment	350-500
Telodendria	20-75
Myelin Sheath Surface	2

BIG QUESTION 4: What is the Gibbs-Donnan effect and how does it influence ion distribution?

Broad Answer: The Gibbs-Donnan effect describes the uneven distribution of permeable ions across a semi-permeable membrane due to the presence of impermeable charged molecules (e.g., proteins). These proteins attract counter-ions and repel co-ions, leading to osmotic and electrical gradients.

Donnan effect / Gibbs-Donnan equation



DETAILED QUESTIONS

Q4.1: What causes the Donnan effect?

Answer: The presence of large impermeable proteins in one compartment.

Q4.2: How are Na^+ and Cl^- distributed in Gibbs-Donnan equilibrium?

Answer:

- Na^+ is attracted into the protein-containing compartment.
- Cl^- is repelled from the protein-containing compartment.

Q4.3: What compartments are involved in Donnan equilibrium?**Answer:**

- ICF (A): Contains impermeable proteins.
- ECF (B): Does not contain proteins.

Q4.4: What is the outcome of the Gibbs-Donnan effect?**Answer:** A disruption of normal ion distribution, creating an **electric potential** across the membrane.**Q4.5: Explain the sequence of ionic distribution changes that occur due to the Gibbs-Donnan effect when impermeable proteins are added to one compartment.****Answer:**

Na⁺ and Cl⁻ given into compartments ICF(A) & ECF (B) respectively

↓

Distribution of Na⁺ and Cl⁻ across both the compartments equally through Semi permeable membrane

↓

Later, proteins (negative charge) which are impermeable are given in A compartment only

↓

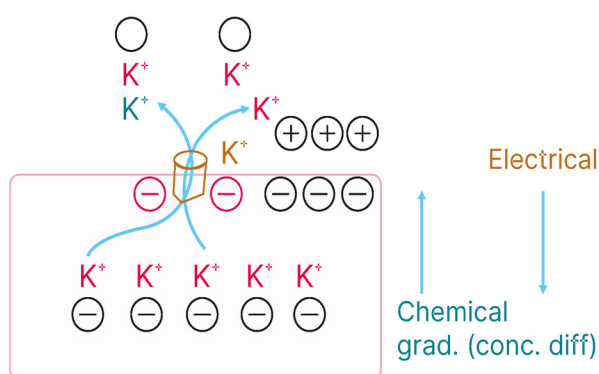
Proteins (negative charge) repels Cl⁻ ions & attracts more Na⁺ ions

↓

Thus large impermeable molecule like proteins disrupt the normal distribution of ions and molecules across the compartments through the semi permeable membrane

BIG QUESTION 5: What are leak channels and how do they contribute to resting membrane potential?

Broad Answer: Leak channels are **non-gated ion channels** that allow ions (especially K⁺) to flow across the membrane during resting conditions. They help establish the **resting membrane potential** by allowing K⁺ to move out of the cell, creating a negative internal environment, until equilibrium is achieved.

**DETAILED QUESTIONS****Q5.1: What are leak channels?****Answer:** Ion channels that remain **open** even at **resting conditions**.**Q5.2: Which ion primarily moves through leak channels?****Answer:** Potassium (K⁺).

Q5.3: What happens to K^+ during leak channel activity?**Answer:**

- K^+ moves out of the cell due to its concentration gradient.
- Later, it trickles back in, reaching equilibrium.

K^+ inside the cell moves out through leak channels



After sometime K^+ ions will trickle back into cell and this continues till equilibrium is maintained (outward = inward movement of K^+)



But the charge of the cell membrane (inside and outside of cell) is different



E_p /Isoelectric potential

Q5.4: What does the equilibrium of K^+ movement across leak channels define?**Answer:** The equilibrium potential or isoelectric potential of the membrane.**Q5.5: Why is there a voltage difference across the membrane despite K^+ equilibrium?****Answer:** Because chemical gradients and electrical charge distribution across the membrane differ.**BIG QUESTION 6: What is the Nernst Equation and how is it applied to neuronal physiology?****Broad Answer:** The Nernst Equation calculates the equilibrium potential (E_n) of an ion across a semi-permeable membrane, based on its intracellular and extracellular concentrations. The simplified form of the equation is:

$$E_q = \frac{RT}{ZF} \log \frac{[x]_{\text{outside}}}{[x]_{\text{inside}}} \quad \text{PYQ: INICET 2023} \quad \text{PYQ: NEET PG 2023}$$

$$E_q = 2.3 \times \frac{RT}{ZF} \log_{10} \frac{[x]_{\text{outside}}}{[x]_{\text{inside}}}$$

DETAILED QUESTIONS**Q6.1: What does the Nernst Equation calculate?****Answer:** It calculates the equilibrium potential (E_n) of an ion.**Q6.2: Write the complete thermodynamic Nernst equation.****Answer:**

$$E_q = \frac{RT}{ZF} \log \frac{[x]_{\text{outside}}}{[x]_{\text{inside}}}$$

Q6.3: What is the simplified form of the Nernst equation for monovalent ions at body temperature?**Answer:**

$$E_q = 2.3 \times \frac{RT}{ZF} \log_{10} \frac{[x]_{\text{outside}}}{[x]_{\text{inside}}}$$

$$E_{\text{ion}} = 61 \cdot \log \left(\frac{[\text{ion}]_{\text{outside}}}{[\text{ion}]_{\text{inside}}} \right)$$

Q6.4: What does each variable in the Nernst equation represent?**Answer:**

Symbol	Meaning
R	Universal gas constant
T	Absolute temperature (Kelvin)
z	Valence of the ion
F	Faraday constant
[ion]	Concentration of ion

Q6.5: How is the Nernst equation applied to determine the RMP of a model cell?**Answer:**

In a **model cell**, only **K⁺ permeability** is considered. Given:

- [K⁺]inside= 140 mM
- [K⁺]outside= 14 mM

Using the Nernst equation:

$$E_K = 61 \cdot \log \left(\frac{[K^+] \text{ outside}}{[K^+] \text{ inside}} \right)$$

$$E_K = 61 \cdot \log \left(\frac{14}{140} \right) = 61 \cdot \log (0.1) = 61 \cdot (-1) = -61 \text{ mV}$$

Thus, the equilibrium potential for potassium (and RMP of this model cell) is **-61 mV**.

Q6.6: Why does the potential become negative?

Answer: Because the **inside concentration is higher**, the logarithm becomes negative.

BIG QUESTION 7: What are the equilibrium potentials of various ions in mammalian cells?

Broad Answer: Different ions have different equilibrium potentials, contributing variably to RMP. These are determined by the **concentration gradient** and calculated via the Nernst equation.

Ion	Equilibrium Potential (mV)
Sodium	+63
Chloride	-70
Potassium	-90
Calcium	+132

DETAILED QUESTIONS**Q7.1: What is the equilibrium potential of sodium in mammalian cells?**

Answer: +63 mV

Q7.2: What is the equilibrium potential of chloride?

Answer: -70 mV

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Q7.3: What is the equilibrium potential of potassium?

Answer: -90 mV

Q7.4: What is the equilibrium potential of calcium?

Answer: +132 mV

Q7.5: What is the Goldman equation and why is it used?

Answer:

- RMP is calculated using Goldman-Hodgkin-Katz-Flux Reaction
- The **Goldman equation** calculates the **resting membrane potential (RMP)** of cells more accurately by considering the **permeability and concentration of multiple ions**, not just one like Nernst.

Q7.6: When is the Goldman equation preferred over the Nernst equation?

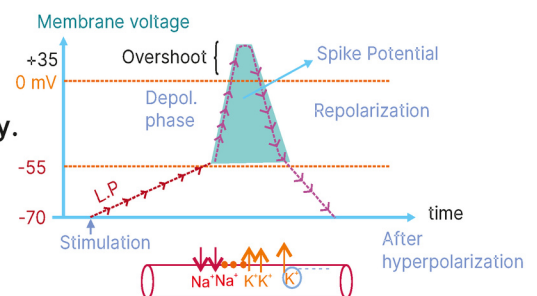
Answer: When multiple permeable ions contribute to the RMP.

BIG QUESTION 8: What are the physiological implications of equilibrium potential and membrane permeability?

Broad Answer:

- The **resting membrane potential** is closest to the equilibrium potential of K^+ , because the membrane is most permeable to it.
- The ion with the **highest permeability** influences RMP the most.
- RMP is slightly more positive than EK due to minimal Na^+ permeability.
- RMP (~ -70 mV) is also maintained by the Na^+/K^+ ATPase pump.

🔍 PYQ: NEET PG 2023 🔍 PYQ: FMGE 2018



DETAILED QUESTIONS

Q8.1: Which ion has the highest permeability in resting cells?

Answer: Potassium (K^+)

Q8.2: Why is RMP more positive than EK alone?

Answer: Because of low Na^+ permeability, which pulls RMP slightly upward from EK.

Q8.3: What actively maintains RMP?

Answer: The Na^+/K^+ ATPase pump.

Q8.4: What is the order of permeability in a resting neuronal membrane?

Answer: $K^+ \gg Cl^- > Na^+$

Q8.5: What are the resting membrane potentials (RMPs) of various excitable cells?

Answer: Different excitable cells have varying resting membrane potentials based on their ionic permeability and function.

Cell Type	RMP (mV)
Neuron	-70 to -90
Skeletal muscle	-85 to -95
Cardiac muscle	-80 to -90
Smooth muscle	-50 to -60
Oocyte	-70
Erythrocyte	-8 to -12
Photoreceptor	-40 (dark) to -70 (light)

BIG QUESTION 9: How is the action potential generated and propagated in neurons?

Broad Answer: The action potential in neurons is initiated when the local membrane potential reaches a threshold (around -55 mV), leading to activation of voltage-gated Na^+ channels. This involves:

 PYQ: NEET PG 2019

- **Depolarization phase:** Membrane potential rises sharply toward +35 mV due to Na^+ influx.
- **Repolarization phase:** Closure of Na^+ channels and opening of voltage-gated K^+ channels.
- **Hyperpolarization:** K^+ efflux exceeds normal RMP.

DETAILED QUESTIONS

Q9.1: What triggers the action potential in neurons?

Answer: A local potential reaching threshold potential (-55 mV) activates voltage-gated Na^+ channels.

Q9.2: What happens during the depolarization phase of an action potential?

Answer:

- Rapid Na^+ influx occurs.
- Membrane potential reaches +35 mV.

Q9.3: What happens during the repolarization phase?

Answer:

- Na^+ channels close.
- Voltage-gated K^+ channels open, leading to K^+ efflux.

Q9.4: What is the hyperpolarization phase?

Answer: Excessive K^+ efflux causes membrane potential to fall below RMP.

Q9.5: What maintains RMP after an action potential?

Answer: Na^+/K^+ ATPase pump restores ion gradients post-action potential.

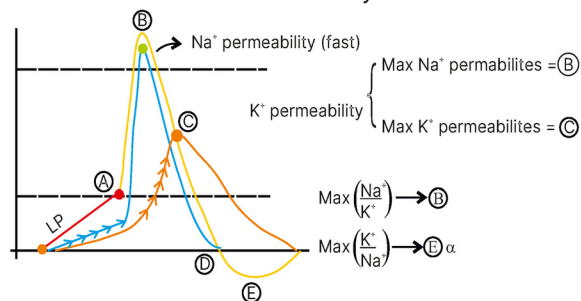
Q9.6: How does ion channel permeability change during the action potential?

Answer:

- Permeability of the sodium channel would be maximum at Point B.
- Permeability of the potassium channel would be maximum at point C.

- The point where the ratio of permeability of Na, K is maximum → Point B.
- Point where the permeability of K, Na ratio is maximum → Point E (infinity).
- Na^+ channels open early and rapidly during depolarization.
- K^+ channels open later, during repolarization.
- Each channel type has **distinct kinetics** contributing to AP shape.

Action Potential: Ionic Permeability



PYQ: NEET PG 2022

PYQ: AIIMS 2020

Q9.7: When do Na^+ channels open and close during the AP?

Answer:

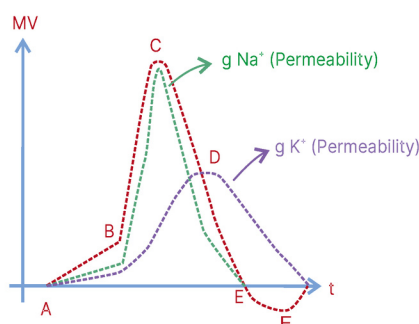
- Open quickly at **threshold**
- Close at **peak of depolarization**.

Q9.8: When do K^+ channels open and close during the AP?

Answer:

- Open later (start of repolarization).
- Close after **hyperpolarization**.

Q9.9: What does the purple curve in the K^+ channel graph represent?



Answer: The ionic current due to K^+ efflux, affecting membrane potential.

Q9.10: What is the sequence of permeability during AP?

Answer:

Phase	Ion Channel Activity
Depolarization	$\uparrow \text{Na}^+$ permeability
Repolarization	$\downarrow \text{Na}^+$, $\uparrow \text{K}^+$ permeability
Hyperpolarization	$\downarrow \text{K}^+$ (slow return to RMP)

Q9.11: Compare the changes in conductance/permeability of Sodium and Potassium channels during an action potential using appropriate conductance curves.

Answer:

PYQ: AIIMS 2018

Sodium Channel	Potassium Channel
• gNa^+ (green curve) represents Na^+ permeability • Local potential generation opens Na^+ channels. • Number of open Na^+ channels increases rapidly on reaching threshold. • After peak, Na^+ channels inactivate. • The green curve = gNa^+: represents sodium conductance/permeability during action potential.	• gK^+ (purple curve) represents K^+ permeability • K^+ channels also open with Na^+ channels but more slowly. • Delayed peak permeability occurs after Na^+ channels inactivate. • K^+ channels stay open longer and help in repolarization. • The purple curve = gK^+: represents potassium conductance/permeability during action potential.

BIG QUESTION 10: How are nerve fibers classified and what are their conduction properties?

Broad Answer: Nerve fibers are classified by myelination, diameter, conduction velocity, and function. Two major classification systems:

- **Erlanger & Gasser** for somatic fibers (A, B, C).
- **Lloyd & Hunt** for muscle afferents (I, II, III, IV).

PYQ: FMGE 2023 **PYQ: NEET PG 2020, 2022**

Myelination	Fiber type		Numerical Classification (By Lloyd and Hunt)	Functions	Conduction velocity (m/sec)	Diameter
Myelinated	A	Alpha	Ia Ib	Proprioception and somatic motor	70-120	12-20
		Beta	II	Touch and pressure	30-70	5-12
		Gamma		Efferent to muscle spindle	15-30	
		Delta	III	Pain (fast) and temperature (cold)	12-30	2-5
Myelinated	B			Preganglionic autonomic	3-12	
Unmyelinated	C		IV	Pain (slow), temperature (warm), and postganglionic sympathetic	0.5-2	

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DETAILED QUESTIONS

Q10.1: Which fiber is fastest and largest in diameter?

Answer: A α (Alpha) fiber — 70-120 m/s, 13-20 μ m.

Q10.2: What is the classification and function of C fibers?

Answer:

- Type IV
- Unmyelinated
- Function: **Slow pain, temperature, autonomic post-ganglionic**
- Warm, burning pain, and freezing pain: Carried by C fiber.
 - o Cold sensation: Carried by A δ fiber

Q10.3: Which fibers are affected first in hypoxia, pressure, and local anesthesia?

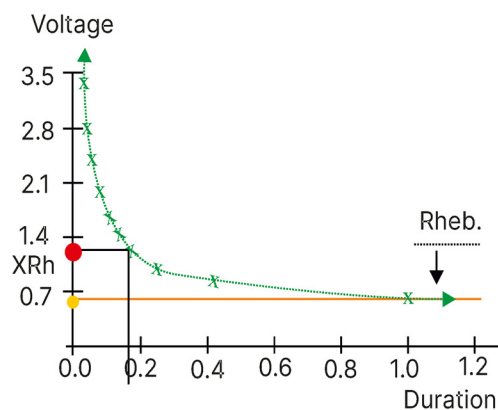
Answer:

Condition	Order of Block
Pressure	A α > B > C
Hypoxia	B > A > C
Local Anesthesia	A γ and A δ >> A α and AB >> B >> C

Q10.4: What is the significance of chronaxie and rheobase in nerve stimulation?

Answer:

- **Rheobase:** Minimum voltage required to trigger AP with long-duration stimulus.
- **Chronaxie:** Minimum time required to stimulate the neuron at **2 $\times$ rheobase** voltage.
- **Utilization Time:** It is the minimum duration of Rheobase voltage that is required to generate an action potential.



Q10.5: What is chronaxie?

Answer: Minimum duration of stimulation at **2 $\times$ rheobase** voltage needed to excite a neuron.

Q10.6: What is rheobase?

Answer: Minimum current intensity of long duration needed to excite a neuron.

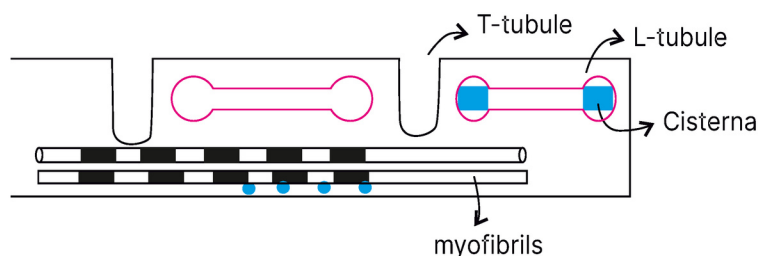
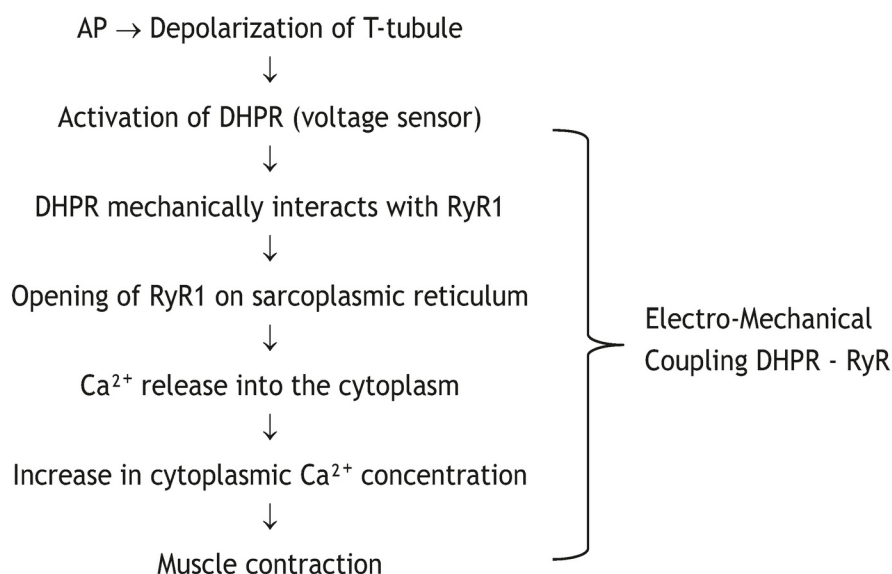
Q10.7: What does a lower chronaxie indicate?**Answer:** Greater neuronal excitability.**Q10.8: What are the typical spike (action potential) durations for A, B, and C nerve fibers?****Answer:**

- C fiber - 2 ms
- B fiber - 1.2 ms
- A fiber - 0.4 to 0.5 ms

BIG QUESTION 11: What is the structure and excitation-contraction coupling pathway in skeletal vs cardiac muscle?**Broad Answer:**

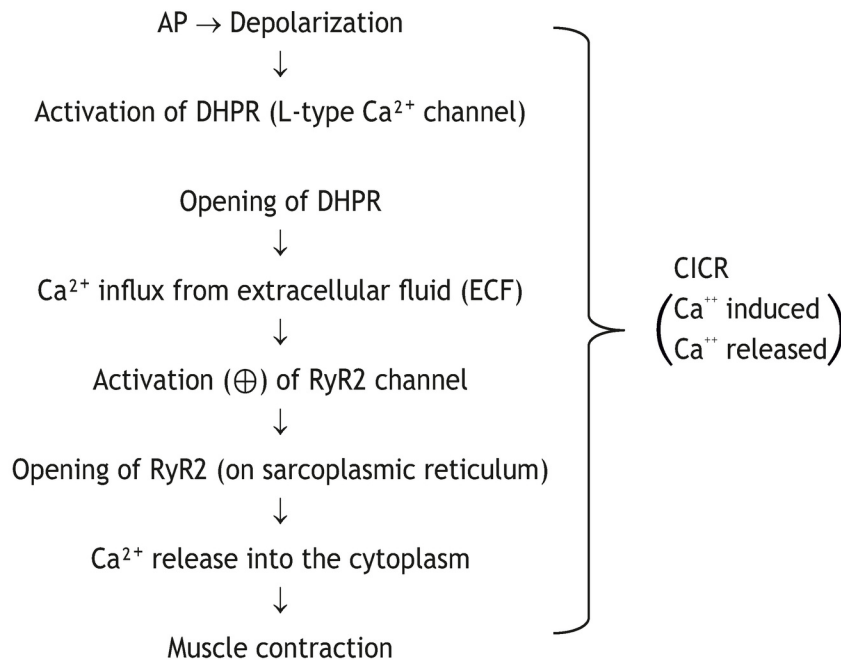
- **Skeletal muscle:** Ca^{2+} comes from SR only, uses **DHP-RyR1 coupling** via mechanical linkage.
- **Cardiac muscle:** Ca^{2+} enters from extracellular fluid via **L-type Ca^{2+} channels**, then triggers **Ca^{2+} -induced Ca^{2+} release** via **RyR2** from SR.

PYQ: INICET 2022

**DETAILED QUESTIONS****Q11.1: What are the components of skeletal muscle triads?****Answer:** 1 T-tubule + 2 Terminal cisternae of SR**Q11.2: What are the excitation-contraction events in skeletal muscle?****Answer:**

Q11.3: What are the excitation-contraction events in cardiac muscle?

Answer:



Q11.4: What is the source of Ca^{2+} in skeletal and cardiac muscle?

Answer:

Muscle Type	Ca^{2+} Source
Skeletal	Sarcoplasmic Reticulum only (Cisternal Calcium)
Cardiac	ECF and SR

Q11.5: What are the clinical implications of ryanodine receptor mutations?

Answer:

- **RyR1 mutations** → Malignant Hyperthermia
- **RyR2 mutations** → Ventricular arrhythmias, CPVT
- Mechanism: Abnormal Ca^{2+} release from SR, causing hypermetabolism or arrhythmias.

Q11.6: What condition is caused by RyR1 mutation?

Answer: Malignant Hyperthermia (triggered by anesthetics like halothane, succinylcholine)

Q11.7: What condition is caused by RyR2 mutation?

Answer: CPVT (Catecholaminergic Polymorphic Ventricular Tachycardia)

Q11.8: What is the biochemical abnormality in malignant hyperthermia?

Answer: Uncontrolled Ca^{2+} release from SR → ↑ATP consumption → Heat generation.

BIG QUESTION 12: What is the molecular mechanism of muscle contraction?

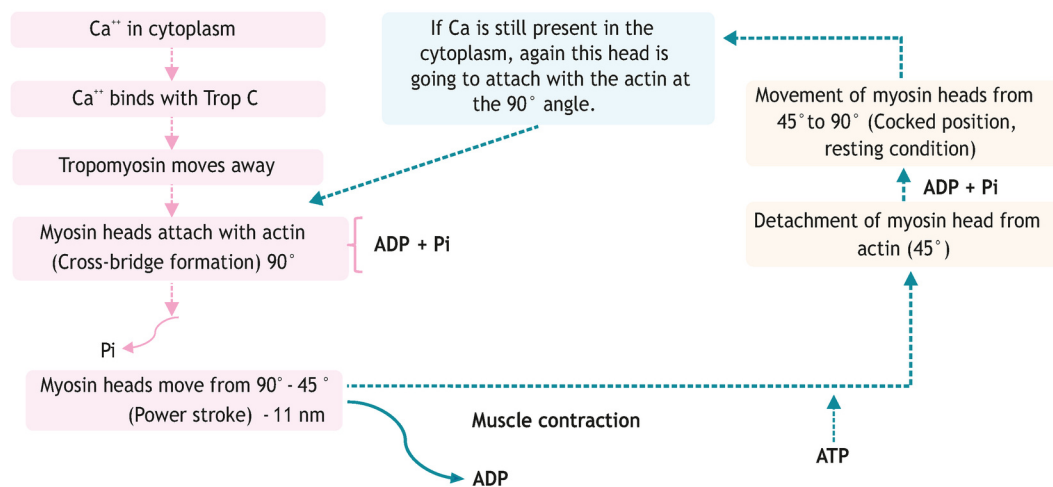
Broad Answer: Muscle contraction follows a **cyclical interaction** between actin and myosin regulated by **calcium** and **ATP**. The **Cross-Bridge Cycle** involves 5 key steps:

1. **Attachment** of myosin head to actin

2. Power stroke
3. Detachment via ATP binding
4. Reactivation via ATP hydrolysis
5. Return to original state ready for next cycle

This cycle continues as long as Ca^{2+} and ATP are available.

PYQ: AIIMS 2019



DETAILED QUESTIONS

Q12.1: What initiates the cross-bridge cycle in skeletal muscle?

Answer: Binding of Ca^{2+} to Troponin-C exposes actin's myosin-binding sites.

Q12.2: What are the 5 steps of the cross-bridge cycle?

Answer:

1. **Attachment:** Myosin head (with $\text{ADP} + \text{Pi}$) binds actin
2. **Power Stroke:** Pi is released $\rightarrow$ myosin bends $\rightarrow$ actin slides
3. **Detachment:** ATP binds myosin $\rightarrow$ myosin detaches
4. **Hydrolysis:** $\text{ATP} \rightarrow \text{ADP} + \text{Pi} \rightarrow$ myosin head resets
5. **Ready State:** Myosin head rebinds actin

Q12.3: What role does ATP play in muscle contraction?

Answer:

- Causes **detachment** of myosin from actin
- **Hydrolysis** reactivates myosin head for the next cycle

Q12.4: What happens in absence of ATP (e.g., after death)?

Answer: Myosin remains bound to actin $\rightarrow$ **Rigor Mortis**

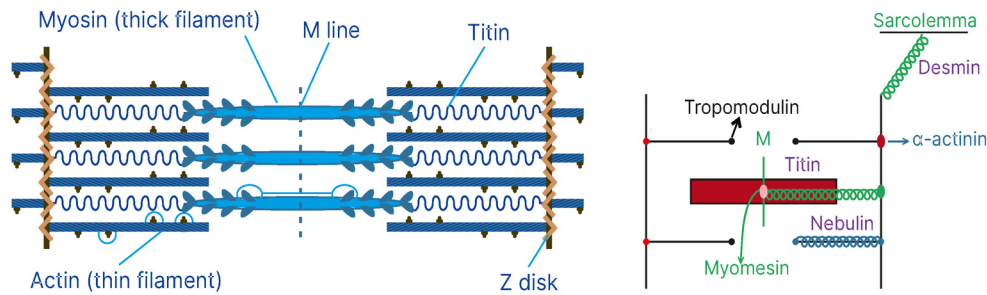
BIG QUESTION 13: What is the structure and activation of the sarcomere during contraction?

Broad Answer:

The **sarcomere** is the contractile unit of muscle and changes structure during contraction.

- H-zone and I-band shorten
- A-band remains constant
- Actin filaments slide over myosin $\rightarrow$ muscle shorten

PYQ: FMGE 2019 PYQ: INICET 2020 PYQ: NEET PG 2021, 2022



DETAILED QUESTIONS

Q13.1: What is the structure of a sarcomere?

Answer:

Z-line to Z-line, includes:

- A-band (dark)
- I-band (light)
- H-zone
- M-line

Q13.2: What band remains constant during contraction?

Answer: A-band

Q13.3: Which zones shorten during contraction?

Answer:

- I-band
- H-zone

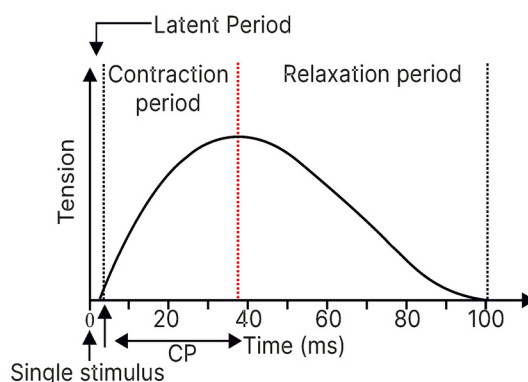
Q13.4: What are the key anchoring proteins of the sarcomere and their associated properties or clinical relevance?

Answer:

Anchoring Proteins	Properties
Alpha Actinin / Cap Z protein	• Z-line protein • Attaches actin with Z-line • Mutation causes Nemaline myopathy
Nebulin	• Attaches actin tip to Z-line (regulates actin length) • Mutation → Nemaline myopathy (more common than alpha-actinin mutation) • Affects axial + facial muscles
Titin	• Connects M-line to Z-line • Forms elastic-like molecules • Mutation causes Dilated cardiomyopathy, HCM

BIG QUESTION 14: What are the properties of different types of muscle twitch responses?

Broad Answer: Muscle twitch properties vary by muscle fiber type and stimulation frequency. A single AP causes a **simple twitch**, while repeated stimuli can produce **incomplete** or **complete tetanus**.



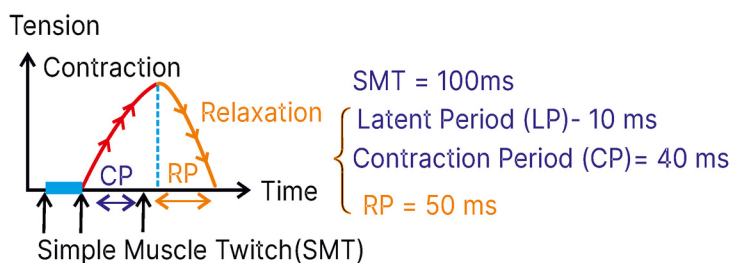
DETAILED QUESTIONS

Q14.1: What are the properties of different muscle fiber types?

Muscle Type	Property
Type I (Slow, Red)	High endurance, low power
Type II A (Fast, Red)	Moderate endurance, moderate power
Type II B (Fast, White)	Low endurance, high power

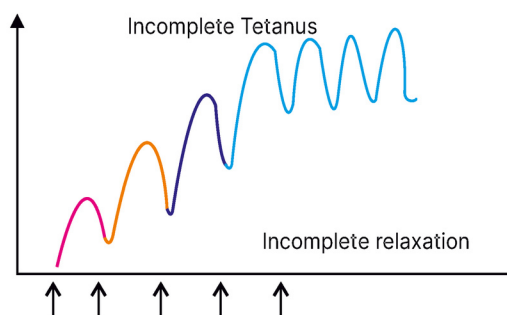
Q14.2: What is a simple twitch?

Answer: A single AP leads to contraction-relaxation cycle → one twitch response.



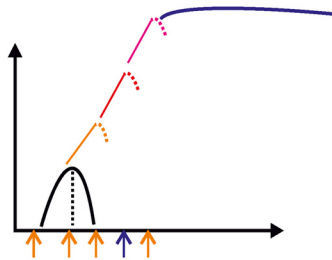
Q14.3: What is incomplete tetanus?

Answer: Repeated stimuli applied **before complete relaxation**, causing **summation** of contractions.



Q14.4: What is complete tetanus?

Answer: Stimuli applied at **high frequency** → no relaxation → **sustained contraction**.



Q14.5: What is the latent period and how long is it?

Answer: Time between stimulus and start of contraction: ~2 msec

Q14.6: A muscle contraction from a single stimulus is recorded in the diagram (tension vs time). Calculate the contraction time.

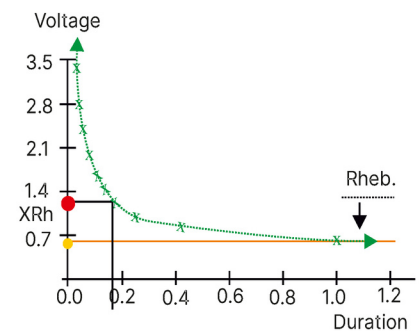
Answer:

$\frac{2}{3} \times \text{Total duration}$

Contraction time = $\frac{2}{3}$ of the total duration (from start of contraction to peak tension)

Q14.7: Find out the chronaxie and rheobase from the graph given:

- A. Chronaxie 0.15ms, rheobase 0.6V
- B. Chronaxie 0.6ms, rheobase 0.15V
- C. Chronaxie 0.6ms, rheobase 0.13V
- D. Chronaxie 0.1ms, rheobase 0.6V



Answer: A. Chronaxie 0.15ms, rheobase 0.6V

- **Rheobase:** Minimum voltage that elicits a response at long durations
- **Chronaxie:** Duration corresponding to **twice the rheobase** voltage on the curve.

Q14.8: What diseases are associated with mutations in the ryanodine receptor type 1 (RYR1) gene?

Answer:

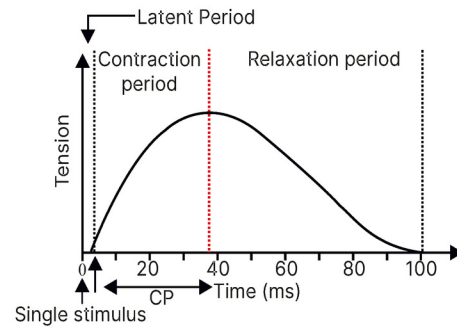
Ryanodine Receptor Type 1 Mutation	
Mutation	Diseases
Gain of RyR1 Mutation	Malignant Hyperthermia • Muscle rigidity, high fever, and rapid heart rate. • Rapid breakdown of skeletal muscle (rhabdomyolysis) and a high plasma K^+ level. • Tx: Dantrolene, a drug that antagonizes ryanodine receptors.
Loss of RyR1 Mutation	Central core disease • Hypotonia (decreased muscle tone) at birth, mild delay in child development, weakness of the facial muscles. • No specific treatment.

Q14.9: The graph below shows a simple muscle twitch recorded in the frog gastrocnemius muscle. Calculate the tetanizing frequency of this muscle.

- A. 25HZ
- B. 20HZ
- C. 10HZ
- D. 5HZ

Answer: A. 25HZ

$$TF = 1/CP = 1000/40 = 25.$$





3. CNS PHYSIOLOGY

BIG QUESTION 1: What are the main cellular components of the central nervous system? What is its significance?

Broad Answer: The central nervous system is mainly composed of neurons and glial cells. Neurons are responsible for transmitting signals, while glial cells support and protect the neurons

DETAILED QUESTIONS

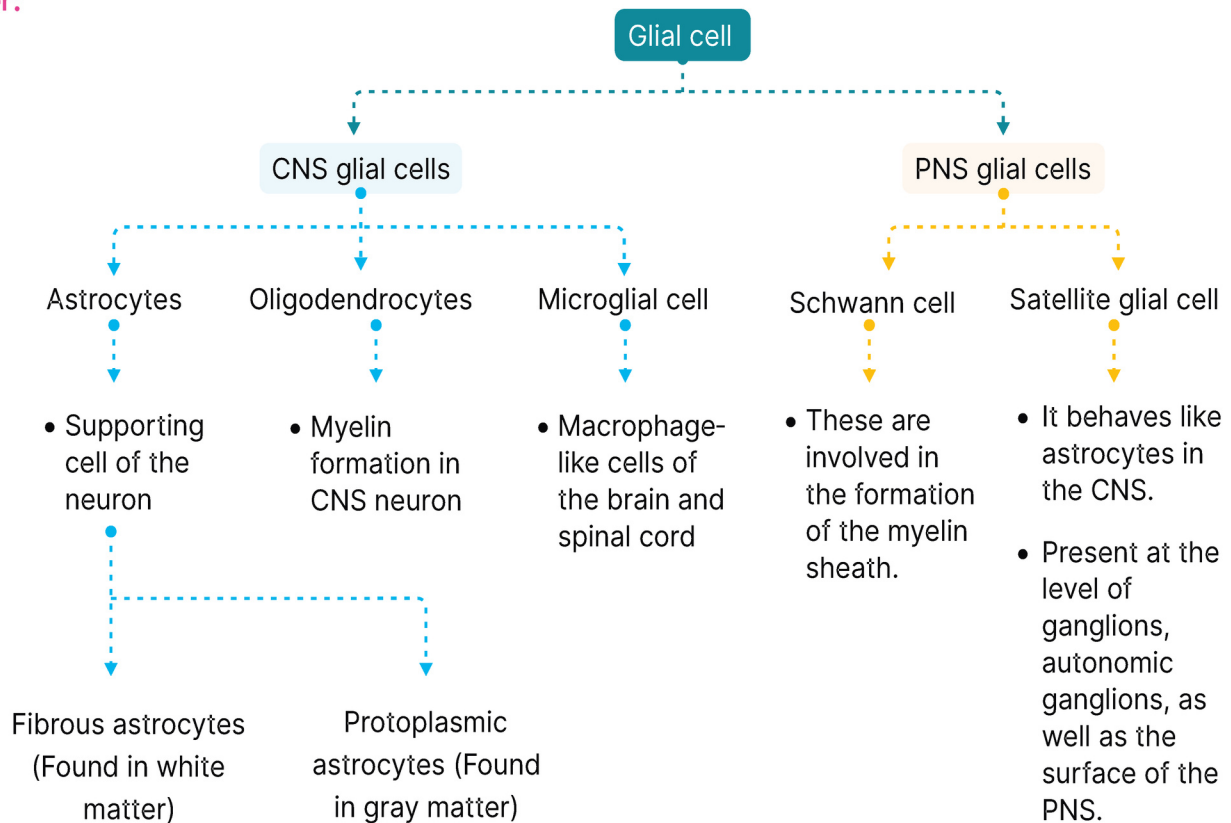
Q1.1: How many neurons and glial cells are in the human CNS?

Answer:

- Neurons: 100 billion.
- Glial cells: 2 to 10 times the neuronal number.

Q1.2: What are the types of Glial cells?

Answer:



Q1.3: What are the functions of Astrocytes?

Answer:

- Secretes various growth factors for neurons.
- Provide nutrition to neurons (connected to neurons at one end and blood vessels on the other end).

- Control the level of glutamate/ K^+ in the intracellular fluid (ICF) of the brain.
- Regulates the Blood Brain Barrier (BBB).

Q1.4: What is multiple sclerosis? Which cells are affected by MS?

Answer:

- Multiple sclerosis is an autoimmune disease caused by the invasion of T-cells into oligodendrocytes in the CNS, causing demyelination of the neurons.
- Demyelination impairs nerve conduction.
- Antibodies against **myelin basic protein** and other components, such as **myelin oligodendrocyte glycoprotein** (MOG), are responsible for demyelination.

BIG QUESTION 2: What are the events that occur during the transmission of a nerve impulse across a chemical synapse?

Broad Answer: Synaptic transmission is the process by which signals are transferred between neurons at a chemical synapse. The process is unidirectional and involves neurotransmitters crossing the synaptic cleft. It plays a key role in modulating and propagating nerve impulses.

DETAILED QUESTIONS

Q2.1: What are the two phases of synaptic transmission?

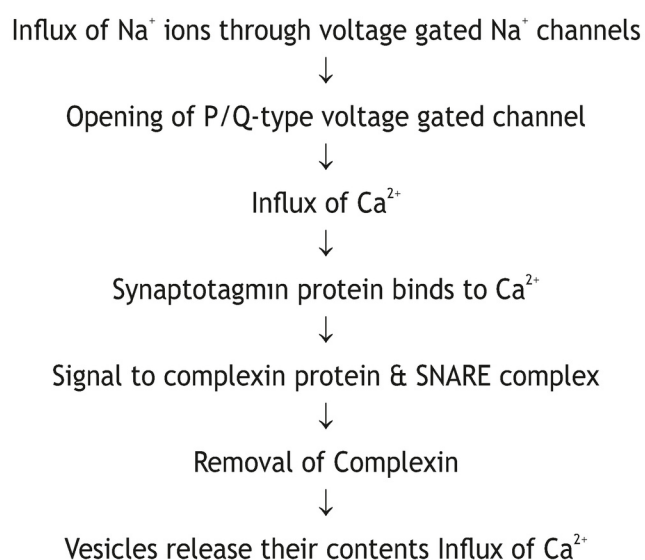
Answer:

Synaptic transmission occurs in two phases:

- Pre-synaptic transmission (neurotransmitter release)
- Post-synaptic transmission (response generation).

Q2.2: What is the sequence of events that occurs in presynaptic transmission?

Answer:

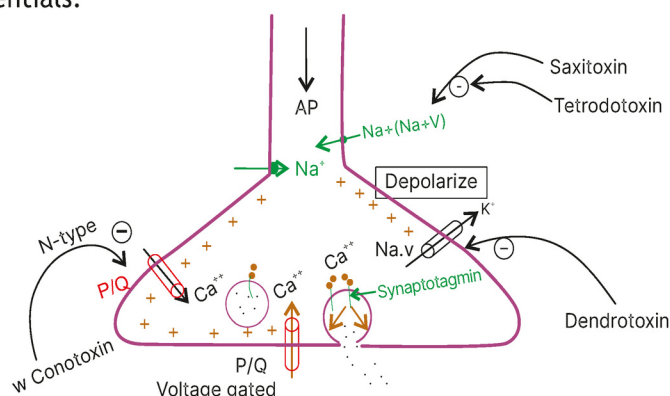


Q2.3: What does the diagram show about toxins acting on presynaptic ion channels?

Answer:

- Depolarization can also occur by blocking the potassium channels present at the level of the presynaptic membrane → Accumulation of positive charges inside the cell membrane.

- Dendrotoxin: Block potassium channels.
- ω -conotoxin: Blocking of the presynaptic calcium channels (mainly N-type $\text{CaV}2.2$).
- Saxitoxin and tetrodotoxin: Block voltage-gated sodium channel $\rightarrow$ Inhibit the entry of sodium ions $\rightarrow$ Prevent the generation of action potentials.



Q2.4: How does blocking presynaptic potassium channels lead to depolarization?

Answer:

- Normally, depolarization happens when an action potential opens voltage-gated sodium channels.
- But, depolarization can also occur by blocking the potassium channels present at the level of the presynaptic membrane.
- This results in the accumulation of positive charges inside the cell membrane, thereby enhancing depolarization.

Q2.5: Which toxin blocks the presynaptic potassium channels?

Answer: Presynaptic potassium channels are blocked by **dendrotoxin**.

Q2.6: Which toxin blocks the presynaptic calcium channels?

Answer: Presynaptic calcium channels (mainly N-type $\text{CaV}2.2$) are blocked by **ω -conotoxin**.

Q2.7: Which toxins block voltage-gated sodium channels, and what is the result of this blockade?

Answer: **Saxitoxin** and **tetrodotoxin** block voltage-gated sodium channels, preventing sodium entry and inhibiting the generation of action potentials.

Q2.8: What are the types of post-synaptic transmission?

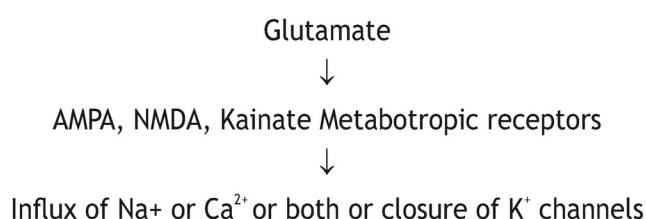
Answer:

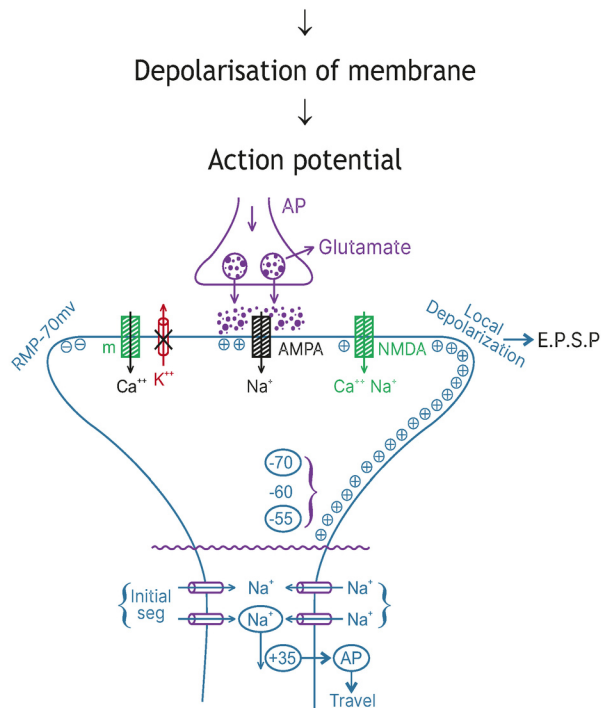
Post-synaptic transmission is of 2 types:

- Excitatory synapse
- Inhibitory synapse

Q2.9: What is the sequence of events that occurs in excitatory post-synaptic transmission?

Answer: The post-synaptic membrane has receptors for glutamate (metabotropic or ionotropic types like AMPA, NMDA, and Kainate).





Q2. 10: What is the characteristic of depolarisation in post-synaptic transmission?

Answer: Depolarisation in post-synaptic transmission is a **local depolarisation** and travels only when the threshold is reached.

Q2. 11: What are the properties of a local potential?

Answer:

- Non-propagated potential
- Decremental potential
- Does not follow the all-or-none law.
- The local potential is also known as graded potential and non-propagative.

Q2. 12: What is the threshold voltage of a neuron, and how is it reached?

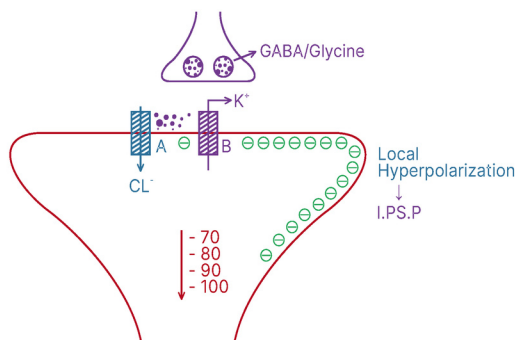
Answer: -55 mV: More glutamate release larger amplitude depolarization higher chance of reaching the threshold.

Q2. 13: What is EPSP?

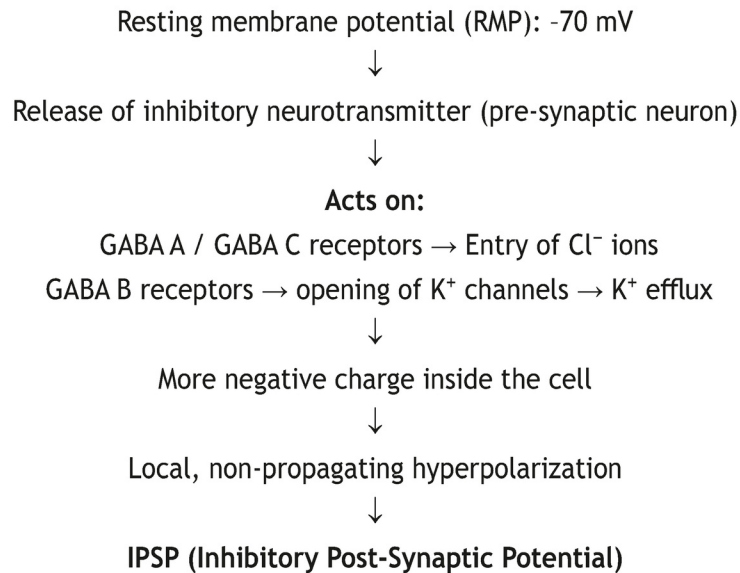
Answer: The excitatory potential generated at the post-synaptic membrane is called EPSP (Excitatory Post-Synaptic Potential)

Q2. 14: What happens to the postsynaptic membrane potential at an inhibitory synapse?

Answer:



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BIG QUESTION 3: What are Somatosensory receptors, and what are their Characteristic features?

Broad Answer: Somatosensory receptors are specialized sensory nerve endings that detect touch, pressure, temperature, pain, and body position.

They include mechanoreceptors, thermoreceptors, nociceptors, and proprioceptors.

These receptors are widely distributed in the skin, muscles, joints, and viscera.

DETAILED QUESTIONS

Q3.1: How are Somatosensory receptors classified?

Answer:

Somatosensory Receptor	Characteristics
Mechanoreceptors	Activated by stretch or tension (mechanical deformation)
Thermoreceptors	Activated by temperature (hot or cold)
Photoreceptors (Electromagnetic receptors)	- Photoreceptors are rod cells and cone cells. - Detect light sensation
Chemoreceptors	Activated by chemical stimuli (smell and taste)
Polymodal Receptors	- Multiple modalities of stimulus - Examples of this polymodal receptor are the nociceptors or pain receptors

Q3.2: What are the types of mechanoreceptors?

Answer:

Mechanoreceptors are subdivided into:

- **Tactile receptor:** Detects touch and pressure.
- **Proprioceptors:** Give sensation about the position of joints in three-dimensional space.
 - o The most important proprioceptor is the muscle spindle.
 - o Golgi tendon organ.
 - o Joint capsule receptor.

- **Nociceptors:** They can be activated by a mechanical stimulus, a thermal stimulus, and also a chemical stimulus.
- **Hair Cells:** They are present at the level of the Organ of Corti

Q3.3: What are the types of Thermoreceptors?

Answer:

Thermoreceptors are of 2 types:

- **Cold receptors:** Activated when the temperature is from 10°C to 40°C. Maximum activity of this cold receptor occurs at 24°C.
- **Warm receptors:** Activated when the temperature is from 30°C to 49°C. Maximum activity of this cold receptor occurs at 45°C

Q3.4: What is the relationship between thermal receptors and nociceptors?

Answer:

- If the temperature is beyond the range of <10°C and >50°C, the thermal receptor will activate the nociceptors.
- Thermal receptors and pain receptors have similar properties.

BIG QUESTION 4: What are Nociceptors, and what role do they play in the sensation of pain?

Broad Answer: Nociceptors are specialized sensory receptors that detect pain. When activated, they generate nerve impulses that are transmitted to the central nervous system, leading to the perception of pain.

They play a crucial protective role by alerting the body to potential injury.

DETAILED QUESTIONS

Q4.1: Where are nociceptors located?

Answer:

- Nociceptors are located on free nerve endings.
- These free nerve endings contain specialized ion channels on their membranes that detect noxious stimuli.

Q4.2: What are the various channels and receptors at free nerve endings?

Answer:

Nociceptor (Free Nerve Ending)			
Mechanical	Thermal		Chemical
• TRPA1 • MRGPR-D	Cold	Hot	• Bradykinin (B1&B2) Serotonin (5HT3) • Acid (ASIC) • NGF (TrKA)
	• TRPA1 • TRPM	• TRPV1 • TRPV2 • TRPV3/V4	

Q4.3: Which receptors are involved in mechanical nociception?

Answer:

- Transient receptor potential ankyrin 1 (TRPA1)
- Mas Related G Protein-Coupled Receptor-D (MRGPCR-D). It was first discovered in Mast cells.

Q4.4: What are the types of thermal nociceptors?

Answer:

Thermal pain receptors are of 2 types.

- Cold receptors (Cold detection)
- Warm receptors (Heat detection)

Q4.5: Which receptors detect cold, and what are their temperature sensitivities?

Answer:

Cold Receptors:

- **TRPA1**
 - o It is sensitive when the temperature is $< 17^{\circ}\text{C}$.
 - o TRPA1 is stimulated by multiple stimuli, i.e., thermal and mechanical, thus it is a polymodal receptor.
- **Transient Receptor Potential Melastatin (TRPM)**
 - o It is sensitive when the temperature is moderate, between $8-28^{\circ}\text{C}$.

Q4.6: What are the types of warm receptors?

Answer:

Warm Receptors:

- **TRP Vanilloid 1 (TRPV1)**
 - o Activated when temperature $> 43^{\circ}\text{C}$, H^{+} ions and capsaicin (the active ingredient of chili).
- **TRPV2**
 - o It is activated when the temperature is $> 53^{\circ}\text{C}$.
- **TRPV3/V4**
 - o They are activated when skin temperature is normal, i.e., around 30°C

Q4.7: What are the nociceptors stimulated by chemicals?

Answer:

Nociceptors stimulated by chemicals are:

- **Bradykinin:** It is the most painful chemical stimulus and acts on B1 & B2 receptors.
- **Serotonin:** It produces pain by acting on 5-HT₃ receptors.
- **Histamine:** It can produce neuropathic pain by acting on H₃/H₄ receptors.
- **H^{+} ions:** Acid-Sensing Ion Channel (ASIC).
- **Nerve Growth Factor (NGF)**
 - o They generally act on Tyrosine Kinase A (TrKA).

Important Information

- **TRPV1 detects acids, and chemicals along with temperature.**
- Nociceptors are polymodal because the channels responsible for pain detection can detect other modes of stimulation.

Q4.8: Which substances directly activate nociceptors, and which ones act as sensitizers to increase their sensitivity?

Answer:

Pain-producing substances (nociceptor activators) and substances that increase the sensitivity of nociceptor neurons (nociceptor sensitizers)		
Substance	Source	Effect on primary afferent
Nociceptor activators		
Bradykinin	Plasma protein	+++
Histamine	Mast cell	+
Serotonin	Platelets	++
K ⁺ (Potassium)	Damaged cell	++
Nociceptor sensitizers		
Prostaglandins	Damaged cell	+/-
Leukotrienes	Damaged cell	+/-
Substance P	Primary afferent	+/-

Q4.9: What is Hyperalgesia?

Answer:

Hyperalgesia is an exaggerated response to a noxious/nociceptive stimulus, i.e., stimuli that are bound to cause pain.

Q4.10: What is Allodynia?

Answer:

- Allodynia is a sensation of pain in response to a normally innocuous stimulus, i.e., a non-pain-producing stimulus.
- E.g., lukewarm water might feel like burning water.

Q4.11: What are the pain-insensitive structures in the brain?

Answer:

- Brain parenchyma
- Ependyma
- Choroid plexus
- Pia mater
- Arachnoid

Q4.12: What is the Gate control theory of pain?

Answer:

- Explains the modulation of pain at the level of the spinal cord
- Proposed by Melzack and Wall
- Gate = Synapse between 1st and 2nd-order neurons in the spinal cord

Q4.13: What is the basic pathway of the Gate control theory of pain?

Answer:

- 1st-order neurons from A δ and C fibers (pain fibers) terminate at the dorsal horn of the spinal cord (Lamina II - Substantia Gelatinosa)

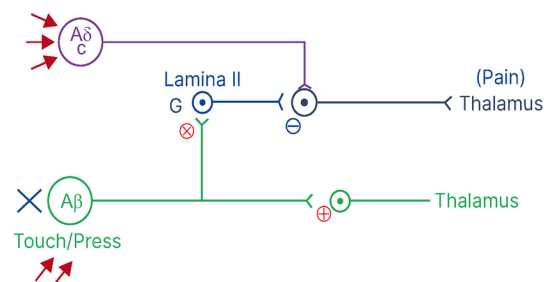
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- They synapse with 2nd order neurons, which project to the thalamus
- In Lamina II, there are inhibitory interneurons called Gelatinosa neurons (G neurons)

Q4.14: How is pain transmitted (without touch)?

Answer:

Pain stimulus (A δ /C fibers)
 ↓
 Terminates at Lamina II (Substantia Gelatinosa)
 ↓
 Activates 2nd-order neuron → Signal travels to thalamus.
 ↓
The gate is Open → Pain is perceived.



Q4.15: What is the role of A β sensory fiber in pain modulation?

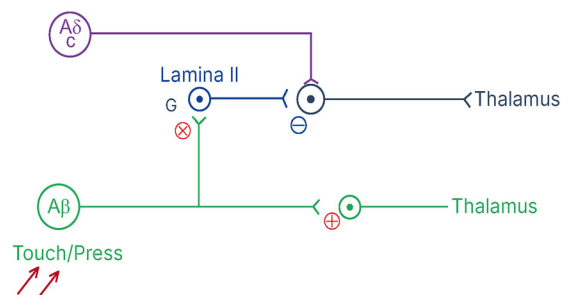
Answer:

- A β fibers carry non-noxious stimuli (e.g., touch, pressure)
- They also terminate in Lamina II.
- When activated results in reduced pain perception

Q4.16: How is pain modulated via touch/ pressure?

Answer:

Touch/pressure stimulus (A β fibers)
 ↓
 Also terminates at Lamina II.
 ↓
 Activates G (Gelatinosa) inhibitory interneuron
 ↓
 Inhibits the 2nd order pain neuron
 ↓
The gate is Closed → Pain perception is reduced.



Q4.17: What is TENS?

Answer: TENS (Transcutaneous Electrical Nerve Stimulation) uses low-frequency vibration/touch to stimulate A β fibers → closes the gate → reduces pain.

BIG QUESTION 5: What are tactile receptors, and how are they classified?

Broad Answer: Tactile receptors are specialized sensory nerve endings in the skin that detect touch, pressure, vibration, and texture. They include mechanoreceptors like Meissner's corpuscles, Merkel discs, Ruffini endings, and Pacinian corpuscles, each adapted to different types of mechanical stimuli. These receptors play a critical role in somatosensory perception and help relay information to the central nervous system for fine touch and proprioception.

DETAILED QUESTIONS

Q5.1: Which part of the body has the highest concentration of tactile receptors?

Answer:

The highest density of tactile receptors is found at the fingertips, followed by the lips.

Q5.2: How are tactile receptors classified?

Answer:

Tactile Receptors	
Rapidly adapting receptor (RA)/ Phasic receptors	Slow-adapting receptor (SA)/ Tonic receptors
• Pacinian Corpuscles • Meissner's Corpuscles • Hair-end organ receptors	• Merkel's Disc • Ruffini's Ending • Free Nerve Ending

Q5.3: What are the characteristics of Pacinian corpuscles?

Answer:

- Largest Tactile receptor
- Most sensitive receptor
- Stimulus:
 - o High-frequency vibration detection of 200 Hz or more.
 - o Deep pressure.

Q5.4: What are Meissner's corpuscles, and what are their features?

Answer:

- The most numerous touch receptors in our body are.
- The best stimulus is moving touch.
- Also, the stimulus for vibration detection with a frequency lower than 50 Hz.
- Located only in non-hairy skin, also known as Glabrous skin.

Q5.5: What are the features of Hair-end organ receptors?

Answer:

- Only present in hairy skin.
- Stimulus: Something moving on hairy skin
- The first touch of anything on the body is detected by the Hair-end Organ.

Q5.6: Where is Merkel's disc located, and what are its functions?

Answer:

- They are located at the level of the epidermis
- The best stimulus is any sharp edge or corner point stimulus.
- Merkel's Disc is the best receptor for the detection of Two-point discrimination, also known as Tactile acuity.

Q5.7: Which type of touch receptors are most effective for tasks requiring fine spatial discrimination, like reading Braille?

Answer: Merkel's discs are more effective than Meissner's corpuscles for reading Braille because they provide detailed spatial resolution.

Q5.8: What are the features of Ruffini's ending?

Answer:

- The size and shape of hand-held objects are best detected by Ruffini's ending.
- The best stimulus is a stretch on the skin by a large object.

Q5.9: What is the best stimulus for a free nerve ending?

Answer: The best stimulus for free nerve endings is an itching and tickling kind of sensation.

BIG QUESTION 6: What are Proprioceptors? What are their features and significance?

Broad Answer: Proprioceptors are specialized sensory receptors that detect the position, movement, and stretch of muscles, tendons, and joints. They include muscle spindles, Golgi tendon organs, and joint receptors. These receptors provide the brain with continuous information about body posture and limb position. They give a sense of the position of joints in 3-D space.

Their function is essential for coordinated movement, balance, and maintaining posture.

DETAILED QUESTIONS

Q6.1: What are the types of Proprioceptors?

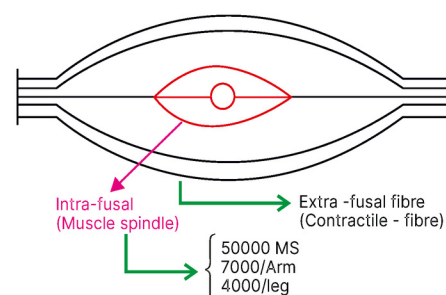
Answer:

- Muscle Spindle
- Golgi Tendon Organ (GTO)

Q6.2: What is the structure of the Muscle spindle receptor?

Answer:

- Present inside the skeletal muscle.
- Spindle-shaped.
- It detects the muscle's length and velocity to sense the joint's position.
- The muscle spindle is composed of a slightly modified skeletal muscle fiber known as an **intrafusal fiber**. It is the non-contractile fiber.
- The white fiber around it is known as the **extrafusal fiber** of skeletal muscle. It is the contractile fiber.



Q6.3: What type of nerve fibers supply the intrafusal fibers of muscle spindles?

Answer: Aγ (gamma) efferent nerve fibers.

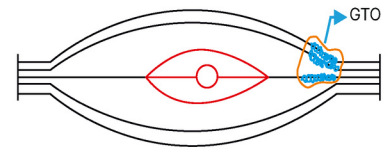
Q6.4: What type of nerve fibers supply the extrafusal fibers of muscle spindles?

Answer: Aα motor neuron (efferent supply).

Q6.5: What is the structure of the Golgi tendon organ?

Answer:

- Golgi Tendon Organ Receptor (GTO) is located at the junction of the muscle and the tendon
- 3-25 fibres (10-15)
- Stimulated by increased tension in the tendon.

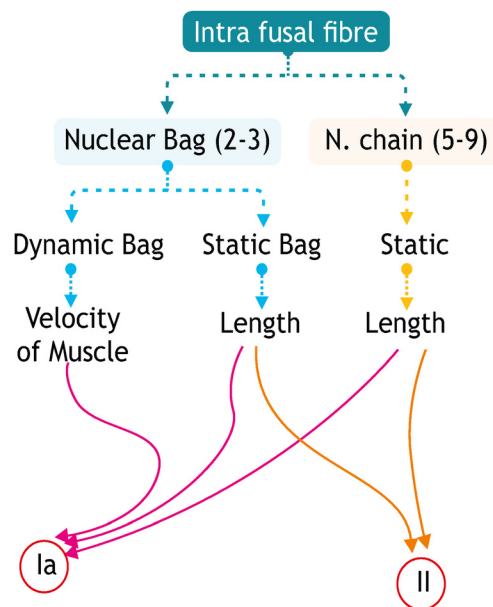


Q6.6: How are Intrafusal fibers classified?

Answer:

Intrafusal fibers are of two types:

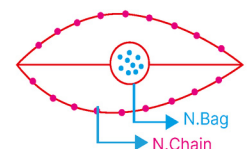
- Nuclear Bag fiber
- Nuclear chain fiber



Q6.7: What is the structure of intrafusal fibers?

Answer:

- Intrafusal fibers are specialized muscle fibers found inside the muscle spindle, surrounded by a connective tissue capsule.
- They include **nuclear bag fibers** and **nuclear chain fibers**.



The above diagram depicts a single muscle spindle (Intrafusal fiber)

Q6.8: What are the characteristics of nuclear bag fibers?

Answer:.

- Fiber contains multiple nuclei together inside a bag-like region.
 - Dynamic nuclear bag fiber: It detects the real-time shortening/lengthening of a muscle, i.e., the velocity of a muscle (length/time).
 - Static nuclear bag fiber: They only detect the increased length of a muscle.

Q6.9: What are the features of Nuclear Chain fibers?

Answer:.

- If these nuclei are spread throughout the muscle at regular intervals, it is known as the nuclear chain fiber.
- They are always static.
- They detect the increased length of a muscle.

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Q6.10: What are the afferent connections of the muscle spindle?

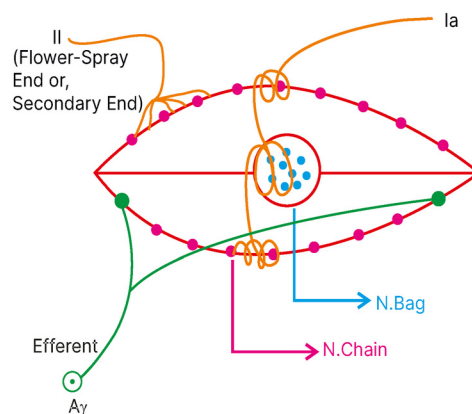
Answer:.

Characteristic	Ia Fiber	II Fiber
Origin	The center region of the spindle	The peripheral region of the spindle
Synapse Type	- Its synapse is in a spiral connection. - Annulo-Spiral Ending (Primary Afferent/ Ending)	- The nerve fiber spreads with the muscle spindle. - Flower-spray Ending (Secondary Ending)
Connections	Connects to dynamic bag fiber, static bag fiber, and static chain fiber, providing an afferent supply to all	Connects to the static bag fiber and the static chain fiber
Function	Detects the velocity and length of the muscle	Detects increased length of the muscle (does not detect velocity)

Q6.11: Describe the afferent and efferent connections of the muscle spindle.

Answer:.

The efferent connection of the muscle spindle originates from an A γ neuron.



BIG QUESTION 7: What is the stretch reflex, and what is its physiological significance?

Broad Answer:

The stretch reflex is a monosynaptic reflex that helps maintain muscle tone and posture by resisting sudden changes in muscle length. This causes the same muscle to contract, restoring its original length. It plays a key role in posture, balance, and smooth voluntary movements.

DETAILED QUESTIONS

Q7.1: What is the neural pathway involved in the stretch reflex that leads to muscle contraction?

Answer:

The stretch reflex is also known as the Myotactic reflex.

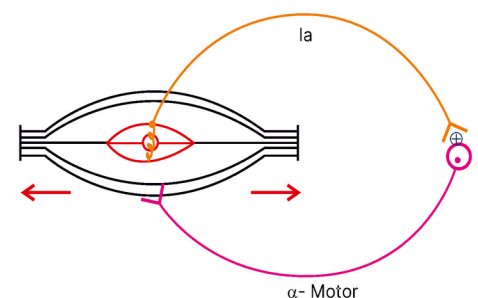
Muscle Spindle Stretch (Increase in Muscle Length)

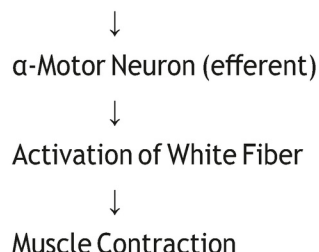


Activates Ia Fiber (Afferent Neuron)



Excitatory Neurotransmitters





Q7.2: Why is the stretch reflex described as monosynaptic?

Answer:

- It's called monosynaptic because the sensory neuron from the muscle spindle makes a direct, single synapse with the α -motor neuron in the spinal cord.
- There's no interneuron between them, so only one synapse is involved in the main reflex pathway.

Q7.3: What are the afferent and efferent for the pathway?

Answer:

- Afferent: Ia Fiber
- Efferent: α -Motor Neuron

Q7.4: What is meant by the reaction time of the stretch reflex pathway?

Answer:

The time taken for the reflex contraction to occur when a muscle is stretched is known as the reaction time of the stretch reflex pathway.

Q7.5: What is an example of a stretch reflex in the human body?

Answer:

Deep Tendon Reflexes (DTR) (Knee jerk reflex)

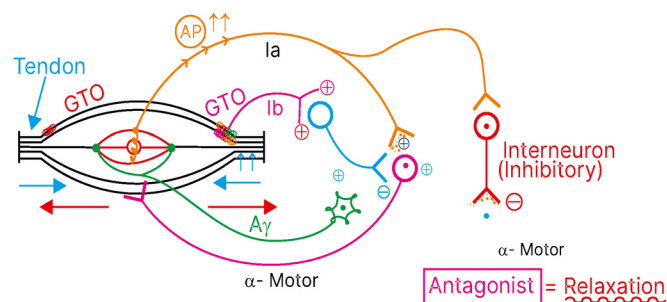
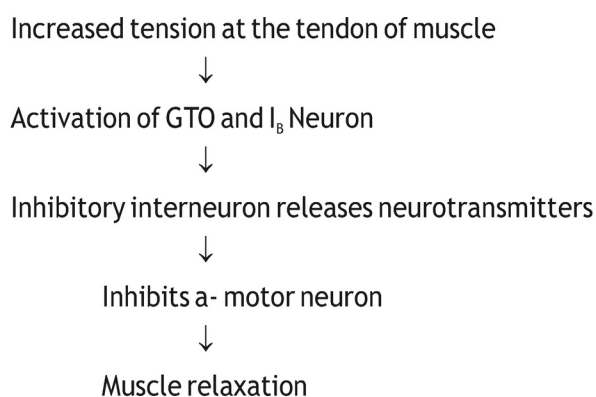
Q7.6: What is the inverse stretch reflex?

Answer:

- Also known as the Autogenic reflex.
- It is a protective reflex in which the sudden contraction of the muscle leads to reflex relaxation, preventing tendon rupture.

Q7.7: What is the pathway involved in the inverse stretch reflex?

Answer:



Q7.8: What is the receptor for the inverse stretch reflex?**Answer:**

The receptor for the inverse stretch reflex is the Golgi tendon organs (GTO)

Q7.9: What are the afferent and efferent for the reflex?**Answer:**

- Afferent: Ia Fiber
- Efferent: α -Motor Neuron

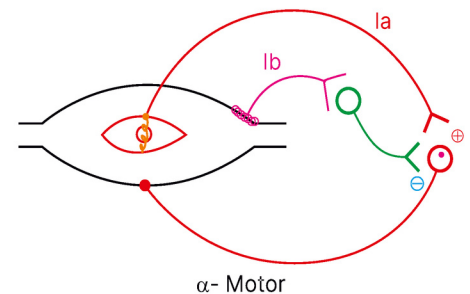
Q7.10: Why is the inverse stretch reflex described as bisynaptic?**Answer:**

The inverse stretch reflex is called a bisynaptic reflex pathway because:

- The Ib afferent fiber from the Golgi tendon organ (GTO) first synapses with an inhibitory interneuron in the spinal cord.
- This interneuron then synapses with the α -motor neuron supplying the same muscle.

Q7.11: What is reciprocal inhibition that occurs during the stretch reflex?**Answer:**

- When the muscle spindle is stretched, Ia afferent fibers are activated.
- These Ia fibers make:
 - o An excitatory synapse directly with the α -motor neuron of the same (agonist) muscle, causing it to contract.
 - o An excitatory synapse with an inhibitory interneuron, which then inhibits ($\ominus$) the α -motor neuron of the antagonist muscle, causing it to relax.
- The above diagram also shows Ib afferents (from GTO) contributing to modulation.



This coordinated mechanism ensures smooth movement by contracting the agonist muscle while simultaneously relaxing the antagonist.

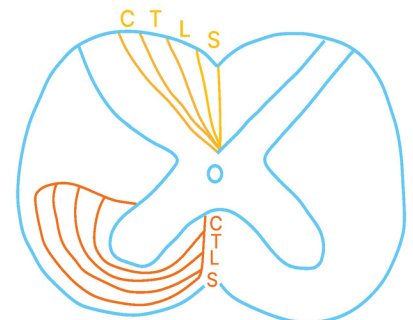
BIG QUESTION 8: What are the ascending Tracts of the Spinal tract, and what is their physiological significance?**Broad Answer:**

Ascending pathways transmit sensory signals like touch, pain, temperature, and proprioception from receptors in the body up to higher brain centers for processing.

DETAILED QUESTIONS**Q8.1: What are the types of ascending tracts?****Answer:**

Depending on location, there are different types of tracts.

- Dorsal column pathway
- Anterolateral spinothalamic pathway

**Q8.2: What are the sensations carried by the Dorsal column pathway?****Answer:**

- Localizing pressure
- Vibration & Fine touch
- Proprioception
- Two-point discrimination

Q8.3: What are the sensations carried by the Anterior Spinothalamic Tract?

Answer:

- Crude touch & pressure
- Tickle and itch
- Sexual sensations

Q8.4: What are the sensations carried by the Lateral Spinothalamic Tract?

Answer:

- Pain
- Temperature

BIG QUESTION 9: What are the descending tracts of the spinal cord? What are their features?

Broad Answer:

Descending tracts of the spinal cord carry motor commands from the brain to muscles and glands. They control voluntary movements, posture, and reflex modulation. These pathways ensure precise, coordinated actions by regulating muscle tone and reflex activity. They are essential for executing conscious and automatic motor functions.

DETAILED QUESTIONS

Q9.1: What are the types of descending tracts?

Answer:

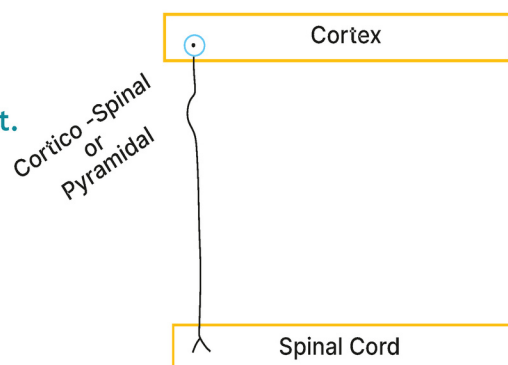
Descending tracts:

- **Pyramidal tracts**
 - o Corticospinal tract
 - Lateral
 - Anterior
 - o Corticobulbar tract
- **Extrapyramidal tracts:**
 - o Rubrospinal
 - o Vestibulospinal
 - o Reticulospinal
 - o Tectospinal

Q9.2: Describe the origin, course, and decussation of the pyramidal tract.

Answer:

- **Origin:** Motor cortex
- **Course:** Internal capsule → brainstem → medullary pyramid
- **Decussation:** The majority cross at the medulla (lateral corticospinal); some remain uncrossed (anterior corticospinal)



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Q9.3: Why is the corticospinal tract called the pyramidal tract?

Answer:

- At the medulla, the tract forms an elevated area called the medullary pyramid.
- Hence, it's termed the pyramidal tract.

Q9.4: Where does the pyramidal tract terminate, and what is its primary function?

Answer:

- **Terminates:** Anterior horn cells of the spinal cord
- **Function:** Voluntary and fine motor control, especially in distal muscles

Q9.5: Differentiate between the lateral and anterior corticospinal tracts.

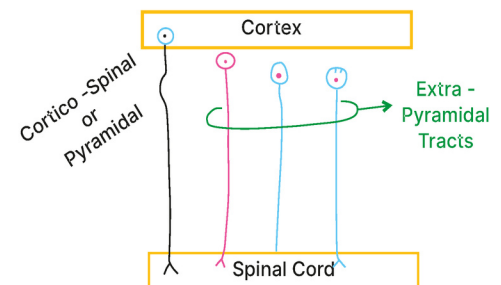
Answer:

- **Lateral corticospinal tract:** Crosses in the medulla, controls distal limb movements
- **Anterior corticospinal tract:** Remains uncrossed initially, and controls axial muscles.

Q9.6: What are extrapyramidal tracts, and where do they originate from?

Answer:

- Extrapyramidal tracts are descending motor pathways that control involuntary motor functions.
- They originate from subcortical areas like the midbrain, pons, medulla, vestibular nuclei, and basal ganglia.



Q9.7: How do extrapyramidal tracts differ from pyramidal tracts anatomically?

Answer:

- Extrapyramidal tracts do not pass through the medullary pyramids.
- Pyramidal tracts pass through the medullary pyramids, hence the name.

Q9.8: Name the main extrapyramidal tracts and their general functions.

Answer:

- Rubrospinal: Facilitates flexor muscle tone
- Vestibulospinal: Maintains balance and posture
- Reticulospinal: Modulates muscle tone and reflexes
- Tectospinal: Coordinates head and eye movements in response to visual/auditory stimuli

Q9.9: What are the functional roles of extrapyramidal tracts in motor control?

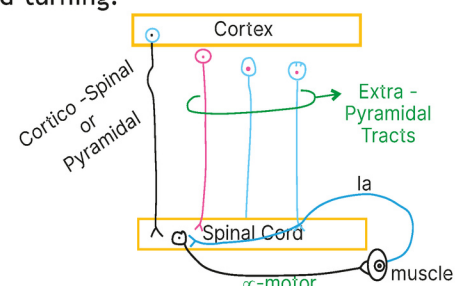
Answer:

- Regulate posture, muscle tone, reflexes, and involuntary movement patterns.
- Coordinate automatic movements like walking rhythm, arm swing, and head-turning.

Q9.10: How do pyramidal and extrapyramidal tracts functionally interact at the spinal level? Illustrate their common final pathway.

Answer:

- Both pyramidal (corticospinal) and extrapyramidal tracts descend from different origins.



- They both ultimately synapse on the α -motor neuron in the anterior horn of the spinal cord.
- The α -motor neuron then carries the signal to the skeletal muscle – this is called the final common pathway.
- This convergence allows for the coordination of voluntary (pyramidal) and involuntary/reflexive (extrapyramidal) control of movement.

BIG QUESTION 10: Describe the classification and functional differences between Upper and Lower Motor Neurons.

Broad Answer: Upper and Lower Motor Neurons are parts of the motor system that control movement.

UMNs originate in higher centers and influence spinal motor activity. LMNs directly supply muscles and carry out the actual movement. Lesions in each result in characteristic clinical differences.

DETAILED QUESTIONS

Q10.1: What is an Upper Motor Neuron (UMN), and what neural tracts are included in it?

Answer:

- Any neuron above the α -motor neuron
- Includes both the pyramidal tract (corticospinal) and extrapyramidal tracts

Q10.2: What is a Lower Motor Neuron (LMN), and why is it known as the final common pathway?

Answer:

- The α -motor neuron in the anterior horn of the spinal cord + its axon
- Called the final common pathway because it is the last output from both UMN and reflex circuits to the skeletal muscle

Q10.3: Explain the stretch reflex pathway and its link with LMN.

Answer:

- Muscle spindle stretch activates Ia afferent neuron
- It directly synapses on the α -motor neuron
- This triggers the contraction of the same muscle
- Thus, LMN is central to reflex activity and motor output

Q10.4: What is the relationship between the pyramidal/extrapyramidal tracts and the α -motor neuron?

Answer:

- Both pyramidal and extrapyramidal tracts influence the α -motor neuron
- The α -motor neuron then activates the muscle
- This illustrates that voluntary and involuntary motor control converges at LMN.

Q10.5: What are the distinguishing clinical features of UMN and LMN lesions?

Answer:

Features	Upper Motor Neuron Lesion	Lower Motor Neuron Lesion
Atrophy	None (disused atrophy)	Severe (loss of trophic factor)
Fasciculation / Fibrillation	None	Present
Tone	Hypertonia (spasticity & rigidity)	Hypotonia (Decreased)

Stretch reflex (Deep tendon reflex)	Hyperactive	Hypoactive or absent
Muscle group affected	Regional (group of muscles)	Segmental/distal (single muscle)
Babinski sign	Present	Absent

Q10.6: What are the distinguishing features between spasticity and rigidity, and how do they relate to different types of motor pathway lesions?

Answer:

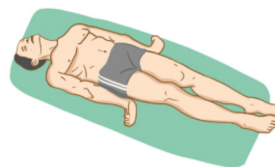
Spasticity	Rigidity
Caused by lesions in the pyramidal tract	Seen in extrapyramidal lesions
It is velocity-dependent (i.e., more noticeable with fast movements)	Velocity independent- does not vary with the speed of movement of the muscle groups involved
- Predominantly affects one group of muscles (generally antigravity muscles). - More resistance in one direction than in the other direction	Affect both groups (flexor and extensor) of muscle.
- If an upper limb is contracted because of paralysis, if we try to open the limb, more tone in the initial part of involvement, suddenly resistance will decrease, and the arm will open - "Clasp knife spasticity". - Clasp knife spasticity is due to activation of the Golgi tendon organ. - Weakness present	- Hypertonia throughout the range of motion - Lead pipe rigidity (neuroleptic malignant)- Uniform increase in tone, tension is high throughout the range of motion. - Cogwheel rigidity (Parkinson's)- Tremor superimposed on this hypertonia that results in an intermittent increase in tone during the movement. - Cogwheel rigidity = Lead pipe rigidity + tremors

Q10.7: What is decerebrate rigidity, and what is the anatomical level of lesion associated with it?

Answer:

- Caused by a lesion above the level of the vestibular nucleus but below the red nucleus (i.e., in the pons)
- It results in the extension of all four limbs
- Mediated by the pontine reticulospinal tract

Upper pontine damage
Decerebration



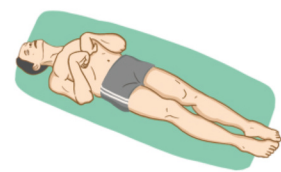
Arms adducted , extended. Wrist pronated , flexed.
Legs extended with Plantar flexion

Q10.8: What is decorticate rigidity, and how does it differ from decerebrate rigidity?

Answer:

- Caused by a lesion above the red nucleus (i.e., in the midbrain or internal capsule)
- Rubrospinal tract preserved → Flexion of upper limbs
- Pontine reticulospinal tract → extension of the lower limbs

Upper midbrain damage
Decortication



Arms adducted , flexed. Wrist and fingers are flexed.
Legs extended with Plantar flexion

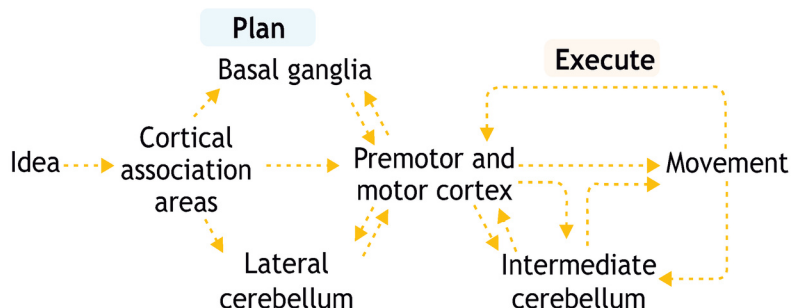
BIG QUESTION 11: Describe the role of the basal ganglia and cerebellum in motor planning and coordination.

Broad Answer: The basal ganglia and cerebellum are crucial for planning, programming, and fine-tuning movement. They work with the cortex to prepare and execute motor tasks smoothly. The cerebellum helps with coordination and timing, while the basal ganglia assists with procedural control. Together, they form feedback loops essential for skilled, purposeful movement.

DETAILED QUESTIONS

Q11.1: How do the cortex, basal ganglia, and cerebellum interact in generating and executing motor plans?

Answer:



Q11.2: Describe the cortical-basal ganglia-cerebellar loop involved in motor planning.

Answer:

- Cortical association areas generate a blueprint of the motor plan.
- This is sent to the premotor/motor cortex, basal ganglia, and lateral cerebellum.
- These structures send input back to the cortex.
- Basal ganglia + premotor/motor cortex: involved in planning & programming.
- The premotor and motor cortices execute the action
- Intermediate cerebellum: gives continuous feedback.

Q11.3: What are the functions of the cerebellum?

Answer:

Vestibulocerebellum	• Involved in vestibular reflexes. • Maintenance of balance and posture
Spinocerebellum	• It is involved in the integration of sensory input with motor commands to produce adaptive motor coordination • Coordinates & smoothens voluntary movements
Cerebrocerebellum	• It is involved in the planning and timing of movements. E.g., Learning to hit a football by a trial-and-error process (motor learning). • In addition, it is involved in the cognitive functions of the cerebellum, such as language function.

Q11.4: What are the clinical features of cerebellar disease?

Answer:

- Dysdiadochokinesia, Dysmetria (past-pointing) & Decomposition of movement
- Ataxia (drunken gait)

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- Nystagmus
- Intention tremor
- Slurred/scanning speech (skilled movement)
- Hypotonia (same side of the body)
- No sensory deficit · No abnormalities at rest (except the changes in stretch reflexes)

Q11.5: What are the functions of the Basal ganglia?

Answer:

Motor function	• The basal ganglia and motor cortex form a processing loop for planning & programming of movements.
Cognitive Function	• Basal ganglia form loops with the prefrontal association cortex and the limbic cortex. • Through these loops, the basal ganglia are thought to play a role in cognitive function. • The striatum is also involved in skill & procedural memory.

Q11.6: What are the disorders of the basal ganglia?

Answer:

Basal ganglia disorders are of 2 types:

- **Hypokinetic diseases**
 - **Parkinsonism:** Substantia Nigra (decrease in dopamine level), some hyperkinetic features.
- **Hyperkinetic diseases**
 - **Hemiballismus:** Due to the destruction of the subthalamic nucleus
 - **Huntington's disease:** Mainly due to the destruction of an indirect pathway of the striatum
 - **Dystonia:** Direct pathway, including basal ganglia, is inhibited - thalamocortical network problem

BIG QUESTION 12: Describe the physiology of sleep with reference to EEG changes, sleep stages, and polysomnographic features.

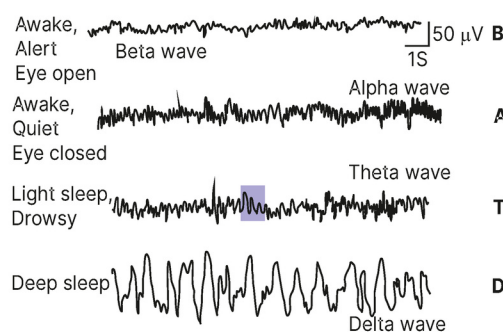
Broad Answer:

- Sleep consists of REM and NREM stages, each with distinct EEG patterns and physiological features.
- NREM sleep has three stages and shows progressively slower brain waves. REM sleep shows wake-like activity but with muscle atonia.
- EEG helps classify these stages based on wave types.
- Polysomnography is the gold standard for studying sleep disorders using multiple parameters.

DETAILED QUESTIONS

Q12.1: What are the EEG wave patterns seen during different states of consciousness?

Answer:



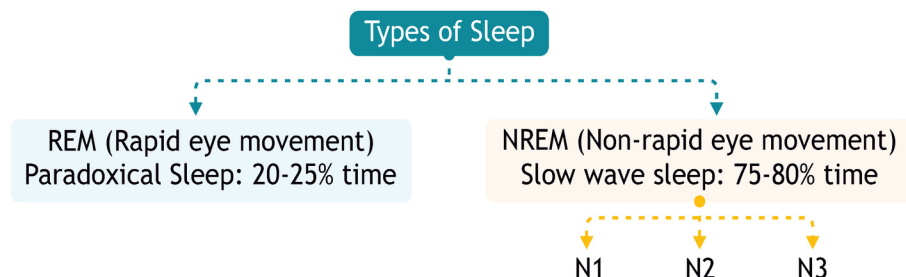
Q12.2: What are gamma waves, and when are they seen?

Answer:

- Gamma waves occur during:
 - Problem-solving
 - Meditation

Q12.3: What are the types of sleep?

Answer:



Q12.4: What are the stages of NREM sleep and their characteristics?

Answer:

Recent stage	Percentage	Features
N1	5%	α to θ wave
N2	55%	Sleep spindles, K complexes
N3	15%	δ wave

Q12.5: What is Polysomnography?

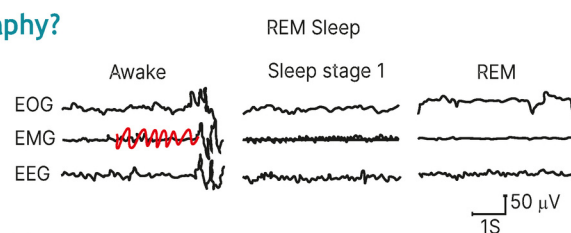
Answer:

- Polysomnography is the study of Sleep
- It consists of multiple recordings that are done simultaneously to identify the stage of sleep.

Q12.6: What are the parameters that are recorded in polysomnography?

Answer:

- EEG: Electroencephalography
- EOG: Electrooculogram
- EMG: Electromyogram
- Additional recordings: SpO₂, ECG (electrocardiogram), and breathing pattern



Q12.7: What are the REM disorders?

Answer:

- Nightmare
- Narcolepsy

Q12.8: What are the NREM disorders?

Answer:

- Sleepwalking (somnambulism): Stages 3 and 4 can occur during REM sleep.
- Sleep talking (Somniloquy): Stage 1 and 2 mainly, but possible in all.

- Bruxism: Stage 2 and 1 mainly.
- Nocturnal enuresis: All stages of NREM and REM except stage 1 (Max. during stage 2).
- Night terrors: Transition from stage 3 to stage 4.

BIG QUESTION 13: What are the major hypothalamic nuclei and their functions?

Broad Answer: The hypothalamus contains several nuclei responsible for key autonomic and behavioral functions. Each nucleus has a specific role, such as regulating body temperature, hunger, circadian rhythm, or emotional behavior.

DETAILED QUESTIONS

Q13.1: What are the various hypothalamic nuclei?

Answer:

- Anterior Nucleus
- Posterior Nucleus
- Supra-chiasmatic
- Lateral
- Ventero-medial
- Medial forebrain bundle
- Periaqueductal Gray (PAG)

Q13.2: What are the functions of hypothalamic nuclei?

Answer:

Nucleus	Functions
Anterior Nucleus	• Thermoregulation (Hot) • Sexual behavior • Osmoreceptors
Posterior Nucleus	Thermoregulation (shivering)
Supra-chiasmatic	Biological rhythm (circadian)
Lateral	Thirst and hunger
Ventero-medial	Satiety
Medial forebrain bundle	Reward Centre
Periaqueductal Gray (PAG)	Punishment center (rage center)

BIG QUESTION 14: What are the types and forms of memory in the human brain, and how are they processed?

Broad Answer:

- Memory is classified based on duration and conscious processing.
- Working memory is a form of short-term memory used briefly to hold information.
- Depending on processing and retention time, memory is further divided into short-term, long-term, explicit, and implicit types.
- Different brain regions like the hippocampus, prefrontal cortex, and neocortex are involved in each form.

DETAILED QUESTIONS

Q14.1: What is working memory, and what are its key features?

Answer:

- A form of short-term memory.
- Lasts only 18-20 seconds unless revised.
- Capacity: 7 ± 2 chunks (e.g., fruits, numbers).
- Controlled by the prefrontal cortex.

Q14.2: How is short-term memory defined, and which brain area is responsible?

Answer:

- Lasts from seconds to several hours.
- Controlled by the ventral hippocampus, especially the CA1 region.

Q14.3: What are the features of long-term memory and its anatomical center?

Answer:

- It can last for years or a lifetime.
- The neocortex is the main center for long-term memory.

Q14.4: What are the two main types of memory based on processing?

Answer:

Memory Type	Characteristics	Example
Explicit (Declarative)	• Words, rules, and language. • Requires conscious processing at the hippocampus & medial temporal lobe.	• Recalling the first day in college
Implicit (Non-declarative)	• Does not require awareness or processing at the hippocampus.	• Remembering how to brush your teeth



4. CVS PHYSIOLOGY

BIG QUESTION 1: How does the cardiac action potential function across different regions of the heart?

Broad Answer: The cardiac action potential differs between myocardial (contractile) tissue and nodal (pacemaker) tissue. It consists of 5 phases (0-4) in myocardial tissue and 3 main phases in nodal tissue. Each phase is driven by specific ion channel activity. The depolarization and repolarization of the heart follow a defined sequence through the conduction system.

DETAILED QUESTIONS

Q1.1: What is the resting membrane potential of myocardial and Purkinje fibers?

Answer:

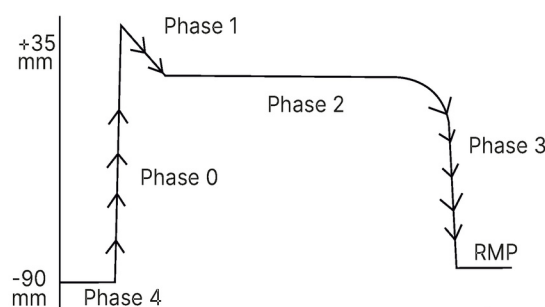
- Myocardium: -90 mV
- Purkinje fibers: -80 mV

Q1.2: What are the phases of the myocardial action potential and their ionic basis?

PYQ: NEET PG 2022

Answer:

Phase	Event	Ionic Basis
0	Depolarization	Influx of Na^+ via fast voltage-gated Na^+ channels
1	Transient rapid repolarization phase	Transient outward K^+ current
2	Plateau	Ca^{2+} influx (L-type channels) balanced by K^+ efflux
3	Repolarization	Efflux of K^+
4	Resting membrane potential	Na^+/K^+ ATPase maintains resting potential



Q1.3: What initiates Phase 0 in myocardial tissue?

Answer: Influx of Na^+ through fast voltage-gated sodium channels (-90 - +35 mV).

Q1.4: What happens in Phase 1 of myocardial AP?

Answer:

- Transient repolarization due to closure of Na^+ channels and activation of transient outward K^+ channels.
- Efflux of potassium from the cell, the membrane potential returns to RMP but the return is incomplete.

Q1.5: What occurs during the plateau phase (Phase 2)?**Answer:**

- Influx of calcium through voltage-gated calcium channels
- Efflux of potassium from the cell

Q1.6: What causes repolarization (Phase 3)?**Answer:**

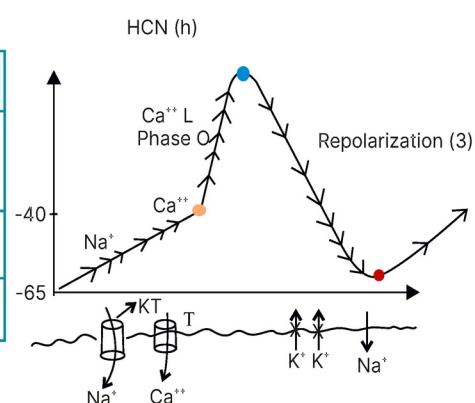
- The calcium channel is closed
- Efflux of potassium

Q1.7: What is the shape of the action potential in nodal tissue (SA/AV node)?

Answer: Triangular or slope-shaped due to slow depolarization (prepotential).

Q1.8: What are the phases of the nodal action potential?**Answer:**

Phase	Description	Ionic Event
4	Prepotential / Pacemaker potential	Influx of Na^+ (funny current via HCN channels)
0	Depolarization	Ca^{2+} influx through L-type calcium channels
3	Repolarization	K^+ efflux

**Q1.9: What is the resting potential of nodal tissue?**

Answer: Approximately -65 to -70 mV.

Q1.10: What is the ionic basis of nodal Phase 4 (prepotential)?**Answer:**

- Influx of sodium (slow) and efflux through HCN channel → Known as Funny Current
- Influx of calcium through T-type calcium channel (rapid Ca channel)
- Closure of potassium channel

Q1.11: What is the ionic basis of nodal Phase 0?

Answer: Influx of L-type calcium (slow type)

Q1.12: What is the ionic basis of nodal Phase 3?**Answer:**

- The calcium channel is closed
- Efflux of potassium

Q1.13: The pacemaker potential is due to:

- Fast sodium channel
- Decrease in potassium permeability
- Slow calcium channel
- Rapid repolarization

Answer: B. Decrease in potassium permeability

- Decreased K^+ efflux + increased Na^+ influx through funny channels causes pacemaker potential.

Q1.14: False about the funny channel in heart:

- A. It is a sodium-potassium channel
- B. It is responsible for spontaneous rhythm-producing tissue of the heart
- C. They are voltage-dependent channels only
- D. They are responsible for diastolic depolarization

Answer: C. They are voltage-dependent channels only

- Funny channels are both voltage- and time-dependent.

Q1.15: How does depolarization and repolarization spread across the heart?

Answer:

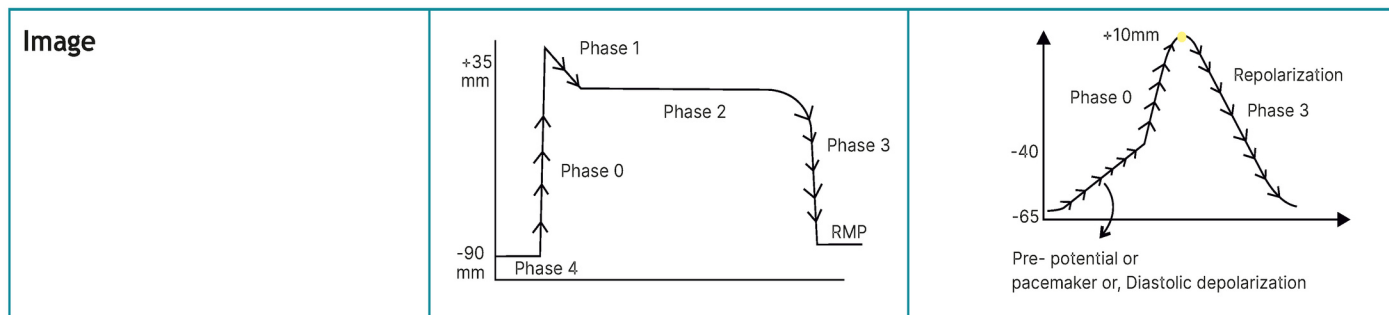
- Atrial depolarization: Starts at the SA node.
- Atrial repolarization: Starts near the SA node.
- Ventricular depolarization:
 - o First part to depolarise: Left endocardial surface of the interventricular septum is the at the middle portion.
 - o Last part to depolarise: Uppermost part of the interventricular septum and the postero-basal epicardial surface of the left ventricle.
- Ventricular repolarization:
 - o First to repolarize: Apical epicardial surface
 - o Last to repolarize: Base endocardial surface.

Q1.16: What are the comparative features of Action Potential in Myocardium vs Nodal Tissue?

Answer:

 PYQ: FMGE 2019, 2022

Feature	Myocardium / Purkinje Fibers	Nodal Tissue (SA/AV Node)
Resting Membrane Potential	-90 mV (Myocardium) / -80 mV (Purkinje)	-65 to -70 mV
Phase 4	True resting membrane potential	Prepotential (slow depolarization)
Phase 0 (Depolarization)	Rapid rise: Fast Na^+ channels	Slow rise: Ca^{2+} influx (L-type Ca^{2+} channels)
Phase 1 (Initial Repolarization)	Present: Transient outward K^+ current	Absent
Phase 2 (Plateau)	Present: Ca^{2+} influx balanced with K^+ efflux	Absent
Phase 3 (Repolarization)	K^+ efflux	K^+ efflux
Automaticity	No	Yes (due to Phase 4 prepotential)
Fast response vs. Slow	Fast-response AP	Slow-response AP



BIG QUESTION 2: What are the phases, timing, and mechanical/electrical events of the cardiac cycle?

Broad Answer: The cardiac cycle lasts **0.8 seconds** and consists of **diastole (0.5 s)** and **systole (0.3 s)**. Diastole includes atrial and ventricular filling phases, while systole includes isovolumetric contraction and ejection. The cycle is tightly coordinated with heart sounds, ECG changes, and valve events.

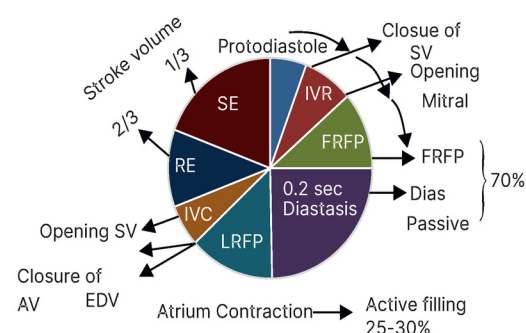
DETAILED QUESTIONS

Q2.1: What is the total duration of the cardiac cycle?

Answer: 0.8 seconds

Q2.2: What is the duration of diastole and systole?

Answer:



Diastole	Systole
Duration: 0.5 seconds	Duration: 0.3 seconds
Diastolic duration of Atrium: 0.7 seconds	Systolic duration of Atrium: 0.1 seconds
Ventricular diastole consists of: • Protodiastole (0.04 seconds) • Isovolumic relaxation (0.06 seconds) - o Begins with the closure of the semilunar valves - o Ends with the opening of mitral valves • First rapid-filling phase • Diastasis (0.2 seconds) • Last rapid filling phase - Corresponds with atrium contraction/systole. - o Filling in this phase is also called active filling phase. - o Contributes 25 to 30% of total ventricular filling.	Consists of: • Isovolumic contraction (0.05 seconds): This phase is responsible for opening semilunar valves. - o Begins with closure of the atrioventricular valves - o Ends with opening of semilunar valves • Rapid ejection - 2/3 (66%) of total ejection occurs during this phase. • Slow ejection - 1/3 (33%) of total ejection occurs during this phase.

Q2.3: What are the phases of diastole and their features?

Answer:

Diastolic Phase	Description
1. Protodiastole (0.04 sec)	Begins with closure of semilunar valves
2. Isovolumetric relaxation	Closure of semilunar valves; ends with opening of AV valves
3. First rapid filling phase	Passive filling of ventricles
4. Diastasis (0.2 sec)	Reduced filling
5. Last rapid filling phase	Due to atrial contraction; contributes 25-30% of ventricular filling

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Q2.4: What are the passive filling phases of the cardiac cycle?**Answer:**

- First rapid filling phase
- Diastasis

Together they contribute about **70%** of total ventricular filling.

Q2.5: What is the importance of the last rapid filling phase?**Answer:**

- Occurs due to **atrial contraction**
- Contributes **25-30%** of ventricular filling
- Also known as **active/atrial kick**

Q2.6: What are the phases of systole and their timing?**Answer:**

Systolic Phase	Description
1. Isovolumetric contraction	AV valves close, pressure rises, all valves closed
2. Rapid ejection	66% of total stroke volume is ejected
3. Slow ejection	33% of total stroke volume ejected

Q2.7: When do all heart valves remain closed during the cardiac cycle?**Answer:**

- Isovolumetric contraction
- Isovolumetric relaxation

Q2.8: What is stroke volume?**Answer:**

- Stroke volume = End Diastolic Volume (EDV) - End Systolic Volume (ESV)

Q2.9: What are the key points on stroke volume from the note?**Answer:**

- Volume of blood ejected in rapid + slow ejection = **Stroke volume**
- Occurs between opening and closing of **semilunar valves**

Q2.10: What is shown in the cardiac cycle pie chart?**Answer:**

- 70% of filling occurs during diastole
- Last part (30%) due to atrial contraction
- Mechanical events synchronized with **ECG and heart sounds**

Q2.11: What are the characteristics of first and second heart sounds?**Answer:**

Feature	First Heart Sound	Second Heart Sound
Phase	Start of isovolumetric contraction	Start of isovolumetric relaxation
Duration	Long (0.15 s)	Short (0.12 s)
Frequency	Low (25-45 Hz)	Sharp/High (50 Hz)
Amplitude	Loud / High	Soft / Low
ECG correlation	Corresponds R wave peak	Corresponds end of T wave

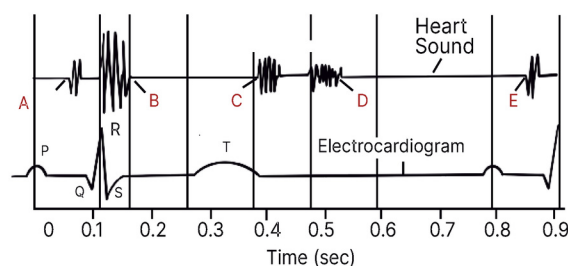
Q2.12: What are the auscultation-based extra heart sounds mentioned?

Answer:

- 3rd heart sound: Corresponds with first rapid filling phase, corresponding with isoelectric line of ECG.
- 4th heart sound: Corresponds with the last rapid filling phase, which corresponds with the end of the wave of ECG.
- Both sounds are inaudible with a stethoscope.

Q2.13: ECG is recorded with phonocardiogram to identify the different heart sounds given below. The first heart sound will appear at which point in the given diagram.

- A. Point A
- B. Point B
- C. Point C
- D. Point D



Answer:

Point B

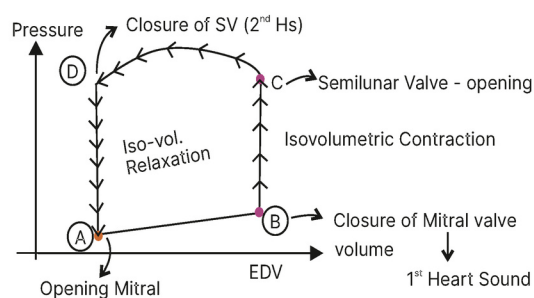
- Point A: Fourth Heart Sound (Corresponds to the P-wave of ECG)
- Point B: First Heart sound (Corresponds to the Peak Of R- wave of ECG)
- Point C: Second Heart Sound (Corresponds to the End of T-wave on ECG)
- Point D: Third Heart Sound: (Corresponds to the isoelectric line on ECG).

Q2.14: What does the PV (Pressure-Volume) loop represent?

Answer:

- At A: Mitral valve are open
 - o Volume at point A: End-systolic volume
- A to B: Ventricular filling phase, volume at B represents the end diastolic volume, mitral valve are closed.
- B to C: Isovolumic contraction phase, which ends with the opening of semilunar valves.
- C to D: Ejection phase of the ventricle. It has
 - o Rapid phase - Ventricular pressure increases.
 - o Slow phase - Pressure and volume will decrease.
- At point D: Closure of semilunar valves.
- From point D to A: Isovolumic relaxation.

PYQ: FMGE 2022 **PYQ: NEET PG 2020**

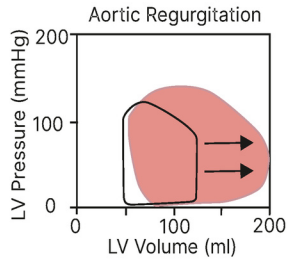
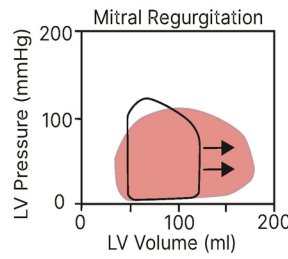
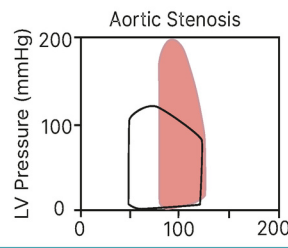
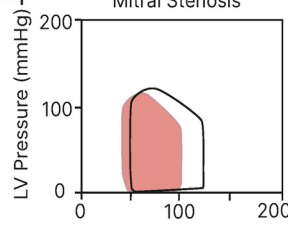


Q2.15: What changes occur in PV loop under pathological conditions?

Answer:

The PV loop shifts:

- Right side: End-diastolic volume increases.
- Left side when contractility increases.
- Upward direction when afterload increases (aortic pressure).

Condition	Change in PV Loop
Aortic Regurgitation	Loop shifted right, no isovolumetric phases  The graph shows LV Pressure (mmHg) on the y-axis (0 to 200) and LV Volume (ml) on the x-axis (0 to 200). The loop is shifted to the right, indicating an increase in end-diastolic volume. The isovolumetric contraction and relaxation phases are absent, replaced by a continuous flow of blood during both systole and diastole.
Mitral Regurgitation	EDV increases, loop shape altered  The graph shows LV Pressure (mmHg) on the y-axis (0 to 200) and LV Volume (ml) on the x-axis (0 to 200). The loop is shifted to the right, indicating an increase in end-diastolic volume. The shape of the loop is altered, with a more rounded appearance during systole and a more pronounced isovolumetric relaxation phase.
Aortic Stenosis	Peak pressure increases, narrow loop  The graph shows LV Pressure (mmHg) on the y-axis (0 to 200) and LV Volume (ml) on the x-axis (0 to 200). The loop is narrow, indicating a decrease in stroke volume. The peak pressure is increased, reflecting the high resistance to flow during systole.
Mitral Stenosis	Low EDV, smaller loop  The graph shows LV Pressure (mmHg) on the y-axis (0 to 200) and LV Volume (ml) on the x-axis (0 to 200). The loop is smaller, indicating a decrease in end-diastolic volume and stroke volume. The pressure is relatively low, reflecting the obstruction to flow during diastole.

Q2.16: Stroke volume =

Answer: EDV - ESV

Q2.17: Normal pressure volume loop of the left ventricle is recorded from an adult person and given below. Which of the following indicates isovolumetric contraction phase of the ventricle?

- d to a
- a to b
- b to c
- c to d

Answer: B. a to b

$$\text{Stroke volume} = 130 - 50 = 80$$

BIG QUESTION 3: What are the features and clinical implications of Jugular Venous Pulse, Cardiac Output, and ventricular loading conditions?

Broad Answer: The Jugular Venous Pulse (JVP) reflects right atrial pressure and has positive and negative deflections related to atrial and ventricular activity. Cardiac Output depends on stroke volume and heart rate, influenced by preload, afterload, and contractility. Preload and afterload are defined by the timing of ventricular loading relative to contraction and are influenced by venous return and aortic pressure respectively.

DETAILED QUESTIONS

Q3.1: What does Jugular Venous Pulse (JVP) represent?

Answer: JVP is a surface recording of the **right atrial pressure**.

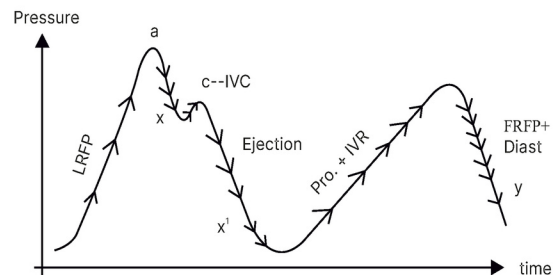
Q3.2: What are the positive waves seen in JVP and their causes?

Answer:

🔍 PYQ: NEET PG 2020, 2022, 2023

🔍 PYQ: FMGE 2019, 2020, 2021

Wave	Cause
a wave	Atrial contraction (last rapid filling phase)
c wave	Bulging of tricuspid valve cusp into atrium
v wave	Venous return during ventricular systole



Q3.3: What are the negative descents seen in JVP and their causes?

Answer:

Wave/Descent	Cause
x descent	Atrial relaxation
x' descent	Downward pulling of tricuspid valve during systole
y descent	Opening of tricuspid valve; rapid ventricular filling

Q3.4: What is the pressure event summary table?

Answer:

Phase	Waves Involved
Late Rapid Filling Phase	a
Isovolumic Contraction	c
First Rapid Filling + Diastasis	v, y
Ejection Phase	x, x'

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Q3.5: What is the formula for Cardiac Output (CO)?

Answer: Cardiac Output = Stroke Volume × Heart Rate

Q3.6: How is blood pressure related to CO?

Answer: Blood Pressure = Cardiac Output × Total Peripheral Resistance (TPR)

Q3.7: What factors influence heart rate?

Answer:

- Autonomic Nervous System:
 - o Sympathetic stimulation increases HR
 - o Parasympathetic (vagal) stimulation decreases HR
- Reflexes:
 - o Bainbridge reflex: ↑ HR in response to venous return
 - o Baroreceptor reflex: ↓ HR in response to ↑ BP

Q3.8: What regulates stroke volume?

Answer:

- Preload (EDV):
 - o Determined by venous return
- Contractility:
 - o ↑ by sympathetic system
 - o ↓ by parasympathetic system
- Afterload:
 - o Determined by aortic pressure
 - o ↑ afterload → ↓ stroke volume

Q3.9: What are the effects described by Starling, Bowditch, and Anrep laws?

Answer:

Law	Description
Frank-Starling	Contractility will increase when the end-diastolic volume is high. ↑ EDV → ↑ Stretch → ↑ Contractility
Bowditch Effect	When the heart rate increases, the contractility will also increase. ↑ Heart Rate → ↑ Contractility (Force-frequency relationship)
Anrep Effect	When afterload increases, the contractility will also increase. ↑ Afterload → Reflex ↑ in Contractility (intrinsic myocardial response)

Q3.10: What are the JVP abnormalities and their clinical associations?

Answer:

Finding	Associated Condition
Prominent a wave	Tricuspid stenosis, Pulmonary hypertension
Cannon a wave	Complete heart block, Junctional rhythm

Absent a wave	Atrial fibrillation
Large v wave	Tricuspid regurgitation
Sharp y descent	Constrictive pericarditis (Friedreich's sign)
Sharp x descent	Cardiac tamponade, Constrictive pericarditis

Q3.11: What is preload and how is it defined?

Answer:

- Definition: Load on the ventricle before contraction begins
- Best Index: End-Diastolic Volume (EDV)
- Main cause: Initial stretch of the ventricle
- Determinant: Venous return

Q3.12: What is afterload and how is it defined?

Answer:

- Definition: Load on the ventricle after contraction begins
- Best Index: Aortic pressure
- Main cause: Systolic wall tension
- Determinant: Total Peripheral Resistance (TPR)

Q3.13: Compare preload and afterload in a tabular format.

Answer:

Feature	Preload	Afterload
Timing	Before contraction	After contraction
Definition	Initial ventricular stretch	Resistance faced by ventricle during ejection
Best Index	End-Diastolic Volume (EDV)	Aortic Pressure
Main Cause	Venous return	Systolic wall stress
Determinant	Venous return	Total Peripheral Resistance (TPR)

BIG QUESTION 4: How is blood pressure regulated and what are the related reflexes and physiological mechanisms?

Broad Answer: Blood pressure regulation involves rapid, intermediate, and long-term mechanisms including neural reflexes (baroreceptor, chemoreceptor, CNS ischemic response), hormonal actions (RAAS, ADH, ANP), and mechanical factors (fluid shifts, myocardial reflexes). Baroreceptors and chemoreceptors sense pressure and chemical changes, influencing autonomic output. Reflexes like Bainbridge and Bezold-Jarisch contribute to heart rate and vascular tone adjustments in response to volume or chemical stimuli.

DETAILED QUESTIONS

Q4.1: What are the rapidly acting mechanisms that regulate blood pressure?

Answer:

- Baroreceptor reflex → Fastest mechanism

- Chemoreceptor reflex
- CNS ischemic response → Second most potent
- Epinephrine secretion from adrenal medulla

Q4.2: What are the intermediate-acting mechanisms of BP regulation?

Answer:

- Capillary fluid shift mechanism
- Stress relaxation and reverse stress relaxation
- Renin-angiotensin-mediated vasoconstriction

Q4.3: What are the long-term mechanisms that control blood pressure?

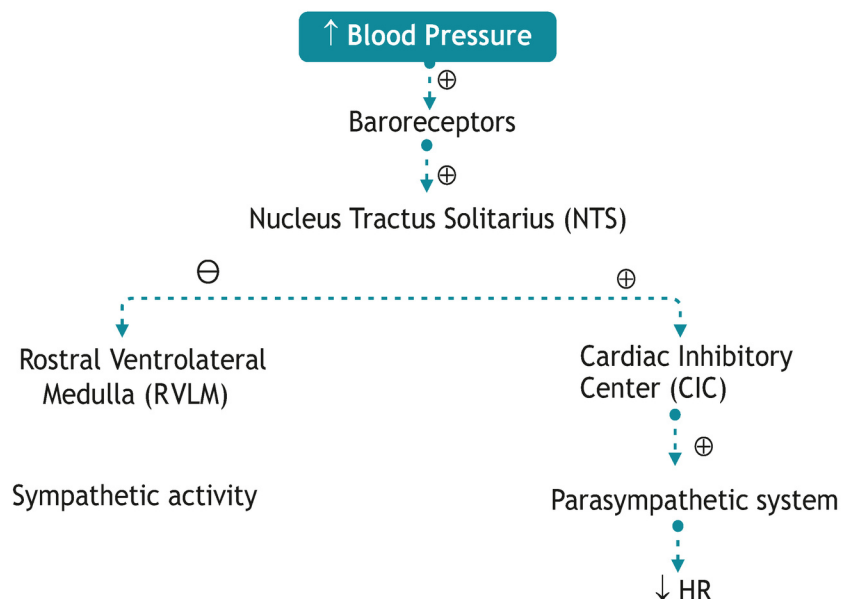
Answer:

- **Renal fluid conservation mechanism**
 - o Hormones involved
 - ADH (Vasopressin)
 - ANP
 - Aldosterone
- **Renin-Angiotensin-Aldosterone System (RAAS)**
 - o **Most potent** long-term mechanism

Q4.4: What are the features of the baroreceptor system?

Answer:

Feature	Details
Location	Carotid sinus wall, Aortic arch wall
Receptor Type	Stretch/Mechanoreceptors
Sensitivity	Most sensitive to pulse pressure , followed by mean arterial pressure
Afferent Nerve	Cranial Nerves IX (Glossopharyngeal) and X (Vagus)
Center	Medulla
Efferents	Sympathetic and Parasympathetic systems



Q4.5: What are the features of the chemoreceptor system?

Answer:

Feature	Details
Location	Carotid body and Aortic body
Cell type	Glomus cell Type I
Stimuli	$\downarrow$ pO ₂ , $\uparrow$ pCO ₂ , $\uparrow$ H ⁺ concentration
Afferent Nerve	Cranial Nerves IX and X
Center	Medulla
Efferents	Sympathetic and Parasympathetic system
Effects	Mild activation $\rightarrow$ $\downarrow$ HR
	Severe activation $\rightarrow$ $\uparrow$ Ventilation $\rightarrow$ $\uparrow$ HR (indirectly)

Q4.6: What happens in CNS ischemic response?

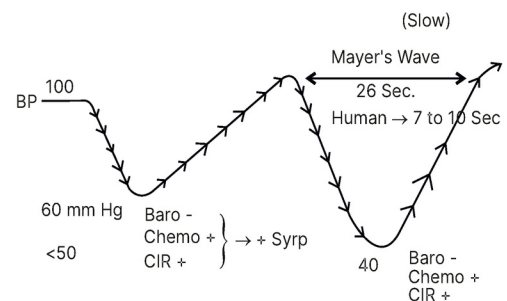
Answer:

- Triggered when BP < 50 mmHg
- CNS ischemia activates the sympathetic system
- Results in vasoconstriction and $\uparrow$ blood pressure
- Also known as "Last ditch response"

Q4.7: What are Mayer waves?

Answer:

- **Slow oscillations** in BP during progressive blood loss
- In **humans**: Occur every **7-10 seconds**
- In **animals**: Every **26 seconds**



Q4.8: How does inspiration and expiration affect BP and HR?

Answer:

Event	Venous Return	Stroke Volume	Blood Pressure	Heart Rate
Inspiration	$\uparrow$ to right heart	$\downarrow$ to left heart	$\downarrow$	$\uparrow$ (Sinus rhythm)
Expiration	$\uparrow$ to left heart	$\uparrow$	$\uparrow$	$\downarrow$ (Sinus rhythm)

Q4.9: What is the Traube-Hering wave?

Answer: Fluctuation in blood pressure during inspiration and expiration

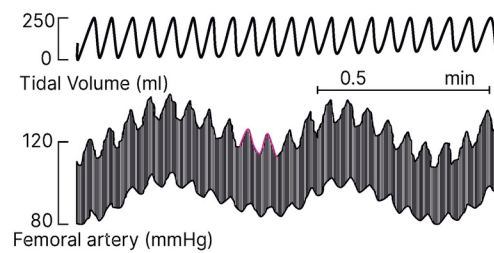
Q4.10: What is sinus rhythm in respiratory physiology?

Answer:

- HR increases during inspiration
- HR decreases during expiration

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Q4.11: The following recording is from a bleeding dog. Wavelets of BP marked by red arrows are known as:



Answer: Traube-Hering wave

Q4.12: What is Bainbridge reflex and what triggers it?

Answer:

- Triggered by ↑ stretch in right atrium
- Effects:
 - o Activation of **Bainbridge reflex**
 - o ↑ **ANP** secretion from right atrium
 - o ↓ **ADH** secretion from hypothalamus

Increase in stretch of right atrium

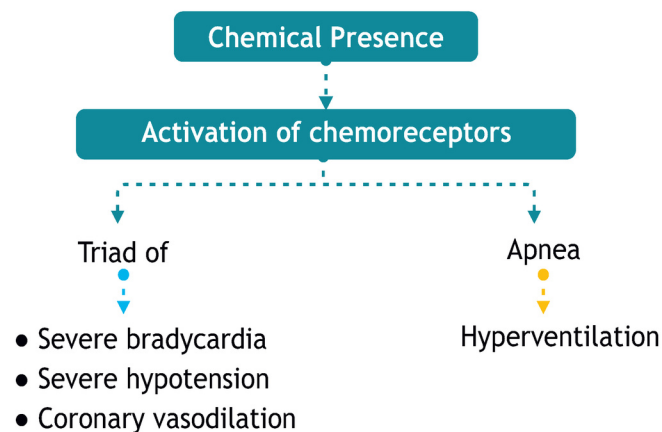
Receptors are activated

Increase in heart rate

Q4.13: What are the receptors and chemical triggers of the Bezold-Jarisch reflex?

Answer:

Feature	Details
Location	Left ventricular wall and interventricular septum
Activators	Serotonin, Veratridine, Capsaicin
Triggered by	Coronary reperfusion therapy, Angiography, High spinal anesthesia, Vaso-vagal syncope



Q4.14: What is the triad seen in Bezold-Jarisch reflex?

Answer:

- Severe bradycardia
- Severe hypotension
- Coronary vasodilation

Q4.15: Chemoreceptors are activated by?**Answer:**

- Decrease in the partial pressure of oxygen in arterial blood.
- Increase in the partial pressure of CO in arterial blood
- Increase in hydrogen ion concentration.

BIG QUESTION 5: How do Valsalva Maneuver, blood vessel types, flow patterns, and exercise influence cardiovascular physiology?

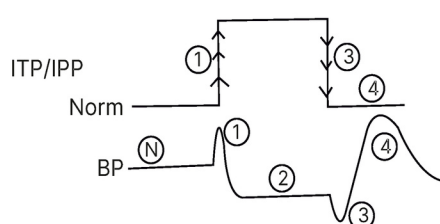
Broad Answer: These physiological mechanisms and adaptations affect cardiac output, blood pressure, and vascular resistance during different conditions such as forced expiration, rhythmic or static exercise, and under various anatomical configurations of blood vessels. Each has a role in ensuring adequate perfusion and oxygenation.

DETAILED QUESTIONS**Q5.1: What is the Valsalva Maneuver?**

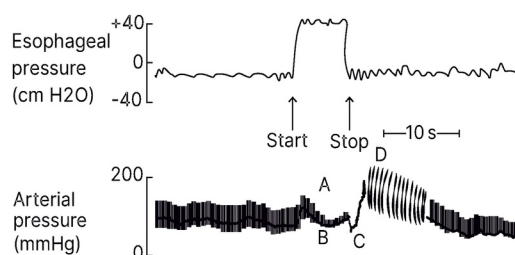
Answer: Forceful expiration against a closed glottis leading to increased intra-thoracic pressure.

Q5.2: What happens during the four phases of Valsalva Maneuver?**Answer:**

Phase	Event
Phase 1	Onset of strain: Increased blood pressure due to the emptying of the pulmonary capillaries, and veins into left ventricle leading to increased stroke volume.
Phase 2	Continued strain: Decrease in blood pressure due to decrease in venous return because of severe compression at the level of the thoracic cavity.
Phase 3	Release of strain: Further decrease in blood pressure due to pooling of blood at the level of pulmonary capillaries.
Phase 4	Recovery phase: Increase in blood pressure due to increased venous return.



Q5.3: The diagram below is the response to straining (Valsalva maneuver) in a normal man, recorded with a needle in the brachial artery. The increase in BP at point A is due to:



Answer: D. Compression of the aorta and pulmonary circulation

Q5.4: What are the types of blood vessels and their functions?

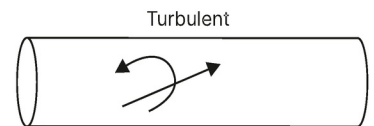
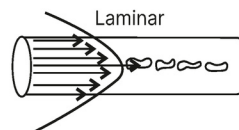
Answer:

Type	Function	Example
Wind-Kessel vessels	Absorb pressure fluctuations	Aorta
Conduit vessels	Conduct blood between body parts	Arteries
Resistance vessels	Regulate blood distribution to organs	Arterioles
Exchange vessels	Exchange gases, nutrients, and wastes	Capillaries
Capacitance vessels	Store large volumes of blood	Venous system
Shunt vessels	AV anastomosis with rich innervation (fingertips, earlobes)	Shunt vessels

Q5.5: What are the characteristics of different types of blood flow?

Answer:

- Laminar blood flow: Maximum flow velocity will be in the center.
 - Cells present in the center -Streamline flow
 - Parabolic blood flow is maintained.
- Turbulent blood flow: Noisy irregular blood flow.



Q5.6: What is Reynolds number and what does it determine?

Answer:

- Formula:

$$Re = \frac{\rho \cdot D \cdot V}{\eta}$$
 - Re = Reynolds number
 - ρ = Density
 - D = Diameter of vessel
 - V = Velocity of blood flow
 - η = Viscosity of the fluid
- Determinants:
 - Density (ρ), Diameter (D), Velocity (V), Viscosity (η)
 - Most important: Velocity

Q5.7: What are the thresholds of Reynolds number?

Answer:

- $Re \gg 3000$: Turbulent
- $Re \ll 2000$: Laminar

Q5.8: What is the Hagen-Poiseuille equation and implication in resistance change?

Answer:

- Formula:

$$R = \frac{8 \eta L}{\pi r^4} \quad R \propto \frac{L}{r^4}$$

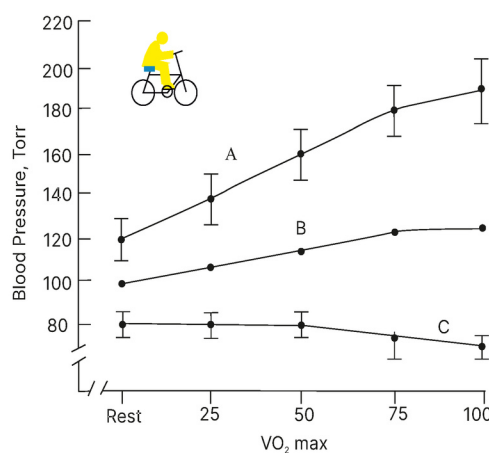
- **Key concept:** Resistance is inversely proportional to the 4th power of radius
- A 50% decrease in radius increases resistance by 16 times

Q5.9: What is isotonic exercise and its cardiovascular effects?

Answer:

- Muscle length changes; load constant (e.g., cycling, jogging)
- HR $\uparrow$ due to psychic stimulation, sympathetic activation, and catecholamines
- SV $\uparrow$ ($\uparrow$ EDV, $\downarrow$ ESV)
- CO $\uparrow$ and SBP $\uparrow$
- TPR $\downarrow \rightarrow$ DBP $\downarrow$
- MAP $\uparrow$

Q5.10: Recording of blood pressure (SBP, DBP, MAP, PP) during isotonic exercise is done in a normal healthy person. Curves A, B, and C represent:



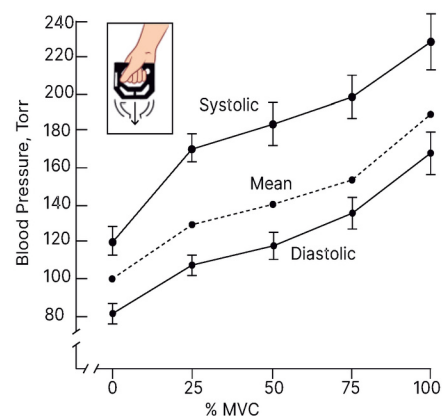
Option	Curve A	Curve B	Curve C
a	MAP	SBP	DBP
b	DBP	MAP	PP
c	PP	SBP	MAP
d	SBP	MAP	DBP

Answer: Option d

Q5.11: What is isometric exercise and how does it differ from isotonic?

Answer:

- Muscle length constant, tone $\uparrow$ (e.g., pushing wall)
- HR $\uparrow$ due to sympathetic stimulation
- SV constant (venous return unaltered)
- CO $\uparrow$, SBP $\uparrow$, DBP $\uparrow$ (due to $\uparrow$ TPR)



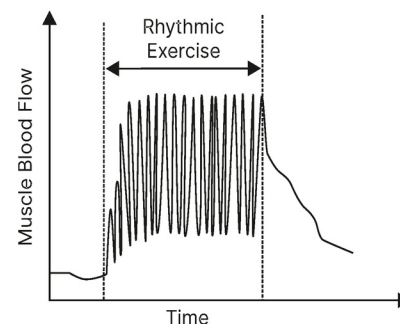
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Q5.12: The effect of isotonic exercise on blood flow in the calf muscle during strong rhythmic contractions is shown below. The blood flow was much less during contraction than between contractions. Rhythmic increase in blood flow is mainly caused by:

- A. Increase in arterial blood pressure
- B. Metabolic vasodilation
- C. Sympathetic mediated vasodilation
- D. Cholinergic sympathetic system

Answer: B. Metabolic vasodilation

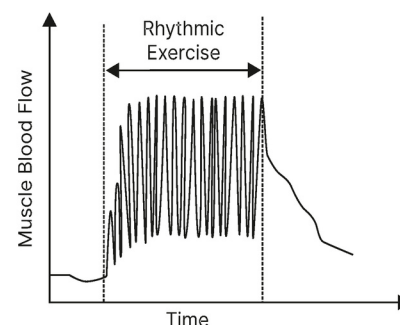
- Metabolic vasodilation
 - o Two most important factor in increasing blood flow:
 - Opening of the capillaries by relaxation of the arteriole
 - Opening the precapillary sphincter
 - o This can be done by local metabolites produced during exercise.
- Increase in arterial blood pressure - 2nd most important cause.
- Sympathetic mediated vasodilation - 3rd most important cause



Q5.13: What are the mechanisms involved in increased blood flow during muscle relaxation?

Answer:

Mechanism	Rank
Metabolic vasodilation	1st
Increase in arterial BP	2nd
Sympathetic vasodilation	3rd



Q5.14: What happens to SBP/DBP during exercises?

Answer:

- SBP ↑ in both isotonic & isometric
- DBP ↓ in isotonic, but **never falls** in isometric

Q5.15: Important Blood Flow Table in Organs

Answer:

Important Table						
	Blood Flow		Oxygen Consumption		Arteriovenous O ₂ difference	Percentage of total Cardiac Output
	ml/min	ml/100g/min	ml/min	ml/100g/min	ml/min	%
Liver	1500	57.7	51	2	34	28
Kidney	1260	420	18	6	14	23
Brain	750	54	46	3.3	62	14
Heart	250	84	29	9.7	114	4.7
Skeletal Muscle	840	2.7	50	0.2	60	15.6
Skin	462	12.8	12	0.3	25	8.6

Q5.16: Calculate the change of resistance in a vessel after a 50% decrease in vessel radius.

Answer: 16 times

Radius = r

New radius = $1/2r$

$$R = \frac{1}{\left(\frac{1}{2}r\right)^4}$$

R = 16 times



5. RESPIRATORY PHYSIOLOGY

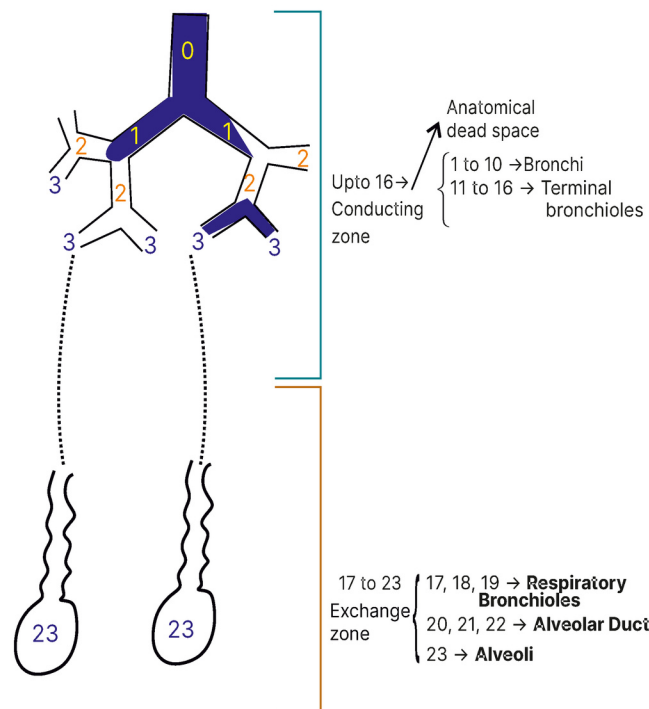
BIG QUESTION 1: What is the functional anatomy of the respiratory system, and how is airway resistance distributed?

Broad Answer: The respiratory tract is divided into airway generations, which contain the conducting zone and the exchange zone. Airflow resistance varies according to different airway generations and is affected by lung volume.

DETAILED QUESTIONS

Q1.1: What is Weibel's classification of the respiratory tract divisions?

Answer:



- **Division number 23:** Alveoli (last division).
- **Divisions 0-16:** No gas exchange (**Conducting zone**)
 - o 1-10 → Bronchi
 - o 11-16 → Terminal bronchioles
- **Divisions 17-23:** Exchange zone
 - o 17-19 → Respiratory bronchioles
 - o 20-22 → Alveolar ducts
 - o 23 → Alveoli

Q1.2: Which divisions show maximum resistance to airflow (as per Weibel's classification) and why?

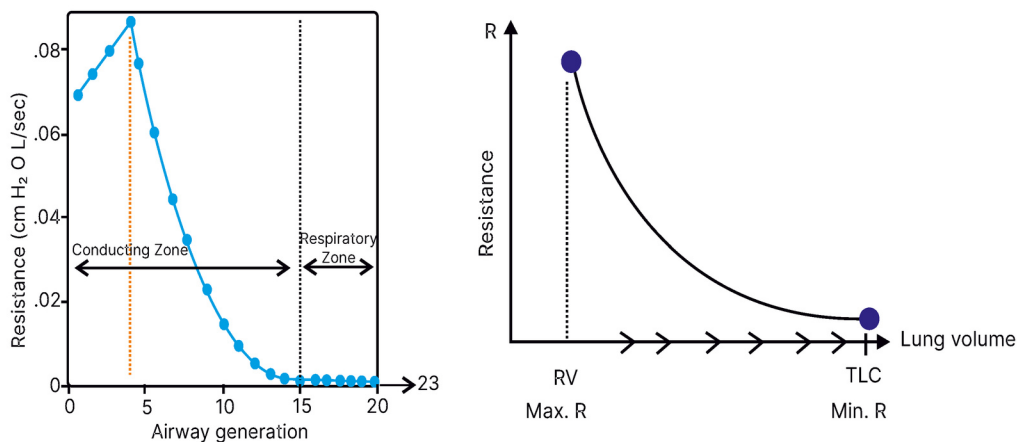
Answer:

- Maximum resistance is seen around airway divisions 4-5 (lobar/segmental bronchi).
- These are medium to large-sized bronchi where airflow is most obstructed.
- Resistance decreases significantly after these divisions due to branching.

Q1.3: How does lung volume affect airway resistance?

Answer:

- At Residual Volume (RV): Maximum resistance due to narrowed airways.
- When the lung volume is fully expanded to total lung capacity (TLC), Bronchi and bronchioles are dilated, → minimum resistance.



BIG QUESTION 2: What is the mechanism of respiration, and what is its physiological significance?

Broad Answer: Lung expansion and contraction are governed by the elastic recoil of the lung and chest wall, balanced by the negative intrapleural pressure. This pressure keeps the lungs expanded. Conditions that alter this pressure balance lead to collapse or expansion, depending on the situation.

DETAILED QUESTIONS

Q2.1: What is the anatomical significance of the Chest wall in respiration?

Answer:

- The lungs are surrounded by visceral pleura.
- The elastic tissues are connected to the visceral pleura through the elastic septum.
- The parietal pleura is attached to the chest wall and the muscles.
- All the structures present in the chest are elastic; when they are stretched, they tend to recoil.

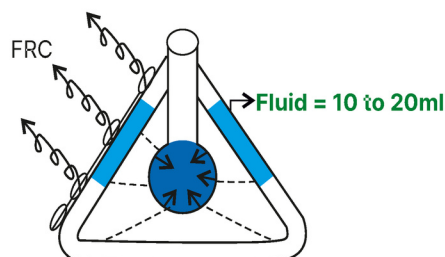
Q2.2: What is the value of Intra-pleural pressure (IPP)?

Answer: Intra-pleural pressure is ~ -5 cm H₂O or -2.5 mmHg.

Q2.3: Why is the Intra-pleural pressure negative?

PYQ: NEET PG 2021, 2022

Answer: The negative pressure is created because the elastic recoiling of the lung tissue is opposite to the elastic recoiling of the chest wall.



Intra-pleural pressure = Negative
Diagrammatic representation of FRC

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Q2.4: What is Trans-pulmonary Pressure?

Answer:

- Trans Pressure or Trans-Pulmonary Pressure (TPP): The pressure difference is measured between Intra-Alveolar Space (IAP) and Intrapleural Space (IPP).
- Trans-pulmonary pressure (TPP) = Intra-alveolar pressure (IAP) - Intra-pleural pressure (IPP)
- $TPP = [0 - (-5\text{ cm H}_2\text{O})] = +5\text{ cm H}_2\text{O}$

Q2.5: What is Trans-thoracic pressure?

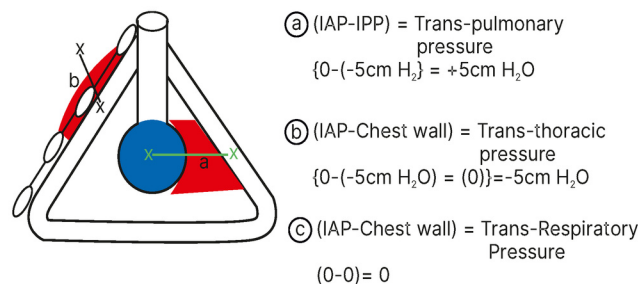
Answer: The pressure difference between the intrapleural space and outside the chest wall, which has the thoracic wall in between, is also known as the trans thoracic pressure (IPP-chest wall).

Q2.6: What is Trans- respiratory pressure?

Answer: It is the pressure difference between the Intra-alveolar pressure (IAP) and the outside chest wall, representing the total pressure needed to move air through the entire respiratory system, including both the lungs and chest wall.

Q2.7: Explain trans-pulmonary pressure, trans-thoracic pressure, trans- respiratory pressure in the form of a diagram.

Answer:



Q2.8: What happens when trans-thoracic pressure becomes zero?

Answer: When the Trans respiratory pressure is zero, the volume of the lung is FRC.

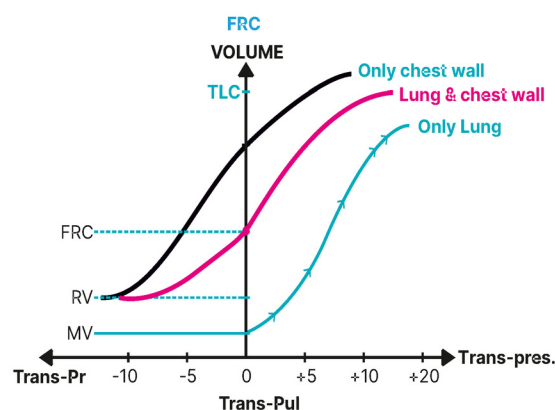
Q2.9: What happens to lung volumes in resting conditions?

Answer:

- When the trans respiratory pressure is zero, both the lung and chest wall are in normal condition, and the volume present is the FRC.
- The maximum volume to which the lung and chest wall can be expanded is the TLC; the minimum volume it can reach is the residual volume.
- The neutral condition, or where the trans respiratory pressure is zero, is the FRC.

Q2.10: What happens when the chest wall is removed?

Answer: When the chest wall is removed, trans-pulmonary pressure becomes zero. Lung collapses to minimal volume (~500 mL).



Q2.11: What happens when the lungs are removed (only the chest wall intact)?**Answer:**

- When the transthoracic pressure is zero in the neutral condition, the volume is 70% of TLC.
- When only the chest wall is present, the maximum point that can be reached is a little beyond the total limit, but the minimum volume that can be reached is at the level of the residual volume. **This is known as the only chest wall curve.**
- To identify the curve, one must look at the trans pressure, which is zero.
- When the Trans-thoracic pressure is zero, it is cut at the FRC. **It is the lung and chest wall curve.**
- When the curve is cut at approximately 70% of the TLC. **This is the only chest wall curve.**
- The only chest wall curve and the lung and chest wall curve are reaching a minimal volume, which is the residual volume.
- When the curve reaches a minimal point below the residual volume, the minimal volume is the **only lung curve.**

BIG QUESTION 3: What is the significance of the ventilation-perfusion (V/Q) ratio in the lungs, and how does it vary?

Broad Answer: Ventilation-perfusion (V/Q) refers to the relationship between air reaching the alveoli (ventilation) and blood reaching the alveoli via the capillaries (perfusion). For efficient gas exchange, there must be a good match between these two. Any imbalance can lead to impaired oxygenation. This V/Q balance is critical for maintaining normal respiratory function.

DETAILED QUESTIONS**Q3.1: What are the average values of ventilation and perfusion in the lungs?****Answer:**

- Ventilation (V) $\approx$ 4.2 L/min
- Perfusion (Q) $\approx$ 5.4 L/min
- Hence, V/Q ratio $\approx$ 0.8
 - o Average ratio in supine position

Q3.2: Where is the ventilation/perfusion ratio maximum, and what is the reason for this?

Answer: The ventilation/perfusion ratio is maximum at the apex because perfusion decreases drastically.

Q3.3: Where in the lung are ventilation and perfusion highest?

Answer: Ventilation and perfusion, separately, are maximum at the base of the lung

Q3.4: How does the V/Q ratio vary at different levels of the lung?

 PYQ: NEET PG 2020  PYQ: FMGE 2019

Answer:

	V (L/min)	Q (L/min)	V/Q ratio
Apex	2 $\uparrow$	0.6 $\uparrow$	= 3.3 (max)
Middle	4	5	= 0.8
Base	6	10	= 0.6 (min)

Q3.5: What are the normal partial pressures of oxygen and carbon dioxide?**Answer:**

- $\text{PaO}_2 \approx$ 100-104 mmHg
- $\text{PaCO}_2 \approx$ 40 mmHg

Q3.6: A 55-year-old man has a large pulmonary embolism that completely blocks the blood flow to the right lung. Which point on the V/Q line of the O_2 - CO_2 diagram above corresponds to the alveolar gas of his right lung?

- A. A
- B. B
- C. C
- D. D
- E. E

Answer: E. E

Explanation:

- If there is an embolus blocking a capillary, ventilation to the alveoli is intact while its perfusion is zero.

- In such cases, the V/Q ratio is infinite.

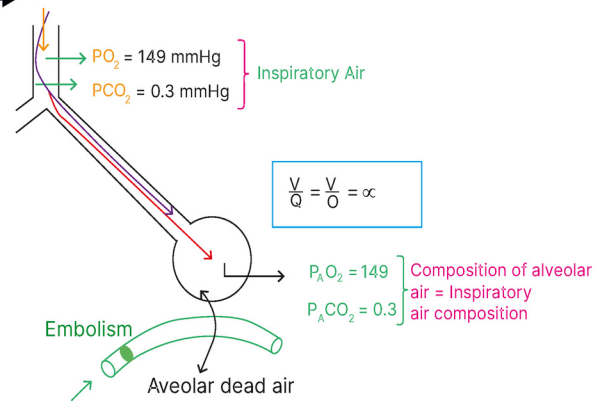
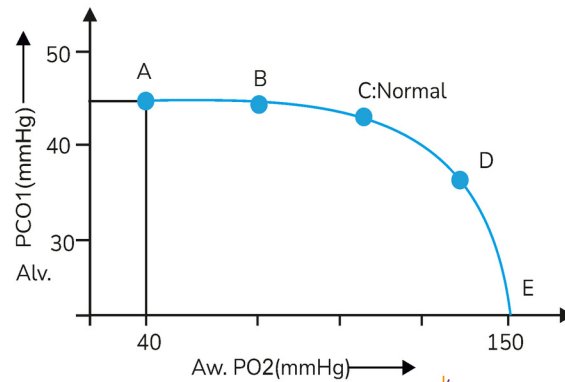
- The inspiratory air that enters the alveoli does not take part in any gas exchange.

- PO_2 of the inspiratory air will be 149 mmHg and PCO_2 will be 0.3 mmHg.

- The partial pressure of oxygen in alveoli are the same as that in inspiratory air in case of embolism.

- The conducting gas composition is also known as Inspiratory air gas composition.

- In the above question, the point marked as E, at the end of the curve, has the highest PO_2 and the Lowest PCO_2 .



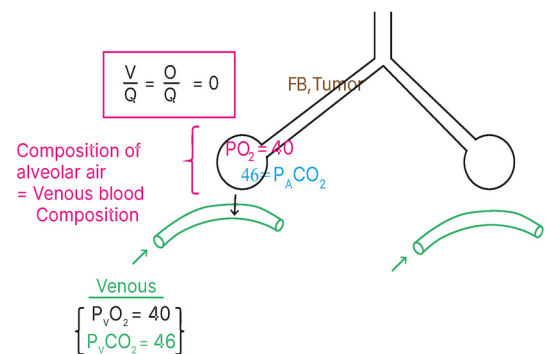
Q3.7: What happens to the V/Q ratio when there is obstruction of the bronchiole?

Answer:

- Whenever a tumor or any foreign body obstructs one bronchiole completely, the ventilation of that alveoli is zero, which means the V/Q ratio is 0.

- O_2 and CO_2 exchange will continue for some time until equilibrium is reached.

PYQ: FMGE 2021



Q3.8: What is the respiratory gas composition when V/Q = 0 or ∞ ?

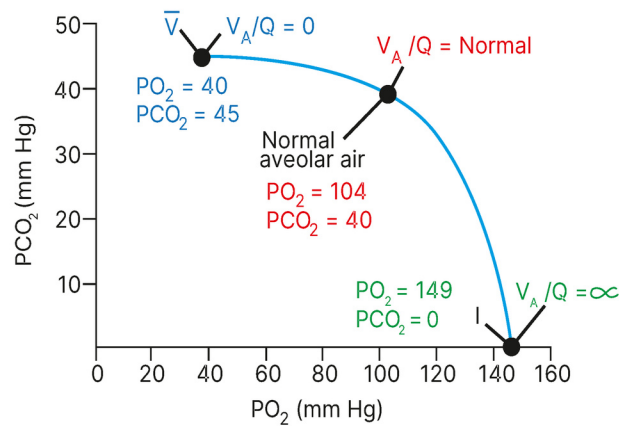
Answer:

- $V/Q = 0 \rightarrow PO_2 = 40$ mmHg, $PCO_2 = 46$ mmHg
- $V/Q = \infty \rightarrow PO_2 = 149$ mmHg, $PCO_2 = 0.3$ mmHg
 - o Ventilation: Intact; Perfusion: Zero $\rightarrow$ Embolus blocking a capillary.
 - o Inspiratory air that enters the alveoli does not take part in any gas exchange.

Q3.9: Summarise the V/Q ratio in a graph.

Answer:

- The normal V/Q ratio is 0.8 and PaO_2 is 100, and $PaCO_2$ is 40.
- If the V/Q ratio is infinite, PaO_2 is 150, and $PaCO_2$ is 0.
- If the V/Q ratio is zero, the PaO_2 is 40 and $PaCO_2$ is 46.
- From the normal point of V/Q ratio, if the V/Q ratio is:
 - o Increasing: $PO_2 \uparrow$ and $PCO_2 \downarrow$
 - o Decreasing: $PO_2 \downarrow$ and $PCO_2 \uparrow$



BIG QUESTION 4: What are the various Lung volumes and capacities, and what is their physiological and clinical importance?

Broad Answer: Lung function is described by a set of volumes that can be directly recorded on a spirogram by an instrument called a spirometer, and by capacities formed from their combinations. Some volumes are not obtainable by simple spirometry and need special gas-dilution or plethysmography techniques. Dynamic indices derived from forced expiration assess airway patency and help distinguish obstructive from restrictive patterns.

DETAILED QUESTIONS

Q4.1: How are lung volumes and capacities classified?

Answer: Lung volumes and capacities are classified into:

- Static volumes/capacities
- Dynamic volumes/capacities

Q4.2: What is the difference between static and dynamic lung volumes and capacities?

Answer:

- **Static:** Measured without regard to time; includes TV, IRV, ERV, RV, and their combinations.
- **Dynamic:** Measured during timed respiratory efforts, primarily to assess airway resistance (e.g., FEV₁, FVC).

Q4.3: What is tidal volume? What is its Normal value?

Answer: Tidal volume (TV) is the amount of normal volume coming in and out of the lungs during normal breathing. Normal TV is approximately 500 ml.

Q4.4: What is inspiratory volume? What is its Normal value?

Answer: When the subject breathes in (an extra amount of air) during maximum inspiratory effort, it is known as the inspiratory reserve volume (IRV). IRV is approximately 2000 ml.

Q4.5: What is Expiratory volume?

Answer: When the subject breathes out (an extra amount of air) during maximum expiratory effort known as expiratory reserve volume (ERV), ERV is approximately 1000 ml.

Q4.6: Which lung volume cannot be measured directly by spirometry, and why?

Answer: Residual Volume (RV) remains in the lungs after maximal expiration and is not recordable on the spirometry.

- It is approximately 1300 ml.

YOURWISH

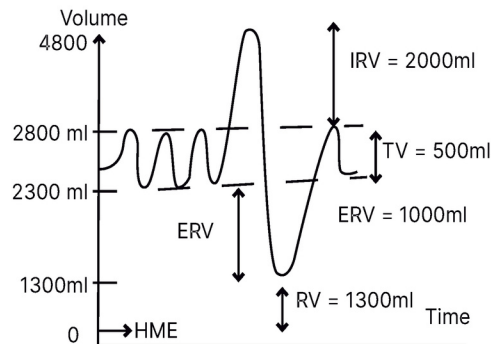
Q4.7: Name three methods used to measure RV**Answer:**

- Helium dilution (most common)
- Nitrogen wash-out (multiple breaths in and out of a closed chamber)
- Body plethysmography (Best method)

Note: Because these methods are used to measure RV, they also measure FRC & TLC as they contain RV.

Q4.8: Describe the static lung volumes with the help of an illustration.

Answer: TV, IRV, ERV, and RV are the static lung volumes.

**Q4.9: How are the various lung capacities defined?**

Answer: Lung capacity is the combination of multiple lung volumes.

- Inspiratory capacity (IC) = TV + IRV
- Vital capacity (VC) = TV + IRV + ERV
- Functional residual capacity (FRC) = RV + ERV
- Total lung capacity (TLC) = TV + IRV + ERV + RV

Q4.10: How are dynamic lung volumes and capacities measured manually?**Answer:**

- Dynamic lung volumes and capacities (like FEV₁ and FVC) are measured using spirometry.
- The subject is instructed to take deep and maximum expiration as possible, the curve will move in a downward direction. At the end of expiration, the maneuver is stopped.

Q4.11: What is Forced Vital Capacity (FVC)?

Answer: The maximum volume that can be forcefully exhaled after a deep inspiration.

Q4.12: What is the minimum time required to complete a forced expiration maneuver?

Answer: At least 3-6 seconds are required for complete forceful expiration.

Q4.13: Define FEV₁.

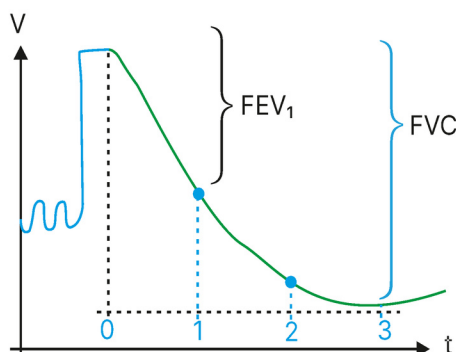
Answer: Forced expiratory volume in 1 second (FEV₁) is the volume of air exhaled in the first second of forced expiration (FVC)

Q4.14: Define FEV₂.

Answer: FEV₂ is the volume of air exhaled after two seconds of forced expiration (from the beginning to the end of the maneuver).

Q4.15: Describe FEV₁ and FEV₂ in the form of a graph.

Answer:



Q4.16: What is the relationship between FVC and FEV₃?

Answer: FVC is almost equal to FEV₃.

Q4.17: FEV₁ is how much percentage of the total volume?

Answer: It is 80% of the FVC.

Q4.18: What is the Tiffeneau ratio?

Answer:

- FEV₁ / FVC ratio = 80%.
- This ratio is known as the Tiffeneau ratio.

Q4.19: What is the clinical significance of the FEV₁/FVC ratio?

Answer: It is used to diagnose different lung diseases (obstructive vs. restrictive).

Q4.20: What percentage of FVC is expired by the 2nd second (FEV₂)?

Answer: FEV₂ is 95% of the total volume (FVC)

Q4.21: What percentage of FVC is expired by the 3rd second (FEV₃)?

Answer: FEV₃ is 99- 100% of FVC.

Q4.22: How do TLC, RV, FEV₁, and FEV₁/FVC change in obstructive and restrictive diseases?

Answer:

🔍 PYQ: INICET 2022, 2023
🔍 PYQ: NEET PG 2019, 2020, 2021
🔍 PYQ: FMGE 2020, 2021, 2022

	Obstructive Disease	Restrictive Diseases		
	Emphysema	Asthma	Parenchymal (fibrosis)	Extra parenchymal (MG)
TLC	↑ 130%	N 100%	↓ 60%	↓ 75%
RV	↑↑ 310%	↑ 120%	↓ 60%	↑↑ 120%
FEV ₁	↓↓ 35%	↓↓ 35%	↓ 75%	↓ 60%
FVC	↓ 60%	↓ 90%	↓↓ 60%	↓ 60%
FEV ₁ / FVC	Reduced	Reduced	Increased	Normal or Variable
Ar	1.5 times	2.5 times	1	1
DLCO	↓↓	↑	↓	↓

BIG QUESTION 5: What are flow-volume loops, and how are they affected in various lung diseases?

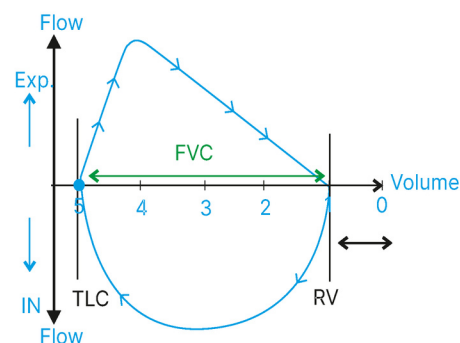
Broad Answer: Flow-volume loops graphically represent the relationship between airflow (Y-axis) and lung volume (X-axis) during forced inspiration and expiration. They help assess pulmonary mechanics and differentiate between obstructive, restrictive, and upper airway obstructive pathologies.

DETAILED QUESTIONS

Q5.1: What are the 2 limbs of a flow-volume loop?

Answer:

- Expiratory limb (TLC → RV)
- Inspiratory limb (RV → TLC)
- Peak expiratory flow at the apex



Q5.2: What is the total volume present in the lung at the end of inspiration?

Answer: 5 liters- this represents the total lung capacity (TLC).

Q5.3: What fixed volume marks the end of expiration on the loop?

Answer: RV ≈ 1 L at end-expiration.

Q5.4: What is the forced vital capacity (FVC) in a flow-volume loop?

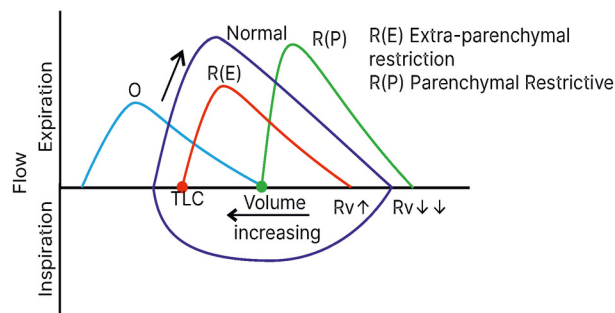
Answer: FVC = 5 - 1 = 4 L.

Q5.5: What are the different patterns seen in a flow volume loop?

Answer:

The above diagram displays four loops:

1. Normal
2. Obstructive (Emphysema)
3. Parenchymal restrictive
4. Extra-parenchymal restrictive



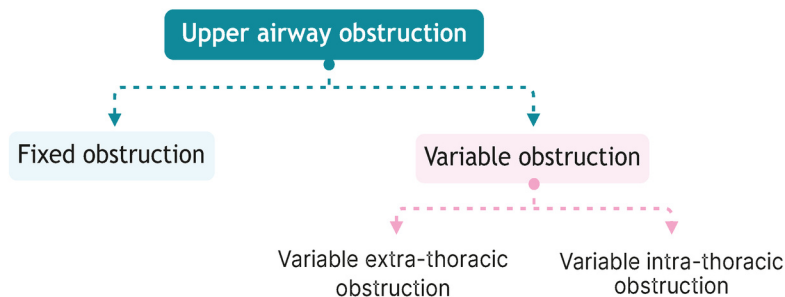
Q5.6: How do various lung diseases differ based on flow-loop parameters?

Answer:

Characteristic	Obstructive (Emphysema)	Parenchymal Restrictive	Extra-Parenchymal Restrictive
Expiratory Peak Flow	Lower than normal	Lower than normal	Lower than normal
Total Lung Capacity (TLC)	Very high (approximately 8.5 liters)	Decreased (from 6 to 4 liters)	Decreased
Residual Volume (RV)	Elevated	Decreased	Higher than normal
Effort-Independent Portion	Scooped-out portion due to dynamic compression	No scooped-out portion due to elasticity	No scooped-out portion due to elasticity
Diagnostic Criteria	Decreased expiratory peak flow, scooped-out portion	Narrow curve decreased TLC and RV, and the difference b/w them is close	Narrow curve, decreased TLC, and high RV

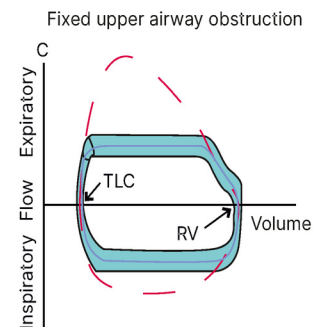
Q5.7: What is upper airway obstruction, and how are they classified?

Answer: Obstruction or restriction in the upper airway (trachea, bronchus).

**Q5.8: What are the flow-volume loop features of a fixed upper airway obstruction?**

Answer:

- Fixed obstruction affects both the inspiratory and the expiratory phases of respiration.
- The curve looks like a box rather than a smooth respiratory portion.
- Ex: Foreign bodies, scarring of airways, or an enlarged thyroid gland.

**Q5.9: What are the features of variable obstruction on flow-volume loops?**

Answer:

Variable extra-thoracic obstruction	Variable intra-thoracic obstruction
The graph shows a flow-volume loop where inspiration is flattened (concave) while expiration is normal. The y-axis is labeled 'Inspiratory Flow' (downward) and 'Expiratory' (upward). The x-axis is labeled 'Volume'. The loop is bounded by TLC (Total Lung Capacity) on the left and RV (Residual Volume) on the right. • In extrathoracic obstruction, inspiration is affected. • Examples are enlarged lymph nodes, paralyzed vocal cords, and flabby pharyngeal muscles.	The graph shows a flow-volume loop where expiration is flattened (concave) while inspiration is normal. The y-axis is labeled 'Inspiratory Flow' (downward) and 'Expiratory' (upward). The x-axis is labeled 'Volume'. The loop is bounded by TLC (Total Lung Capacity) on the left and RV (Residual Volume) on the right. • In intrathoracic obstruction, expiration is affected. • Most commonly caused by Tracheomalacia.

Q5.10: The maximum expiratory flow-volume curve is recorded from a person as given below. What is the total lung capacity (TLC) of this person (in L/min)?

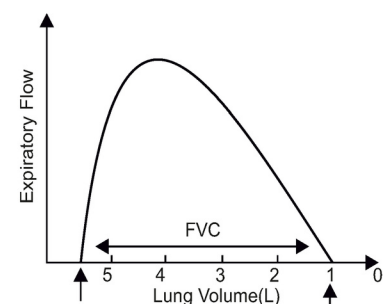
- A. 4.5
- B. 5
- C. 5.5
- D. 6

Answer: C. 5.5

Explanation:

- The x-axis (lung volume) starts at 0 L.
- The curve ends exactly at 5.5 L on the x-axis.

That endpoint on the right (before expiration begins) = TLC = 5.5 L (Option C)



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BIG QUESTION 6: What is lung compliance, and how is it clinically interpreted in normal and pathological states?

Broad Answer: Lung compliance refers to the ease with which the lungs expand in response to changes in pressure. Variations in compliance help differentiate between normal lungs and pathological conditions like fibrosis or emphysema. Compliance curves and calculations are important diagnostic tools. Surfactants produced by alveolar cells play a crucial role in maintaining normal lung compliance by reducing surface tension.

DETAILED QUESTIONS

Q6.1: What is the definition of lung compliance, and how is it calculated?

Answer:

- **Definition:** Lung compliance is the measure of the stretchability or distensibility of the lungs.
- **Formula:**
Compliance = Change in volume (ΔV) / Change in Pressure (ΔP)

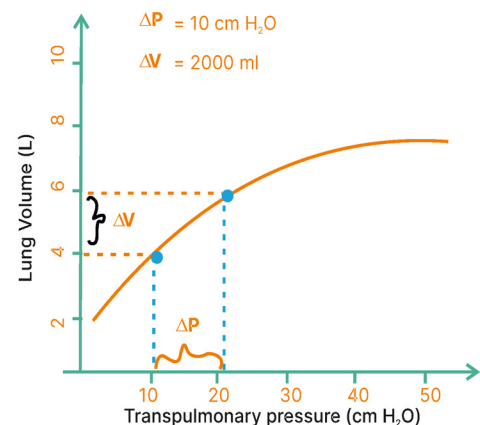
$$C = \frac{\Delta V}{\Delta P}$$

- **Interpretation:**
 - o High compliance → Lungs expand easily
 - o Low compliance → Lungs are stiff and resist expansion

Q6.2: The tidal volume of a person is 600 mL. Upon inspiration, their intrapleural pressure changes from -2 cm of H₂O to -6 cm of H₂O. Calculate the compliance of this person.

Answer:

- The total volume changes inside the lungs equal the tidal volume (TV) of 600mL.
- The change in pressure is 4 cm of H₂O
- $\Delta V = 600 \text{ mL}$; $\Delta P = 4 \text{ cm of H}_2\text{O}$
- Therefore, the compliance will be $600 / 4 = 150 \text{ mL/cm H}_2\text{O}$



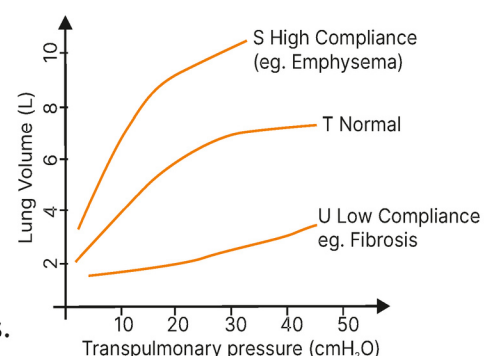
Q6.3: The figure below shows three different compliance curves (S, T, and U) for isolated lungs subjected to various transpulmonary pressures. Which of the following best describes the relative compliances for the three curves?

- A. $S < T < U$
- B. $S < T > U$
- C. $S > T < U$
- D. $S > T > U$

Answer: D. $S > T > U$

Explanation:

- Above Normal Curve: High compliance curve seen in emphysema patients.
- Below Normal Curve: Low compliance curve in restrictive lung diseases like fibrosis.



Q6.4: What is the role of pulmonary surfactant in maintaining lung compliance?

PYQ: FMGE 2021

Answer:

- Surfactant is produced by type II pneumocytes in the alveoli
- It reduces surface tension in alveolar walls → Increases lung compliance → prevents alveolar collapse

Q6.5: What are the components of surfactant?**Answer:**

- Lipids - Mainly
 - o Phospholipids (PL)
 - o Dipalmitoylphosphatidylcholine (DPPC), also k/a dipalmitoyl lecithin, is the most common.
 - o The second most common is phosphatidylcholine
- Proteins - 10%
 - o SP-A, B, C, and D

Q6.6: When does surfactant production begin?**Answer:** Surfactant production begins at 20-24 weeks of gestation.**Q6.7: When does surfactant production mature?****Answer:** Surfactant production matures between 35 and 37 weeks.**Q6.8: When does surfactant appear in the amniotic fluid?****Answer:** It appears in amniotic fluid between 28 and 32 weeks of gestation.**Q6.9: What is a determinant of fetal lung maturity?****Answer:**

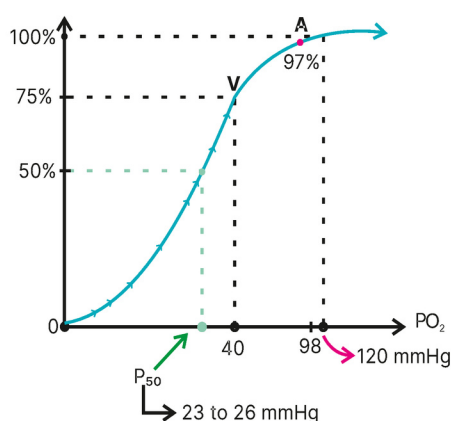
- The Lecithin sphingomyelin (L:S) ratio is a determinant of fetal lung maturity.
- L:S ratio >> 2.0 to 2.5 is indicative of fetal lung maturity.

BIG QUESTION 7: How is oxygen transported in the blood, and what are the factors affecting it?

Broad Answer: Oxygen is primarily transported in the blood bound to hemoglobin, with a small fraction dissolved in plasma. The efficiency of this transport and delivery to tissues depends on several factors, including the partial pressure of oxygen (P_{aO_2}), hemoglobin saturation (S_{aO_2}), and the oxygen-hemoglobin dissociation curve (OHDC). The OHDC shows the relationship between P_{aO_2} and S_{aO_2} . Understanding this curve and its modifiers is essential for interpreting oxygen delivery in both normal and pathological states.

DETAILED QUESTIONS**Q7.1: What are the shape and normal characteristics of the oxygen hemoglobin dissociation curve(OHDC)?****Answer:**

PYQ: NEET PG 2022



- Plots the partial pressure of oxygen on the x-axis, saturation of hemoglobin on the y-axis
- OHDC is a sigmoid or S-shaped curve.
- After a certain point, the curve reaches a plateau

Q7.2: What is the role of P_{50} in oxygen transport?

Answer:

- The value of P_{50} varies from 23 to 26 mmHg.
- Therefore, for 50% of hemoglobin saturation, with oxygen partial pressure of 26 mmHg is required.

Q7.3: What is the relationship between oxygen saturation and partial pressure of oxygen?

Answer:

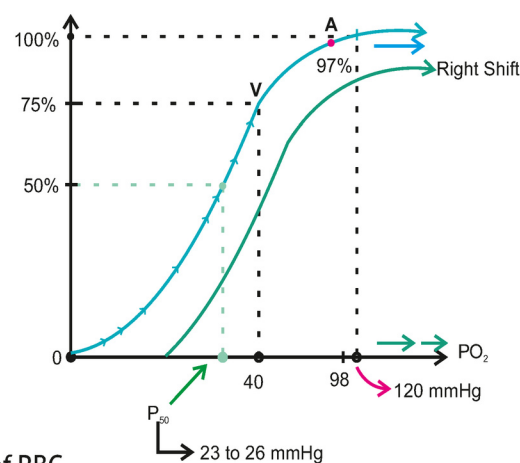
- If partial pressure rises to 40 mmHg, saturation rises to 75%.
- If partial pressure rises to 98 mmHg, saturation rises to 97%.
- If the partial pressure further rises to 120 mmHg, saturation rises to 100%.
- Any further increase in the partial pressure of oxygen will not affect the saturation of hemoglobin, once 100% of hemoglobin is saturated, as all the binding sites of hemoglobin are already occupied.

Q7.4: What are the causes of the Right shift of OHDC?

PYQ: NEET PG 2022

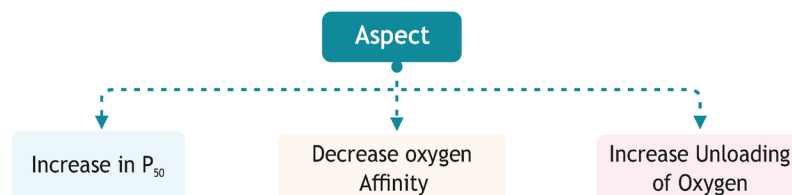
Answer: Causes of the Right Shift of the Curve

- Increase in temperature
 - During exercise, the right shift of the curve occurs mainly due to two reasons:
 - Rise in body temperature
 - Increase in 2,3 DPG
- Increase in PCO_2
- Increase in H^+ ions
- An increase in 2,3 DPG: 2,3 diphosphoglycerate can be seen due to the following conditions.
 - Anaemia (Ex, sickle cell anemia)
 - Hypoxia (Ex, High-altitude hypoxia)
 - During exercise
 - Increase in thyroid, growth, or androgen hormones.
 - Increased amounts of inosine, pyruvate, and phosphate content of RBC



Q7.5: What are the changes seen when the OHDC shifts to the right side?

Answer:



Q7.6: What is the Bohr effect?

Answer:

- The right shifting of the curve due to the $\uparrow PCO_2$ or $\uparrow H^+$ ions is known as Bohr's effect.
- It leads to a decrease in oxygen affinity.
- It occurs in the presence of $\uparrow CO_2$, decreased oxygen affinity of hemoglobin, or increased release of O_2

Q7.7: What are the causes of the left shift of OHDC?

Answer:

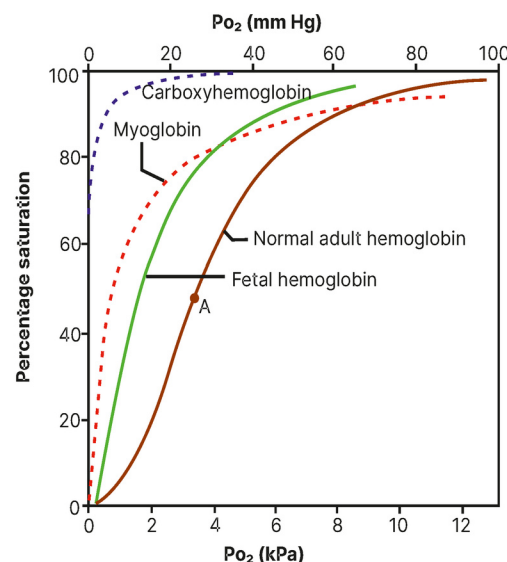
- Causes of the Left Shift of the Curve
- Decrease in temperature.
- Decrease in H^+ ions (Alkalosis)
- Decrease in PCO_2 and 2, 3 DPG.
- Fetal hemoglobin: Due to α_2 and γ_2
- Storage of blood (in blood bank): decrease of 2,3 DPG production.
- CO poisoning: It has 250 times more affinity for hemoglobin than oxygen.
 - CO also impairs the binding of 2,3 DPG with hemoglobin, which leads to the left shift of OHDC.
- Methemoglobinemia: hemoglobin that is attached to the ferrous state will obtain high affinity.
- Thalassemia

Q7.8: What is the shape of the OHDC of Myoglobin?

Answer: OHDC of Myoglobin is rectangular and hyperbolic in shape.

Q7.9: Illustrate the position of carboxyhemoglobin, myoglobin, and fetal hemoglobin in relation to the normal oxygen-hemoglobin dissociation curve.

Answer:



- The above diagram shows carboxyhemoglobin, myoglobin, and fetal hemoglobin on the left-hand side of the normal oxygen-hemoglobin dissociation curve.

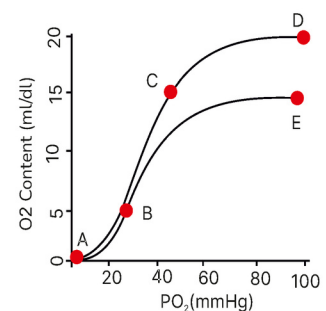
Q7.10: Which of the points on the following figures represent arterial blood in a severely anemic person?

- A. Point A
- B. Point B
- C. Point C
- D. Point D
- E. Point E

Answer: E. Point E

Explanation:

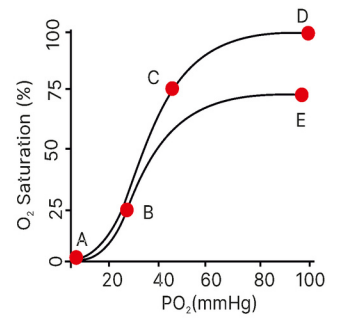
- In anemia, O_2 content is reduced, but PO_2 and saturation remain normal.
- So arterial blood shifts down (lower O_2 content) at normal $PO_2 \rightarrow$ Point E.



YOURWISH

Q7.11: Which of the points on the following figures represent arterial blood in a severely anemic person?

- A. Point A
- B. Point B
- C. Point C
- D. Point D
- E. Point E



Answer: D. Point D

Explanation:

- Despite low Hb, % saturation remains normal in anemia.
- So arterial PO₂ is normal, and saturation is preserved → Point D.

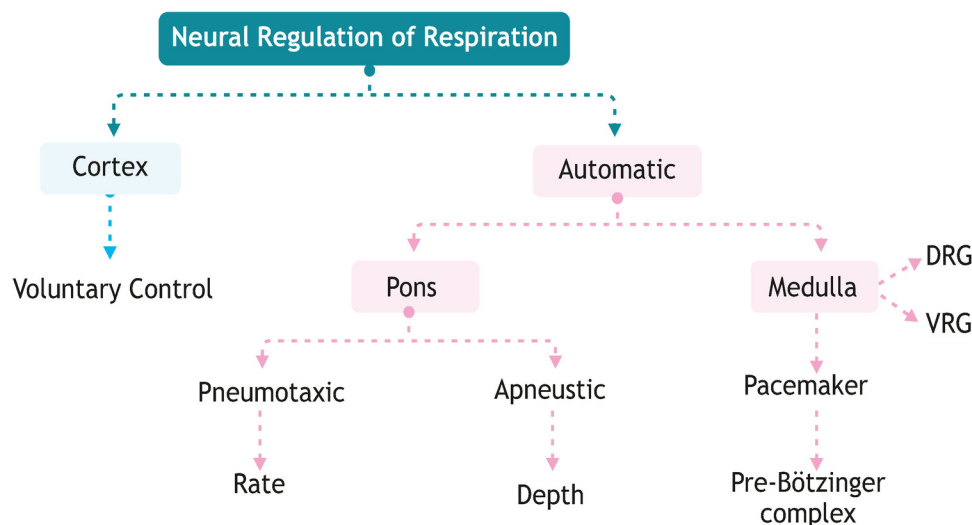
BIG QUESTION 8: How is the process of respiration regulated in the human body?

Broad Answer: The regulation of respiration involves both neural and chemical mechanisms. These systems work together to control the rate and depth of breathing, respond to changes in blood gases, and ensure homeostasis. Various centers in the brainstem and peripheral and central chemoreceptors play a role in this regulation.

DETAILED QUESTIONS

Q8.1: What is the overall neural organization responsible for the regulation of respiration?

Answer:



Q8.2: Which brainstem regions are involved in the automatic regulation of breathing?

Answer:

- Pons
- Medulla

Q8.3: What role do the pons centers play in regulating automatic breathing?

Answer:

- Pneumotaxic center (upper part of the pons) → mainly controls the rate of breathing.
- The apneustic center (Lower part of the Pons) → mainly controls the depth of breathing.
- Both are responsible for controlling the rate and the depth of breathing.

Q8.4: What are the centers for respiration found in the medulla?**Answer:**

Centers for respiration in the medulla are divided into:

- The dorsal respiratory group of neurons (DRG)
- The ventral respiratory group of neurons (VRG)

Q8.5: What are the dorsal respiratory groups of neurons, and what are their roles in automatic breathing?**Answer:**

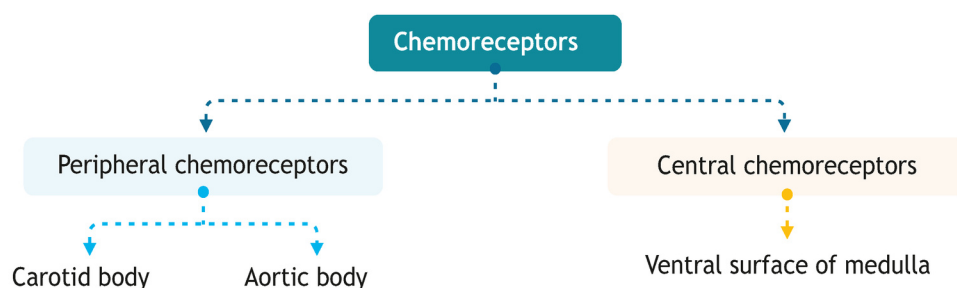
- DRG (Dorsal Respiratory Group) → Sensory integration center
 - DRG is the sensory afferent for the respiratory center.
 - DRG integrates sensory signals from the periphery, such as the lungs or the muscles.
 - It is not responsible for respiratory rhythm generation.

Q8.6: What are the ventral respiratory group of neurons, and what are their roles in automatic breathing?**Answer:**

- The ventral respiratory group of neurons (VRG) is divided into four parts among which one of the parts is a pace-making activity of respiration.
- Consists of four different clear zones.
 - Bötzinger complex (BötC)
 - Pre-Bötzinger complex: Generates respiratory rhythm (Pacemaker)
 - The rostral ventral respiratory group neurons: Control airway dilation of the larynx and pharynx
 - The caudal ventral respiratory group neurons: This mainly works during forceful expiration to control accessory muscle of respiration, abdominal muscle, and external intercostal muscles.
- Thus, the pre-Bötzinger complex is considered the main center for respiration and is known as the pacemaker of respiration.

Q8.7: How is respiration chemically regulated via chemoreceptors?

Answer: Chemical regulation of respiration is made by chemoreceptors present in the body.

**Q8.8: What are the main sensory cells of the chemoreceptor?**

Answer: Type 1 glomus cells are the main sensory cells of the chemoreceptors.

Q8.9: What is the mechanism of activation of glomus cells in peripheral chemoreceptors?**Answer:**

- Glomus cells contain:
 - O₂-sensitive K⁺ channel
 - Na⁺ channel
- Hypoxia, Hypercapnia, and acidosis in the peripheral arterial blood lead to the closure of the O₂-sensitive K channel, which activates the Glomus cell type 1.

Q8.10: Which is the primary or direct stimulus of the peripheral chemoreceptors?

Answer: Hypoxia is the primary or direct stimulus of the peripheral chemoreceptors.

Q8.11: Which is the most sensitive stimulus of the peripheral chemoreceptors?

Answer: Hypercapnia.

Q8.12: Which is the most potent stimulus of the peripheral chemoreceptors?

Answer: The most potent stimulus of the peripheral chemoreceptors will be cyanide poisoning. This is caused by histotoxic hypoxia.

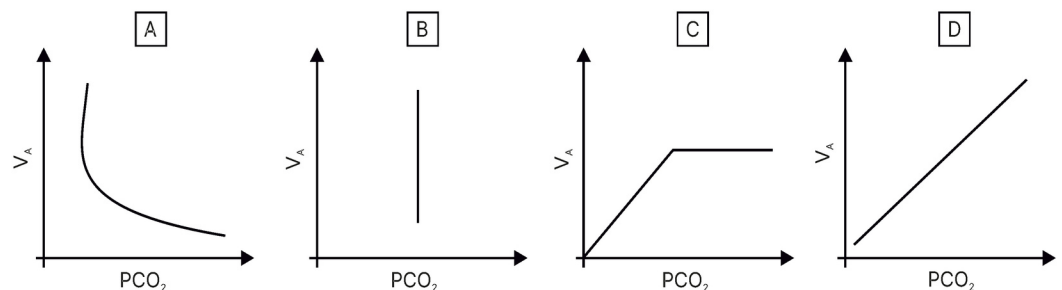
Q8.13: What is the role of central chemoreceptors in respiratory regulation?

Answer:

- Central chemoreceptors are located on the ventral medullary surface just beneath the surface.
- Thus, the main stimulus for central chemoreceptors is the H^+ ion of the cerebrospinal fluid or the interstitial fluid of the brain.
- Hypoxia is a primary stimulus for peripheral and not the central chemoreceptors.
- Acidosis stimulates only peripheral chemoreceptors
- Acidosis cannot stimulate central chemoreceptors because of the Blood-Brain Barrier
- The third stimulus for the peripheral chemoreceptors is hypercapnia.
- Hypercapnia (Direct/primary/most sensitive stimulus) stimulates the central chemoreceptors as well.

Q8.14: Which figure best describes the relationship between alveolar ventilation (V_a) and arterial CO_2 Tension (PCO_2) when the PCO_2 is changed acutely over a range of 35 to 75 mm Hg?

- A. A
B. B
C. C
D. D



Answer: d. D

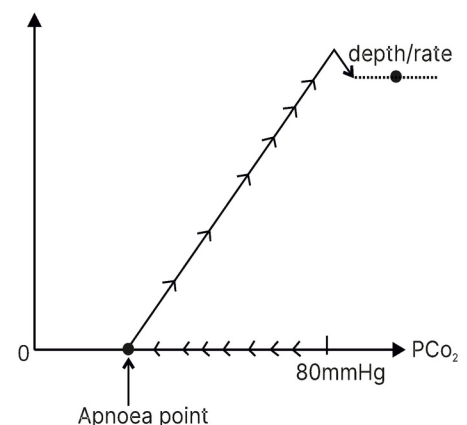
Explanation:

- As PCO_2 rises, $\rightarrow$ chemoreceptors in the medulla and carotid bodies sense it $\rightarrow$ ventilation increases linearly over that range (about 35-75 mm Hg).
- This produces a roughly straight, upward-sloping line $\rightarrow$ option D.

Q8.15: What is the relationship between ventilation and hypercapnia

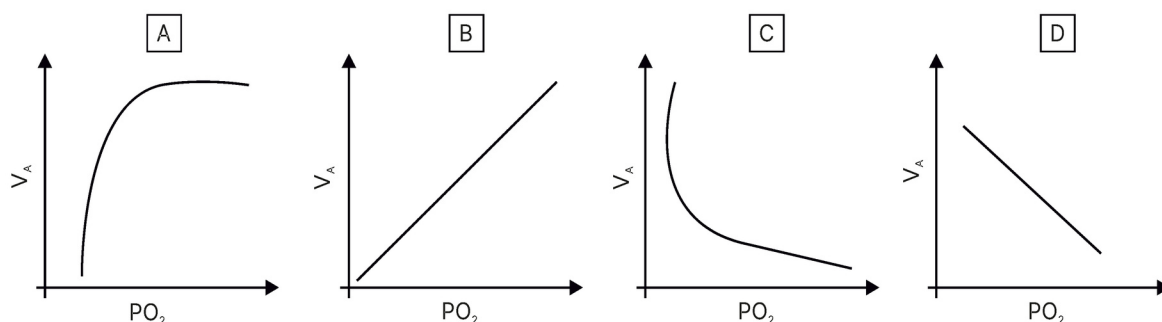
Answer:

- In the graph, the x-axis represents carbon dioxide (PCO_2), and the y-axis represents ventilation.
- When the PCO_2 is beyond 80, an increase in ventilation will not be possible. Rather, there can be depression of ventilation.
- This means that ventilation is stopped for a certain period, and apnea takes place. This point is known as the **apnea point**.



Q8.16: Which figure best describes the relationship between alveolar ventilation (V_A) and arterial O₂ tension (PO₂) when the PO₂ is changed acutely over a range of 0 to 160 mm Hg and the arterial PCO₂ and H⁺ concentration remain normal?

- A. A
- B. B
- C. C
- D. D

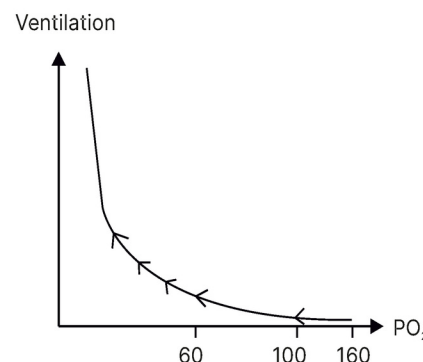


Answer: C. C

Explanation:

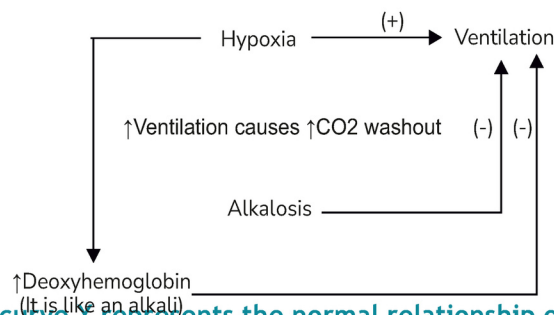
Relationship between hypoxia and ventilation:

- The curve here will not be linear like the CO₂ curve
- When the PO₂ has reduced to 100 mm Hg, there is still no hypoxia, and ventilation rises very little. If the PO₂ is decreasing to 60 mm Hg, the ventilation is not rising to a very high level.
- From 60-100 mm Hg, there is hypoxia, but it does not stimulate the ventilation to high levels, but after 60 mmHg, there is a sudden increase of ventilation.



Q8.17: Why is there not a huge rise in ventilation up to 60 mm Hg of Po₂?

Answer:



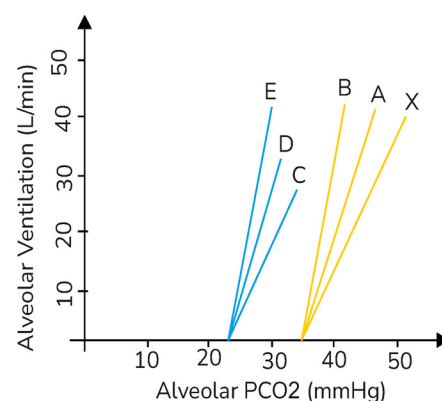
Q8.18: In the following diagram, curve X represents the normal relationship of alveolar ventilation with PaCO₂, when PaO₂ is 100mmHg. If pH changes from 7.4 to 7.3, keeping the oxygen tension the same, the change in alveolar ventilation will shift to which of the curves?

- A. Curve A
- B. Curve B
- C. Curve C
- D. Curve D
- E. Curve E

Answer: C. curve C

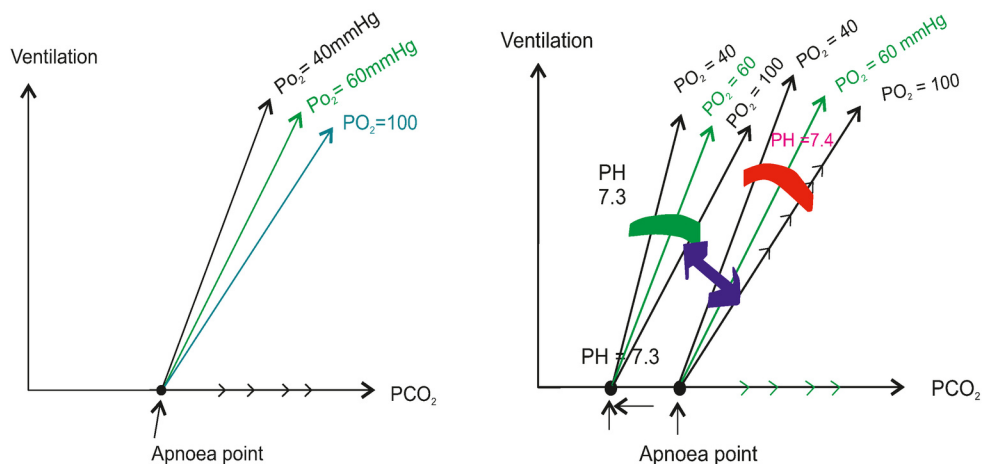
Explanation:

- A fall in pH from 7.4 → 7.3 means more acidic blood → chemoreceptors stimulate ventilation more strongly for the same PaCO₂.
- So, the curve shifts to the left



Q8.19: What happens when both hypoxia and hypercapnia are present?

Answer:



- The relationship between these two was seen to be linear when the PO_2 was normal.
- If there is additional hypoxia in the body, hypercapnia rises in the body and hypoxia. In such a case, a curve can be seen, and here, the PO_2 is 60 mm Hg.
- The more hypoxia and hypercapnia, the curve shifts to the left.
- Thus, hypoxia + hypercapnia = steeper curve/faster rise of ventilation.
- On additional acidosis (7.4), when there is hypercapnia, hypoxia, and acidosis, a similar kind of curve will be seen.
- A curve is drawn that is parallel to the other one.
- However, it has shifted to the left side. The apnoea point has thus shifted to the left.
- Thus, parallel shifting of the curve is observed if there is additional acidosis, with the apnoea point shifting to the left.
- Thus, when there is hypercapnia, hypoxia, and acidosis, the apnoea point will be < 37 .

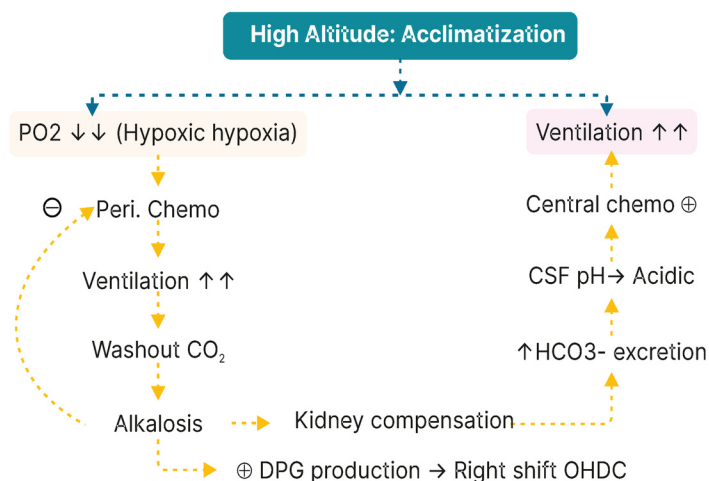
BIG QUESTION 9: What are the physiological changes and clinical consequences seen at high altitudes?

Broad Answer: At high altitudes, hypoxia is the key stimulus leading to compensatory mechanisms in the respiratory, hematologic, and renal systems. These changes can lead to acclimatization or, if severe and rapid, to clinical syndromes like AMS, HACE, and HAPE. Understanding these processes also helps explain exercise physiology and adaptation mechanisms.

DETAILED QUESTIONS

Q9.1: What are the causes and effects of hypoxia at high altitudes?

Answer:



- 🔍 PYQ: NEET PG 2019
- 🔍 PYQ: INICET 2022
- 🔍 PYQ: FMGE 2023
- 🔍 PYQ: AIIMS 2018

- Low PO_2 due to decreased barometric pressure results in **Hypoxic hypoxia**
- This results in
 - Increased alveolar ventilation via peripheral chemoreceptors
 - Results in respiratory alkalosis
 - Stimulates central chemoreceptors → acts to suppress ventilation
 - The body adapts over time via renal compensation and metabolic acidosis

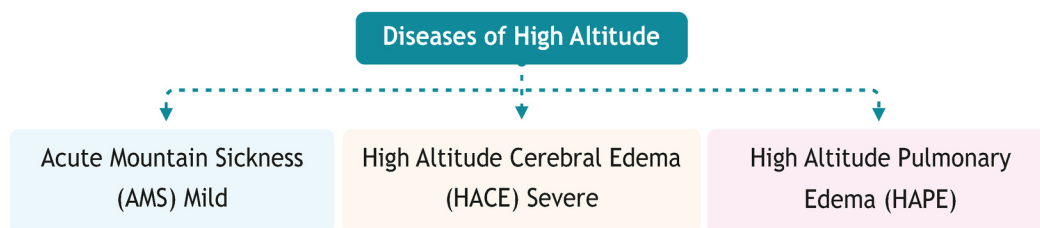
Q9.2: What are the changes that occur during acclimatization at high altitudes?

Answer:

- Increased ventilation
- Increase in diffusion capacity of lungs.
- Increased amount of RBC and Hemoglobin
 - During hypoxia, a hormone called erythropoietin is produced by the kidneys that stimulate RBC and Hemoglobin production in the blood.
- Increased capillary density
- Increased Mitochondria and cytochrome oxidase enzyme

Q9.3: What are the various pathologies/ syndromes associated with high altitude?

Answer:



Q9.4: What are the features of acute mountain sickness (AMS)?

Answer:

- It is developed within 24 hours of ascending high altitude.
- The first 8 hours of ascending high altitude is crucial for the development of AMS.
- The following are the symptoms of AMS:
 - Severe headache
 - Alteration of sleep pattern
 - Anorexia and nausea.

Q9.5: What is high-altitude cerebral edema?

Answer:

- If a person suffers from ataxia or severe mental changes, along with the symptoms of AMS at high altitude, the disease is now known as High altitude cerebral edema (HACE).
- Thus, edema develops in the white matter of the brain.

Q9.6: What are the features of high-altitude pulmonary edema?

Answer:

- It develops within 2-4 days of altitude ascending.
- The following are the symptoms of HAPE:
 - Severe cough

- o Dyspnoea or chest pain
- o White/Pink foamy sputum

Q9.7: What are the treatments for high-altitude-related illnesses?

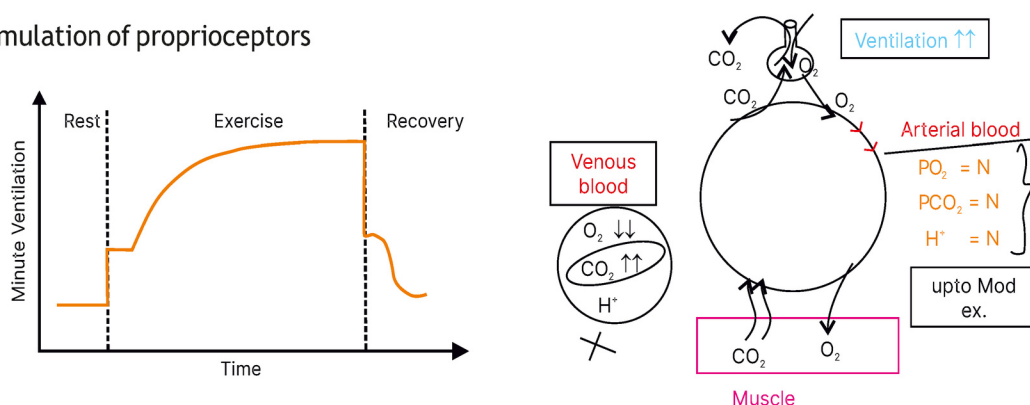
Answer:

- Immediate descent and oxygenation.
- Corticosteroids are used for AMS and HACE: Sumatriptan and Gabapentin are also used.
- Calcium channel blocker drug, Nifedipine is used for curing high-altitude pulmonary edema.
- Acetazolamide can be used to help a patient suffering from AMS, HACE, and HAPE. It enhances the process of acclimatization and helps us cope with adverse environments.
- Thus, acetazolamide is not a treatment of choice, but it is used because it can facilitate the acclimatization process.

Q9.8: Change in ventilation during exercise is graphically shown here. Ventilation increases abruptly with the onset of exercise, which is followed after a brief pause by a further, more gradual increase. The abrupt increase in ventilation is due to which of the following?

- A. Increased Pa CO₂
- B. Decrease PaO₂
- C. Increased lactic acid in muscle
- D. Stimulation of proprioceptors

Answer: D. Stimulation of proprioceptors



Muscle is hyperactive during exercise.

↓
Increased metabolism

↓
Increased O₂ delivery to muscle tissue and CO₂ delivery at the level of venous blood

Venous blood: These changes do not stimulate peripheral chemoreceptors

- Reduced Po₂
- Increased PCO₂
- High H⁺

Arterial blood changes:

- Moderate Exercise induces increased ventilation, which brings in more O₂ that can be replenished and washes out CO₂

- PO₂ is normal
- PCO₂ is normal
- H⁺ is normal
- Increased temperature and potassium ions from muscle tissue are stimuli for increased ventilation during moderate exercise.

Q9.9: What are the mechanisms of ventilation during exercise?

Answer:

- **Abrupt increase:** Neural response
 - o Due to psychic stimuli and proprioceptor stimulation
- **Gradual exercise:**
 - o During Moderate exercise: Due to an increase in body temperature and increased K⁺ levels.
- **During severe exercise:** due to excess lactic acid that gets converted to Co₂

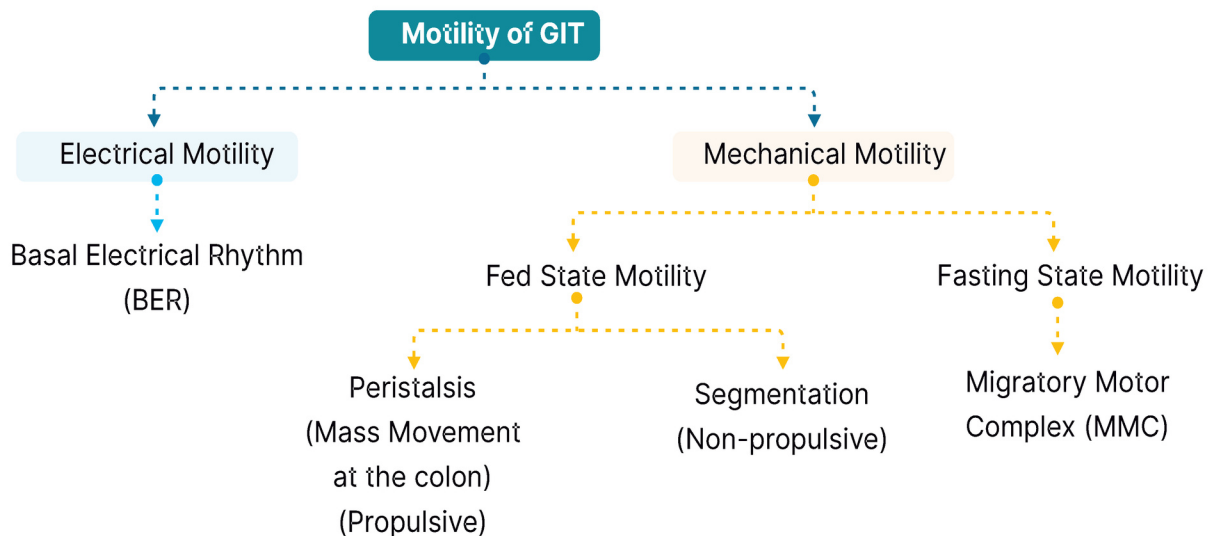


6. GIT PHYSIOLOGY

BIG QUESTION 1: How is gastrointestinal (GIT) motility regulated?

Broad Answer:

- GIT motility is regulated by electrical and mechanical activities involving Basal Electrical Rhythm (BER), spike potentials, and various patterns like segmentation and peristalsis.
- Electrical activity sets the pace for contractions, while neurotransmitters and hormones modulate their strength and occurrence.

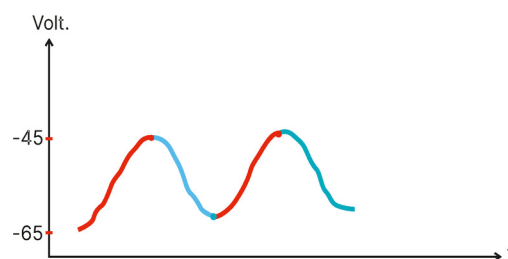


DETAILED QUESTIONS

Q1.1: What is the Basal Electrical Rhythm (BER) in the GIT?

Answer:

- BER is the fluctuating resting membrane potential of GIT smooth muscle ranging from -65 mV to -45 mV.
- Unlike skeletal muscles or neurons (which have fixed RMP), GIT smooth muscles constantly oscillate.
- Generated by Interstitial Cells of Cajal (pacemaker cells) located between smooth muscle layers.
- Marker for Interstitial cells of Cajal: c-KIT.
- Interstitial cells of Cajal also exist at the renal pelvis-ureter junction to help ureter peristalsis.



Q1.2 What is the location of the interstitial cells of Cajal?

Answer: They are located from the mid-part of the stomach up to the level of the anal canal.

Q1.3: What are the rates of BER in different regions of the GIT?

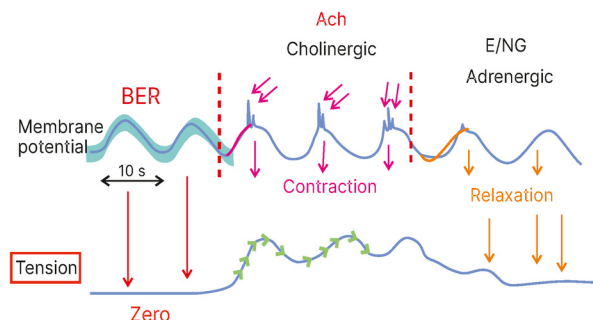
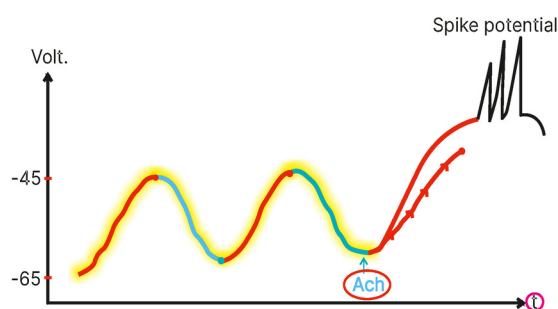
Answer:

Gastrointestinal Region	Rate of BER (cycles/min)
Stomach	4/min
Duodenum	12/min (maximum)
Jejunum	11-12/min
Ileum	8/min
Cecum	2/min (minimum)
Sigmoid Colon	6/min

Q1.4: How do spike potentials relate to BER?

Answer:

- BER alone does not produce muscle contraction.
- Spike potentials (action potentials) superimposed on BER lead to contractions.
- Cholinergic stimulation → BER crosses threshold → produces spike potentials → contraction.
- Adrenergic stimulation → inhibits spike potential → muscle relaxation.



Q1.5: What is segmentation motility, and when does it occur?

Answer:

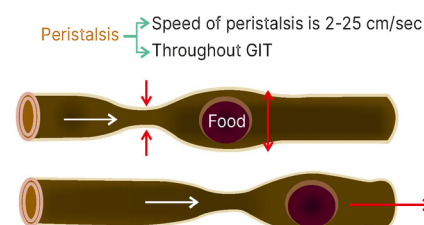
- Segmentation is a form of non-propulsive motility.
- Dominant in the fed state of the GIT.
- Functions to mix luminal contents thoroughly rather than propel them forward.

Q1.6: What is peristalsis, and where is it commonly observed?

PYQ: FMGE 2020

Answer:

- Peristalsis is the **propulsive motility** of the GIT.
- Speed ranges from **5-25 cm/sec**.
- Involves **contraction behind the food bolus** and **relaxation in front**.
- More common in the **sigmoid colon** than transverse colon postprandially.



Q1.7: What happens electrically and mechanically during peristalsis?

Answer:

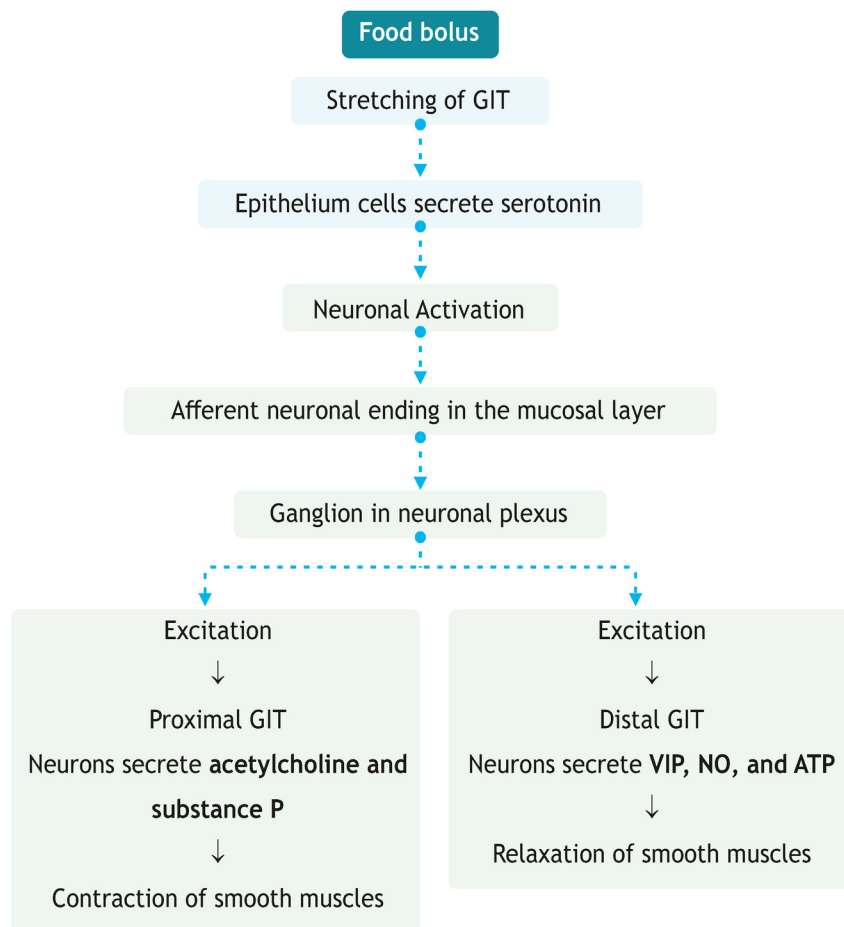
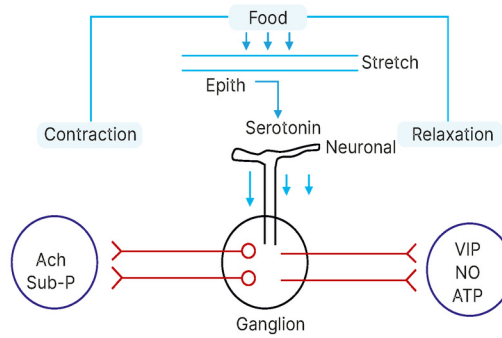
Mechanism of Peristalsis:

- Food bolus stretches GIT wall.
- Epithelial cells release serotonin.
- Neuronal pathways are activated as follows:

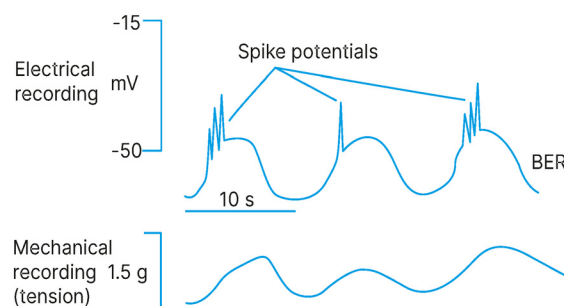
- o Proximal GIT → neurons release Acetylcholine & Substance P → contraction.
- o Distal GIT → neurons release VIP, NO, ATP → relaxation.

PYQ: INICET 2021

PYQ: NEET PG 2019



Q1.8 GIT muscle was isolated and electrical, and mechanical recordings were done. All of the following statements are true about the recording except:



- A. Tension developed proportional to the frequency of spike potential.

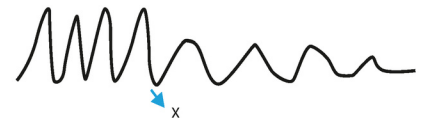
- B. Tension developed is directly proportional to the amplitude of spike potential.
- C. Threshold voltage is -50 mV.
- D. Frequency is around 6/min

Answer: B

- Tension is proportional to the number of spikes, not their amplitude.

Q1.9 A small segment of the mammalian small intestine was mounted over the Dales tissue bath and movement was recorded. A substance was added at the point at 'x'. Which of the following will correspond to x?

- A. Acetylcholine
- B. Adrenaline
- C. KCL
- D. BaCl₂



Answer: B. Adrenaline

⚙️ PYQ: EMGE 2020

⚙️ PYQ: INICET 2020

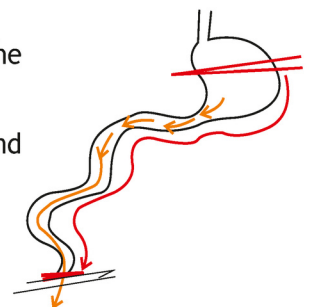
Explanation:

- The graph shows spontaneous rhythmic contractions of intestinal smooth muscle.
- After the point marked 'x', there is a progressive reduction in contractility—suggesting inhibition of smooth muscle activity.
- While Acetylcholine, KCl & BaCl₂ cause contraction; only adrenaline causes decrease in tone and motility.

BIG QUESTION 2: What is the role and regulation of fasting motility in the gastrointestinal tract?

Broad Answer:

- During fasting, the gastrointestinal tract undergoes rhythmic contractions known as the Migratory Motor Complex (MMC).
- These contractions serve a housekeeping function, sweeping residual contents and bacteria from the stomach through the small intestine.
- MMC is initiated by the hormone motilin and recurs cyclically during fasting periods.



DETAILED QUESTIONS

Q2.1: What is the Migratory Motor Complex (MMC)?

Answer:

- MMC is the fasting motility pattern of the GIT.
- It starts in the mid stomach and progresses to the terminal ileum.
- Functions as a “housekeeping wave” to clear debris and bacteria between meals.

Q2.2: When does MMC begin after a meal, and how often does it recur?

Answer:

- Starts 90 minutes after the last meal.
- Contraction cycle recurs every 90-120 minutes.

Q2.3: What is the speed of MMC movement through the GIT?

Answer: Progresses at approximately 5 cm/min.

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Q2.4: Which hormone regulates the MMC, and where is it secreted?**Answer:**

- Motilin regulates and initiates the MMC.
- Secreted by M cells located mainly in:
 - o Duodenum
 - o Jejunum
 - o Stomach
 - o Some parts of the colon

Q2.5: What other physiological mechanism is associated with fasting motility in the stomach?**Answer:**

- Receptive relaxation of the stomach is mediated by the vagus nerve, allowing accommodation of ingested food without a rise in intragastric pressure.
- However, during fasting, the stomach remains contracted except for MMC cycles.

BIG QUESTION 3: How is the pyloric sphincter regulated and what factors influence gastric emptying?**Broad Answer:**

- Gastric emptying is regulated by a combination of neural reflexes and hormonal signals.
- The pyloric sphincter controls the passage of chyme from the stomach to the duodenum.
- Factors such as duodenal content (pH, osmolality, and nutrient type), neural reflexes (like the enterogastric reflex), and hormones (gastrin, CCK, secretin, and GIP) modulate its function.

DETAILED QUESTIONS**Q3.1: Which neural reflex primarily regulates gastric emptying?****Answer:**

- Enterogastric reflex, a vago-vagal reflex, inhibits gastric emptying in response to:
 - o Duodenal distension
 - o Duodenal acidity (pH < 4)
 - o High or low osmolality
 - o Presence of fat and protein in chyme

Q3.2: Which substances most rapidly leave the stomach?**Answer:**

- Isotonic saline empties the fastest.
- Order of emptying:
 - o Carbohydrates > Proteins > Fats

Q3.3: Which hormones stimulate and inhibit gastric emptying?**Answer:**

Hormone	Action on Gastric Emptying
Gastrin	Stimulates (relaxes pyloric sphincter)

CCK	Inhibits (contracts pyloric sphincter)
Secretin	Inhibits
Gastric Inhibitory Peptide (GIP)	Inhibits

Q3.4: What is the role of vagus nerve in gastric relaxation?

Answer:

- Vagus nerve mediates receptive relaxation of the stomach to accommodate food without increasing pressure.
- Essential in coordinating stomach tone and motility.

Q3.5: What is the overall significance of regulating gastric emptying?

Answer:

- Ensures gradual delivery of chyme to the small intestine.
- Prevents overloading the duodenum.
- Allows adequate time for digestion and absorption.

BIG QUESTION 4: What are the major gastrointestinal (GI) hormones and their functions?

Broad Answer:

- GI hormones regulate digestion through secretion, motility, and absorption.
- The three major GI hormones—**Gastrin**, **Cholecystokinin (CCK)**, and **Secretin**—are secreted by specialized cells in different parts of the small intestine and stomach.
- Each hormone has specific stimuli and actions on target organs such as the stomach, pancreas, gallbladder, and intestine.

DETAILED QUESTIONS

Q4.1: What are the major gastrointestinal hormones and their sites of secretion?

Answer:

 PYQ: INICET 2023

GI Hormone	Secreted by	Location of Secretion
Gastrin	G cells	Antrum of the stomach
CCK	I cells	Duodenum and jejunum
Secretin	S cells	Duodenum and jejunum

Q4.2: What are the stimuli for secretion of gastrin, CCK, and secretin?

 PYQ: AIIMS 2019

Answer:

- **Gastrin:**
 - Stimulated by: Peptides, amino acids, stomach distension, parasympathetic input (via gastrin-releasing peptide (GRP))
 - Not stimulated by: Carbohydrates and fats
- **CCK:**
 - Stimulated by: Peptides and fats
 - Not stimulated by: Carbohydrates

- **Secretin:**
 - o Stimulated by: H^+ ions (most potent), peptides (weak)
 - o Fat: Minor role
 - o Not stimulated by: Carbohydrates

Q4.3: What are the main actions of each GI hormone?

Answer:

Hormone	Main Actions
Gastrin	Stimulates H^+ ion (acid) and intrinsic factor secretion from parietal cells
CCK	Stimulates pancreatic enzyme release and gallbladder contraction
Secretin	Stimulates HCO_3^- secretion from pancreatic ductal cells

Q4.4: Which hormone uses GRP as a neurotransmitter instead of acetylcholine?

Answer:

Gastrin release is stimulated via parasympathetic innervation using gastrin-releasing peptide (GRP) instead of acetylcholine.

Q4.5: What is the overall role of GI hormones in digestion?

Answer:

- Coordinate:
 - o Secretion of acid, enzymes, bicarbonate
 - o Gallbladder contraction
 - o Gastric and intestinal motility
- Optimize the chemical environment for digestion and absorption

BIG QUESTION 5: What are the types, volumes, and compositions of gastrointestinal secretions?

Broad Answer:

- The gastrointestinal tract produces around **7-8 liters of secretions daily**, including saliva, gastric juice, pancreatic juice, bile, and intestinal secretions.
- These secretions vary in volume, pH, electrolyte composition, and tonicity.
- They play vital roles in digestion, lubrication, protection, and absorption.

DETAILED QUESTIONS

Q5.1: What are the daily volumes and pH values of various GI secretions?

Answer:

Secretion	Volume (L/day)	pH
Salivary	1	6 to 7
Gastric	1-2	1 to 3.5 (acidic)
Pancreatic	1	8.3 (alkaline)

Bile	0.5-1	7.8
Intestinal	1-3	7-8

- Total GI secretion: 7-8 L/day
- Pancreatic secretion: Most alkaline (pH ~8.3)
- Brunner's glands (duodenum): Produce the most alkaline secretion in GIT (pH 8-9, ~200 ml/day)

Q5.2: Which GI secretion is hypotonic, and which are isotonic?

Answer:

- **Hypotonic secretion:** Salivary secretion
- **Isotonic secretions:** Gastric, pancreatic, bile, intestinal secretions (osmolality similar to plasma)

Q5.3: What is the electrolyte composition of various GI secretions?

Answer:

Component	Plasma	Saliva	Gastric Juice	Pancreatic Juice	Bile	Small Intestine	Large Intestine
Na⁺	150	15	50-90	140	145	120-140	60
K⁺	5	20-30	5-30	5-10	5-10	10-20	30
Cl⁻	110	15	130	75	100	80	40
HCO₃⁻	24	30	0	90-115	35	20-40	0

Q5.4: Which secretion has the highest concentration of bicarbonate?

Answer:

- Pancreatic secretion has the highest HCO₃⁻ concentration.

Q5.5: What are some clinical correlations regarding electrolyte composition of secretions?

Answer:

- Maximum K⁺ concentration: Colonic secretion
- Maximum total K⁺ content: Salivary secretion
- Lowest Na⁺, highest K⁺: Salivary secretion
- Highest Na⁺, lowest K⁺: Biliary secretion
- Highest Cl⁻: Gastric secretion

Q5.6: What is significant about Brunner's gland secretion?

Answer:

- Located in the duodenum
- Produces most alkaline secretion (pH 8-9) in GIT
- Volume ~ 200 ml/day

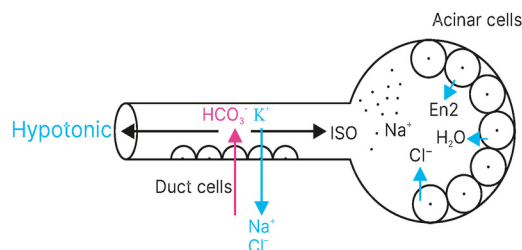
BIG QUESTION 6: What are the mechanisms and phases of salivary secretion?

Broad Answer:

- Salivary secretion occurs in **two phases**—an initial secretion by acinar cells and a subsequent modification in the ducts.

YOURWISH

- The process involves **active ion transport** and produces a hypotonic fluid important for lubrication, digestion, and oral hygiene.



DETAILED QUESTIONS

Q6.1: What are the two phases of salivary secretion?

Answer:

- **Phase 1 (Acinar Secretion):**
 - o Secretion is isotonic
 - o Resembles plasma composition
- **Phase 2 (Ductal Modification):**
 - o Fluid becomes hypotonic
 - o Active ion transport alters composition

Q6.2: What happens during the second phase of salivary secretion?

Answer:

- During ductal modification:
 - o Active Na^+ absorption
 - o Passive Cl^- absorption (follows Na^+ due to electrical gradient)
 - o Active K^+ secretion
 - o HCO_3^- secretion (both active and passive)
- This results in:
 - o Hypotonic saliva
 - o Lower Na^+ , higher K^+ than plasma

Q6.3: What are the relative transport dynamics in the salivary ducts?

Answer:

- Sodium absorption >> Potassium secretion
- Chloride moves passively due to electrical potential
- Bicarbonate secretion is partly active and partly passive

Q6.4: The second phase of salivary secretion involves modification of secretion at the salivary duct. Which of the following is true regarding the second phase?

PYQ: INICET 2022

- Active Na absorption, Passive Chloride absorption, Active K secretion, active and passive secretion of HCO_3^-
- Passive Na absorption, Passive Chloride absorption, Active K secretion, active and passive secretion of HCO_3^-
- Active Na absorption, Active Chloride absorption, Active K secretion, active and passive secretion of HCO_3^-
- Active Na absorption, Passive Chloride absorption, Passive K secretion, active and passive secretion of HCO_3^-

Answer: A

- Sodium actively absorbed
- Chloride passively absorbed
- Potassium actively secreted
- Bicarbonate secreted via both active and passive means

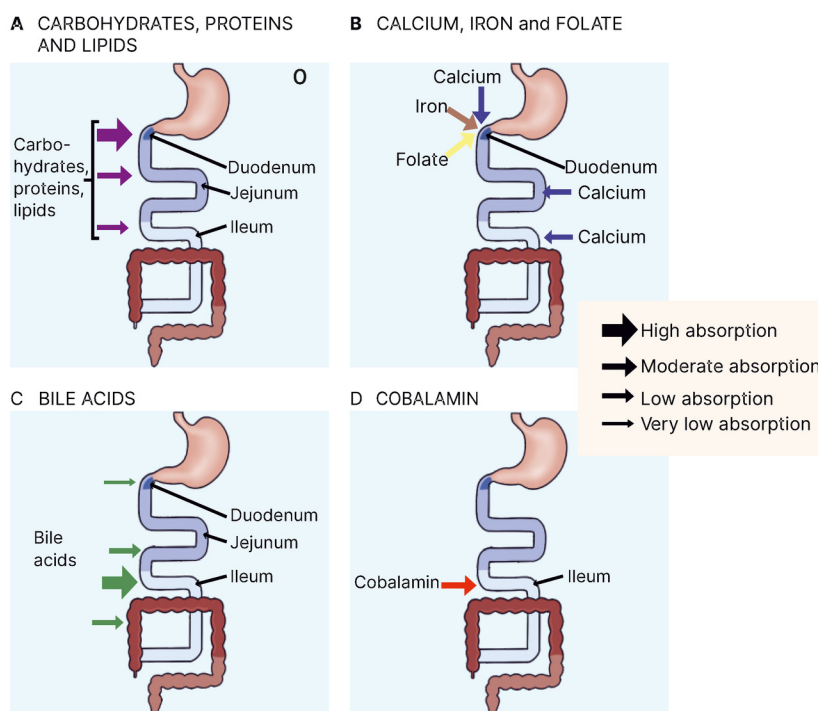
BIG QUESTION 7: How does absorption of nutrients and water occur in the GIT?

Broad Answer:

- The gastrointestinal tract absorbs water and nutrients—carbohydrates, proteins, fats, vitamins, and minerals—primarily in the small intestine.
- The duodenum, jejunum, and ileum each contribute differently to nutrient uptake.
- Water absorption is extensive, leaving minimal fluid in feces.

🔍 PYQ: NEET PG 2020

🔍 PYQ: EMGE 2021



DETAILED QUESTIONS

Q7.1: Where does maximum absorption of carbohydrates, proteins, and fats occur?

Answer:

- Duodenum is the major site.
- Jejunum and ileum also absorb lesser quantities.

Q7.2: What is the daily water volume handled by the GIT, and how is it distributed?

Answer:

- Total fluid in lumen: ~9 liters/day
- Small intestine absorbs: ~8.8 liters/day
 - o Jejunum → ~5.5 L
 - o Ileum → ~2 L
 - o Colon → ~1.3 L
- Feces water content: ~100 mL

Q7.3: Where are specific micronutrients absorbed in the GIT?**Answer:**

- Iron and folate: Duodenum and proximal jejunum
- Bile acids: Distal ileum
- Vitamin B12 (Cobalamin): Ileum



7. RENAL PHYSIOLOGY

BIG QUESTION 1: Which of the following glycoprotein is found in the glomerular basement membrane?

- A. Chondroitin sulphate
- B. Keratan sulphate I
- C. Heparan sulphate
- D. Keratan sulphate II

PYQ: NEET PG 2020

Broad Answer:

Answer: C. Heparan sulphate

Heparan sulphate is the glycoprotein responsible for the negative charge on the glomerular basement membrane (GBM), contributing to the selective permeability of the glomerular filtration barrier.

DETAILED QUESTIONS

Q1.1: What provides negative charge to the glomerular filtration layers?

Answer:

- The presence of negatively charged glycoproteins like **heparan sulphate** on the GBM.

Q1.2: Why is negative charge important in the GBM?

Answer:

- It prevents the filtration of negatively charged molecules, contributing to selective permeability.

BIG QUESTION 2: What is the structure and charge characteristic of the Glomerular Filtration Barrier?

Broad Answer:

The glomerular filtration barrier has three components: endothelium, basement membrane, and podocyte foot processes, each contributing to size and charge-based selectivity.

DETAILED QUESTIONS

Q2.1: What is the structure of the filtration barrier?

PYQ: AIIMS 2020

Answer:

1. **Endothelial cell of glomerular capillary** - Gap: 70 nm
 - o Lined by glycocalyx and glycosaminoglycans (negatively charged)
2. **Basement membrane** - Gap: 10 nm
 - o Contains heparan sulphate proteoglycan (negatively charged)
3. **Foot process of podocyte** - Gap: 25 nm (slit pore)
 - o Slit is not completely open → Contains slit diaphragm (4-14nm pores) and negatively charged glycoproteins

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Q2.2: How does charge affect filtration?**Answer:**

- Positively charged molecules are filtered easily
- Negatively charged molecules are restricted by the negative charge of the barrier

BIG QUESTION 3: How does molecular size and charge affect filterability through the glomerular barrier?**Broad Answer:**

Filterability through the glomerular membrane depends on molecular size and charge, with smaller and positively charged molecules filtered more easily.

DETAILED QUESTIONS**Q3.1: What are the categories of substances based on filterability?****Answer:**

Type	Molecular Radius	Filterability	Examples
Freely Filterable	< 1.8 nm	No hindrance	Na ⁺ , K ⁺ , H ₂ O, Urea, Cl ⁻ , Glucose, Sucrose, Inulin
Selectively Permeable	1.8-4.2 nm	May or may not pass	Lysozyme, Myoglobin, Lactoglobulin, Egg albumin
Impermeable	> 4.2 nm	Not filtered	Bence Jones proteins, Hemoglobin, Serum albumin

Q3.2: What is the filtration pattern based on charge?**Answer:** Filtration preference:

Positively charged >> Neutral >> Negatively charged

- Negatively charged molecule has very less filtration at the level of the barrier (compared to positively charged and neutral molecule).

BIG QUESTION 4: Calculate the net filtration pressure given the following values (Assume that no proteins were filtered):

- P_{GC}: 42 mm Hg
- π_{GC}: 12 mm Hg
- P_{BC}: 16 mm Hg

- A. 34
B. Data not sufficient
C. 14
D. 28

Broad Answer:**Answer:** C. 14

Net filtration pressure is calculated as:

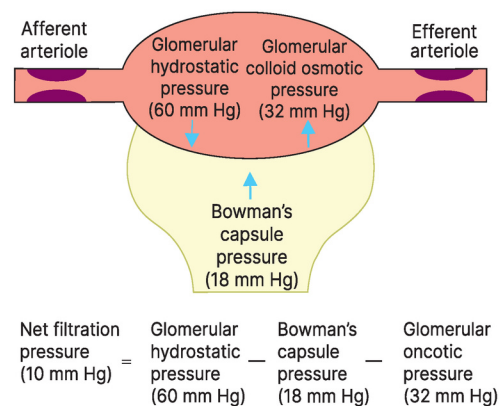
$$\begin{aligned}
 \text{Net force} &= (P_{GC} - P_{BC}) - \pi_{GC} \\
 &= (42 - 16) - 12 \\
 &= 14 \text{ mm Hg}
 \end{aligned}$$

DETAILED QUESTIONS

Q4.1: What are the pressures involved in GFR?

Answer:

1. Glomerular Capillary Hydrostatic Pressure (P_{GC})
2. Bowman's Capsule Hydrostatic Pressure
3. Glomerular Capillary Oncotic Pressure
4. Bowman's Capsule Oncotic Pressure



Q4.2: What assumptions are made?

Answer:

- No proteins are filtered $\rightarrow \pi_B = 0$ (oncotic pressure of Bowman's capsule) = 0

Q4.3: What is the formula for Net Filtration Pressure (NFP)?

Answer: $NFP = (P_{GC} - P_B) - \pi_{GC}$

Q4.4: What are the typical pressure values?

Answer:

- Osmotic pressure depends upon the amount of plasma proteins
- At the level of Bowman's capsule, the amount of plasma protein is very less
 - Because filtration of Plasma protein does not happen
 - Thus, we assume that normally $\pi_B = 0$
- $P_{GC} = 60 \text{ mm Hg}$
- $P_B = 18 \text{ mm Hg}$
- $\pi_{GC} = 32 \text{ mm Hg}$
 - $NFP = (60 - 18) - 32 = 10 \text{ mm Hg}$

BIG QUESTION 5: Which of the following is not involved in GFR Autoregulation?

- A. Myogenic reflex
- B. Tubuloglomerular feedback mechanism
- C. Renin Angiotensin system
- D. Sympathetic supply to kidney

Broad Answer:

Answer: D. Sympathetic supply to kidney

Autoregulation maintains constant blood flow despite BP changes. Sympathetic supply is an extrinsic control, not part of intrinsic autoregulation.

DETAILED QUESTIONS

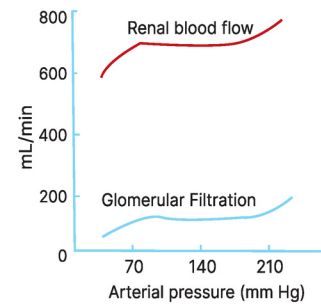
Q5.1: What is Autoregulation?

Answer:

- In the graph, there is no change in RPF when BP changes from 80 to 200 mm/Hg

i.e., the kidney maintains a constant blood flow irrespective of BP - This is autoregulation.

- Constant maintenance of renal blood flow over a BP range (80-200 mm Hg)



Q5.2: What are the three mechanisms of autoregulation?

Answer:

When the blood flow is auto-regulated
↓
GFR is also autoregulated

1. **Myogenic mechanism:** Stretch of afferent arteriole → vasoconstriction
2. **Tubuloglomerular feedback:** Macula densa → adenosine → afferent constriction
3. **Renin-Angiotensin system:** JG cells → renin → systemic vasoconstriction

Q5.3: Why is sympathetic input excluded?

Answer: It modulates renal function during stress but does not maintain baseline GFR

BIG QUESTION 6: Tubulo-glomerular feedback is for:

- A. Controlling plasma volume
- B. Maintenance of systemic BP
- C. Controlling GFR
- D. Controlling blood Na^+ level

Broad Answer:

Answer: C. Controlling GFR

Tubulo-glomerular feedback is a local renal mechanism to regulate GFR by adjusting afferent arteriole resistance in response to sodium chloride concentration sensed in the distal tubule.

DETAILED QUESTIONS

Q6.1: What is the sensing mechanism of tubulo-glomerular feedback?

Answer: It begins with **macula densa** cells sensing increased NaCl concentration in the distal tubule.

Q6.2: What happens after macula densa cells are stimulated?

Answer:

Macula densa cell is stimulated
↓
Releases Adenosine
↓
Adenosine with the help of Lacis cell
↓
Causes contraction of afferent arteriole
↓
Regulate GFR

- They release **adenosine** via Lacis cells, which acts as a vasoconstrictor on the **afferent arteriole**.

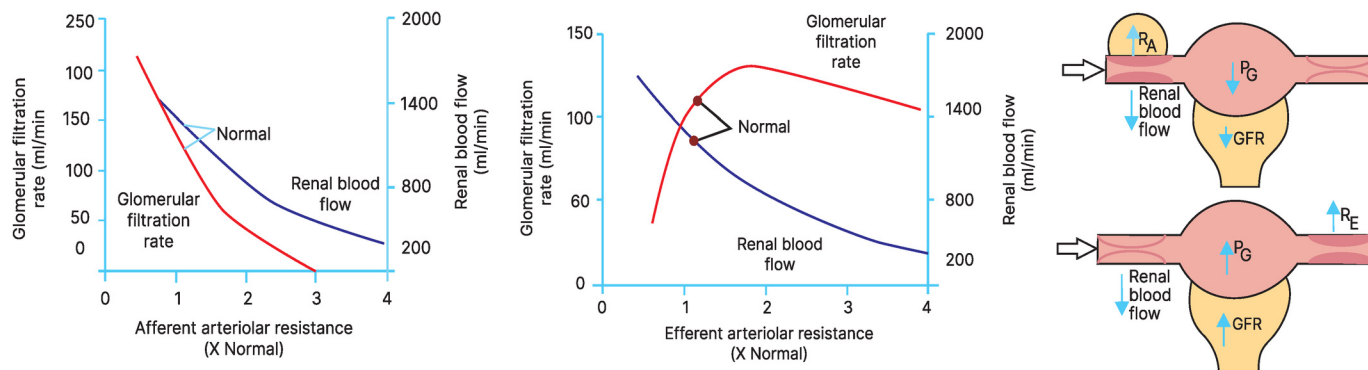
Q6.3: What is the effect of afferent arteriole constriction?

Answer:

- Whenever resistance of afferent arterioles is high → Renal blood flow decreases proportionately → GFR also falls proportionately
- Reduces renal blood flow and subsequently decreases GFR to maintain balance.

Q6.4: What is the effect of efferent arteriole constriction?

Answer:



- Whenever constriction of Efferent arterioles occur → Resistance of efferent arterioles occur → Fall of renal blood flow happens → GFR increases initially when the constriction is mild → GFR decreases when the constriction is severe.
- Mild constriction: **Increases GFR**
- Severe constriction: **Decreases renal blood flow**, eventually reducing GFR

BIG QUESTION 7: In the PCT, luminal concentrations of all of the following will decrease, except?

- A. Chloride
- B. Bicarbonate
- C. Glucose
- D. Aminoacids

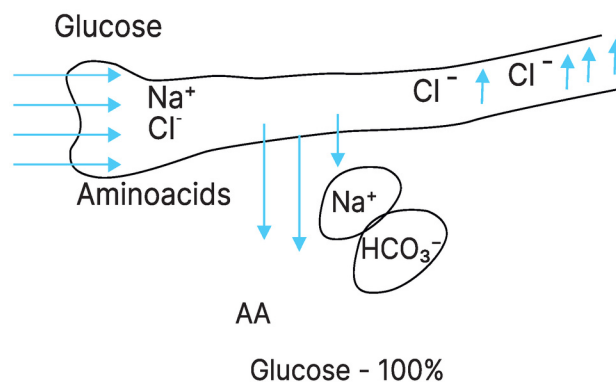
🔍 PYQ: EMGE 2018, 2020, 2021

🔍 PYQ: INICET 2020, 2021

Broad Answer:

Answer: A. Chloride

Although many substances are reabsorbed in the proximal convoluted tubule (PCT), **chloride concentration rises** due to differential rates of reabsorption compared to sodium and co-transported solutes.



DETAILED QUESTIONS

Q7.1: Which substances are fully reabsorbed in the early PCT?

Answer: Glucose and amino acids are reabsorbed 100% in the early PCT.

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Q7.2: How is sodium reabsorbed?

Answer: Sodium is reabsorbed with glucose, amino acids, and bicarbonate.

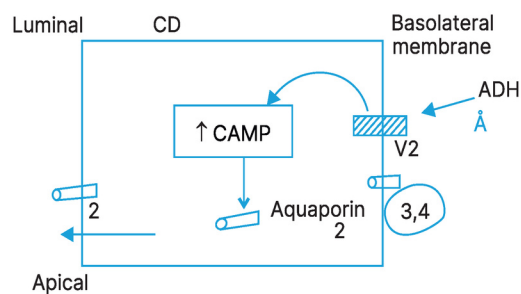
Q7.3: Why does chloride concentration increase in the lumen?

Answer: Because chloride is not reabsorbed as efficiently as sodium, its **relative concentration increases** toward the end of the PCT.

BIG QUESTION 8: ADH regulates which of the following aquaporin?

- A. 1
- B. 2
- C. 3
- D. 4

Broad Answer:



Answer: B. 2

ADH (antidiuretic hormone) increases water reabsorption by promoting insertion of Aquaporin-2 into the apical membrane of collecting duct cells.

DETAILED QUESTIONS**Q8.1: Where does ADH act?**

Answer: On collecting duct, specifically on the **basolateral V2 receptors**.

Q8.2: What is the signaling pathway of ADH?

Answer:

1. ADH binds to V2 receptors.
2. $\uparrow$ cAMP is generated.
3. Stimulates vesicles containing Aquaporin-2.
4. Aquaporin-2 is inserted into the **apical (luminal) membrane**.

Q8.3: Are Aquaporins 3 and 4 regulated by ADH?

Answer:

- No. Aquaporins 3 and 4 are located on **basolateral membrane** and are **not ADH-regulated**.

BIG QUESTION 9: Impaired function of Aquaporin causes which of the following?

Question and Options:

Impaired function of Aquaporin causes which of the following?

- A. Nephrogenic Diabetes insipidus
- B. Liddle syndrome
- C. Cystic fibrosis
- D. Bartter's syndrome

Broad Answer:

Answer: A. Nephrogenic Diabetes insipidus

Nephrogenic Diabetes Insipidus occurs when the kidney fails to respond to ADH, most often due to dysfunctional V2 receptors or Aquaporin-2 channels.

DETAILED QUESTIONS**Q9.1: What is the cause of Nephrogenic DI?**

Answer:

- Mutation in V2 receptor gene (X-linked)
- Normal ADH secretion but defective response

Q9.2: What are the consequences of Aquaporin-2 dysfunction?

Answer:

- Aquaporin-2 is not inserted into apical membrane
- Water reabsorption fails
- Results in **polyuria, nocturia, enuresis, polydipsia**

Q9.3: How is it different from Central DI?

Answer:

- In **Central DI**, ADH secretion is **reduced** due to hypothalamic or pituitary issues
- In **Nephrogenic DI**, ADH secretion is **normal**, but kidney **does not respond**

Q9.4: What are the common clinical features of diabetes insipidus (DI)?

Answer:

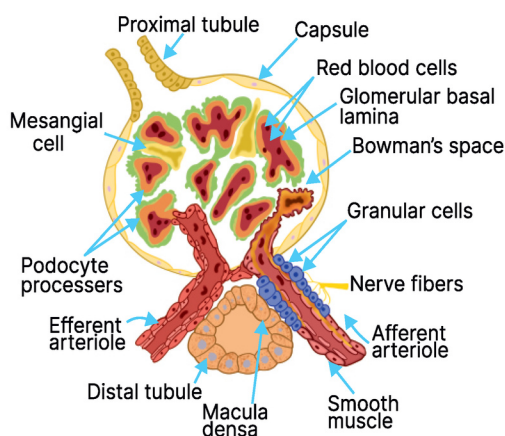
- Large volumes of dilute urine
- Polyuria
- Enuresis
- Nocturia
- Polydipsia

Q9.5: What level of decrease in ADH secretion or action typically leads to diabetes insipidus (DI)?

Answer: A decrease of 75% or more in the secretion or action of ADH .

BIG QUESTION 10: What are the components and roles of the Juxtaglomerular (JG) apparatus?

Broad Answer: The Juxtaglomerular (JG) apparatus helps regulate GFR and systemic BP. It consists of **macula densa**, **JG (granular) cells**, and **Lacis cells**, which function as chemoreceptors, baroreceptors, and mediators respectively.



PYQ: FMGE 2020

PYQ: INICET 2020

PYQ: AIIMS 2020

DETAILED QUESTIONS

Q10.1: What is the role of each cell type in the JG apparatus?

Answer:

Component	Location	Function
JG cells or Granular cells (renin containing granules) or (Intra renal baroreceptor cell)	Afferent arteriole	Modified smooth muscle; secrete renin; act as baroreceptors
Macula densa	TAL-DCT junction	Sense Na^+/Cl^- levels in distal tubule; act as chemoreceptors
Lacis cells	Between afferent & efferent arterioles	Transmit adenosine from macula densa to afferent arteriole; anti-inflammatory role

Q10.2: How does adenosine play a role in this apparatus?

Answer:

- Released by **macula densa** via **Lacis cells**
- Causes vasoconstriction of **afferent arteriole** to decrease GFR when needed

BIG QUESTION 11: Calculate the GFR from the following data:

- Urine flow rate = 1 ml/min
- Urine-Inulin concentration = 100 mg/ml
- Plasma inulin concentration = 2 mg/ml

- A. 200 ml/min
B. 120 ml/min
C. 100 ml/min
D. 50 ml/min

Broad Answer:

Answer: D. 50 ml/min

GFR is measured by the clearance of inulin using the formula: $\text{GFR} = (\text{U} \times \text{V}) / \text{P}$, where U is urine concentration, V is urine flow rate, and P is plasma concentration.

DETAILED QUESTIONS

Q11.1: What is the formula used to calculate clearance?

Answer:

- Clearance = (Urinary concentration × Urine flow rate) / Plasma concentration = UV/P
- So, clearance of Inulin = GFR
= $100 \times 1/2$
= 50 ml/min

Q11.2: How is GFR related to clearance of inulin?

Answer: Inulin is freely filtered and neither secreted nor reabsorbed → Clearance of inulin = GFR

Q11.3: What is the calculated GFR using the given data?

Answer: $\text{GFR} = (100 \times 1) / 2 = 50 \text{ ml/min}$

Q11.4: What are other substances used for clearance studies?

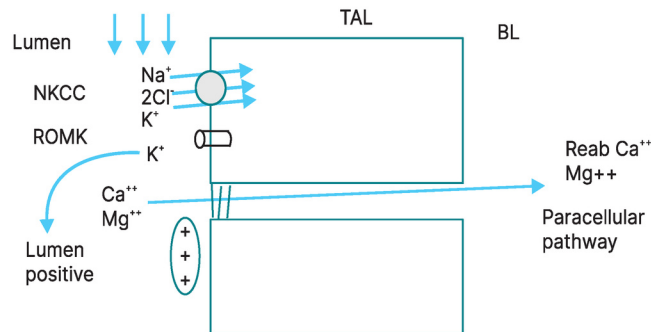
Answer:

- PAH (para-aminohippuric acid): Used to measure renal plasma flow
- Creatinine: Commonly used alternative for GFR

BIG QUESTION 12: The main ion in thick ascending loop of Henle, responsible for reabsorption of divalent ion from lumen is:

- A. Ammonium ion
- B. Hydrogen
- C. Sodium ion
- D. Potassium ion

Broad Answer:



Answer: D. Potassium ion

Potassium recycles into the lumen via ROMK channels, creating a lumen-positive potential that drives paracellular reabsorption of Ca^{2+} and Mg^{2+} .

DETAILED QUESTIONS

Q12.1: What transporter is active in the thick ascending limb (TAL)?

Answer: NKCC ($\text{Na}^+/\text{K}^+/\text{2Cl}^-$ cotransporter) - reabsorbs Na^+ , K^+ , and Cl^-

Q12.2: What is the role of potassium in divalent ion reabsorption?

Answer:

- K^+ diffuses back into lumen via ROMK → creates lumen-positive charge → repels Ca^{2+} and Mg^{2+} into interstitium via paracellular pathway

Q12.3: What effect do loop diuretics like furosemide have here?

Answer: They block NKCC → decrease Ca^{2+} & Mg^{2+} reabsorption → increase urinary excretion

Q12.4: What percentage of divalent ion reabsorption occurs in TAL?

Answer: 70% of Mg^{2+} and 20% of Ca^{2+} is reabsorbed at TAL

Q12.5: What condition results from NKCC mutation?

Answer: Bartter syndrome

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BIG QUESTION 13: Calculate the urine glucose in a diabetic person

- GFR = 90 ml/min
- Blood glucose = 200 mg/dl
- T_m (glucose) = 150 mg/min

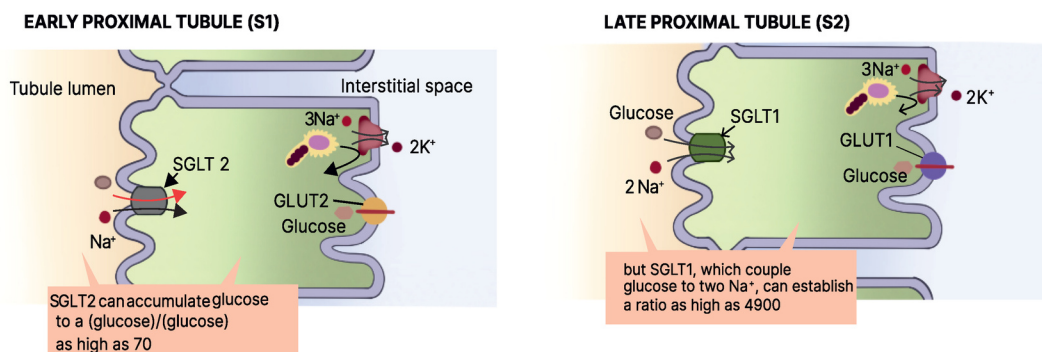
- A. 200 mg/min
- B. 100 mg/min
- C. 50 mg/min
- D. 30 mg/min

Broad Answer:**Answer:** D. 30 mg/min

- Urine glucose = Tubular load - T_m
- Tubular load = $\text{GFR} \times \text{plasma glucose} = 90 \times 2 = 180 \text{ mg/min}$
- Reabsorbed = 150 mg/min $\rightarrow$ Remaining in urine = 30 mg/min

DETAILED QUESTIONS**Q13.1: How is plasma glucose converted to mg/ml?****Answer:** 200 mg/dl = 2 mg/ml**Q13.2: What is the tubular load of glucose?****Answer:** Tubular load = $\text{GFR} \times \text{Plasma glucose} = 90 \times 2 = 180 \text{ mg/min}$ **Q13.3: What is the significance of T_m in glucose reabsorption?****Answer:** T_m = maximum reabsorptive capacity. Here, $T_m = 150 \text{ mg/min} \rightarrow$ so only 150 mg is reabsorbed**Q13.4: How much glucose appears in urine?****Answer:** $180 - 150 = 30 \text{ mg/min}$ **BIG QUESTION 14: The prime driving force for counter current multiplier system is:**

- A. Reabsorption of Na^+ in thick ascending limb
- B. Action of ADH via Aquaporin channels
- C. Urea recycling
- D. Medullary hyperosmolarity

Broad Answer:

Answer: A. Reabsorption of Na^+ in thick ascending limb

The main site of Counter current multiplier system is TAL



At the level of TAL → NKCC transporter is present



NKCC reabsorbs Sodium and Chloride molecule

DETAILED QUESTIONS

Q14.1: What is the role of the thick ascending limb in concentration?

Answer: Reabsorbs Na^+ , K^+ , Cl^- via NKCC; impermeable to water → sets up gradient

Q14.2: What does this gradient do?

Answer: Allows water reabsorption from the descending limb and collecting duct (when ADH is present)

BIG QUESTION 15: Which of the following is not a component of Counter current multiplier system?

- A. Thick ascending loop of Henle
- B. Collecting duct
- C. Vasa recta
- D. Thin ascending loop of Henle

Broad Answer:

Answer: C. Vasa recta

Vasa recta is part of the counter current exchanger, not the multiplier system.

DETAILED QUESTIONS

Q15.1: What is the difference between multiplier and exchanger?

Answer:

- Multiplier = creates medullary gradient (loop of Henle)
- Exchanger = maintains gradient (vasa recta)

Q15.2: What are the components of the multiplier?

Answer:

- Thin descending limb
- Thin ascending limb
- Thick ascending limb

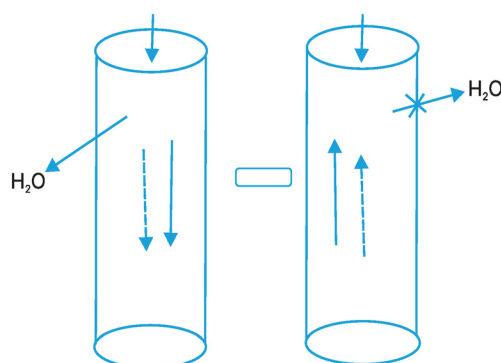
BIG QUESTION 16: What are the essential criteria for effective counter-current flow?

Broad Answer: It has four criteria:

- Two parallel tubes

 PYQ: NEET PG 2018

- Opposite direction of fluid movement
- Close proximity of tubes
- Selectively permeable



DETAILED QUESTIONS

Q16.1: In which locations within the body is a counter-current arrangement typically found?

Answer:

- Intestinal villus
- Skin
- Testis
- Kidneys
- Lungs

Q16.2: What are the two main components of the counter-current system in the kidney?

Answer:

- **Counter current exchanger**
 - Occurs at the level of Vasa recta
- **Counter current multiplier:** Occurs at the level of:
 - **Loop of Henle**
 - Thick ascending loop of Henle (main site) → Sodium and Chloride reabsorption occurs
 - Ascending thin segment
 - **Medullary Collecting Duct (MCD)**
 - Urea re-absorption occurs from MCD

Q16.3: What is the primary function of the counter-current system?

Answer: Counter-current system is responsible for increasing the tonicity of the medulla → Increased reabsorption of water from urine → Concentrated urine.

- **Counter-current Multiplier:** Generation of hypertonicity of the Medulla
- **Counter-current exchanger:** Maintenance of the hypertonicity of medulla
 - Substances that causes hypertonicity of medulla - Na , Cl , Urea

Q16.4: What is the maximum medullary tonicity that can be achieved in the kidney?

Answer: 1200 to 1400 mOsm/L:

- 50 to 60 %: Sodium and chloride
 - 600 mOsm/L: By NaCl
 - If NaCl is separated → Contributions are: Na = 300 mOsm/L; Cl = 300 mOsm/L

- 40 to 50 %: Urea alone
 - 600 mOsmol/L: Urea

Q16.5: Which substance plays the most important role in maintaining medullary tonicity?

Answer:

- Urea → If Na, Cl and urea are given separately
- NaCl → If NaCl and urea are given

Q16.6: What are the key differences between SGLT1 and SGLT2 transporters?

Answer:

SGLT1	SGLT2
Present in PCT	Present in PST
10% glucose reabsorption	Major glucose reabsorption
Absorbs 1 molecule of glucose and 2 molecules of sodium	Absorbs 1 molecule of sodium and 1 molecules of glucose
Coupled with GLUT1	Coupled with GLUT2



8. ENDOCRINE & REPRODUCTIVE PHYSIOLOGY

BIG QUESTION 1: What are the different types of cell signaling mechanisms in the endocrine system, and how are hormones classified based on their receptors?

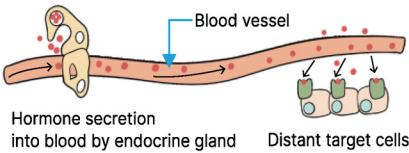
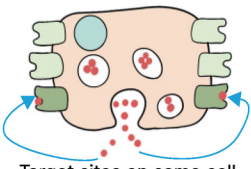
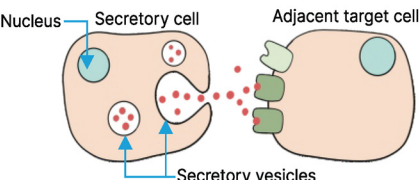
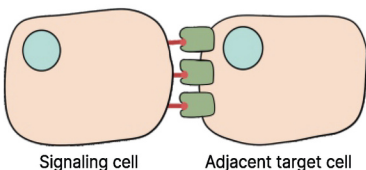
Broad Answer:

- Cell signaling in the endocrine system includes endocrine, paracrine, autocrine, and juxtacrine mechanisms, depending on the distance and mode of target interaction. Hormones are classified based on their receptors into:
 - Membrane-bound receptors:** GPCRs, Guanylyl cyclase, Tyrosine kinase, Cytokine, Serine kinase receptors.
 - Intracellular receptors:** Cytoplasmic or nuclear, depending on the hormone's solubility.

DETAILED QUESTIONS

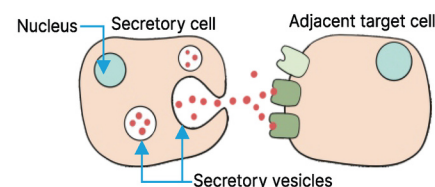
Q1.1: What are the different types of cell signaling mechanisms?

Answer:

Type of Signaling	Description	Example
Endocrine  Hormone secretion into blood by endocrine gland Distant target cells	Secretory products enter the bloodstream and act on distant target cells	Most hormones (e.g., insulin, cortisol)
Autocrine  Target sites on same cell	Secretory product acts on the same cell from which it is secreted.	Platelet-activating factor
Paracrine  Nucleus Secretory cell Adjacent target cell Secretory vesicles	Secretory product acts on neighboring cells	Histamine from ECL cells stimulates parietal cells
Juxtacrine  Signaling cell Adjacent target cell	Ligand of one cell interacts with receptor of adjacent cell (requires close proximity).	Transforming growth factor- α (TGF- α)

Q1.2: Which of the following cell signaling is shown in the given picture?"

- A. Autocrine secretion
- B. Paracrine secretion
- C. Merocrine secretion
- D. Endocrine



Answer: B. Paracrine secretion

- The secretory product from one cell acting on nearby cells is characteristic of **Paracrine** signaling.

Q1.3: How are hormone receptors broadly classified?

🔍 PYQ: FMGE 2022 🔍 PYQ: FMGE 2023 🔍 PYQ: AIIMS 2020

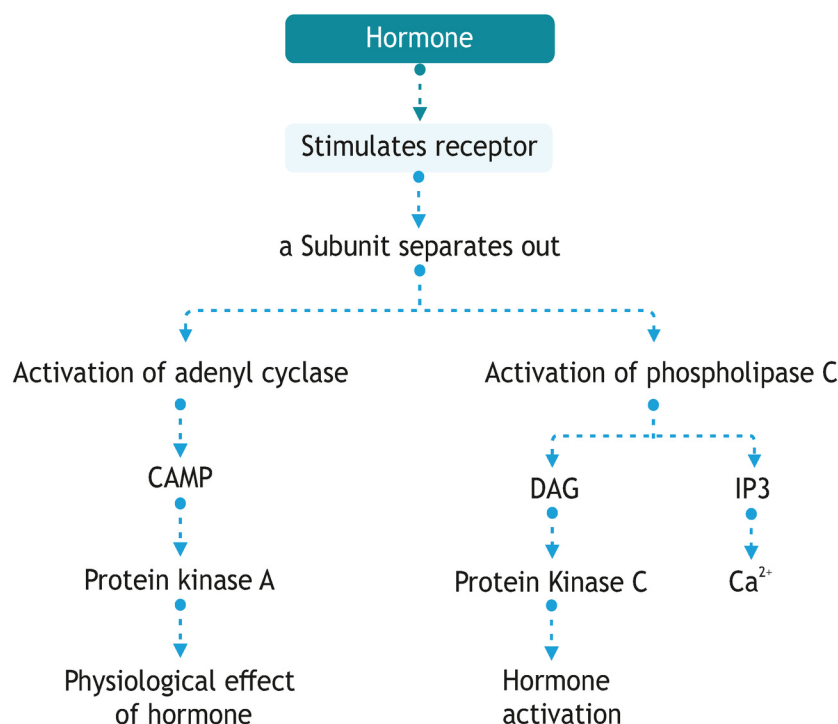
Answer: Hormone receptors are classified into:

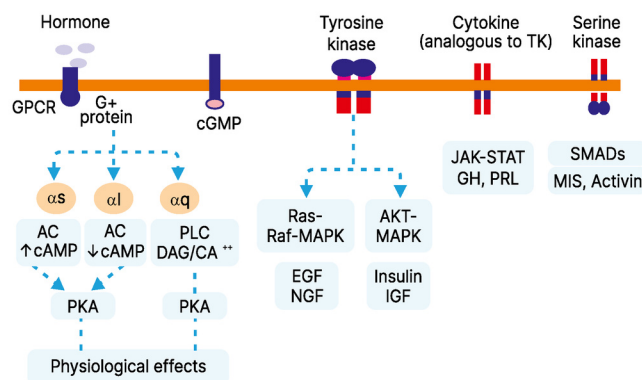
Membrane-bound receptors	Intracellular receptors
• G-protein coupled receptors (GPCRs) • Guanylyl cyclase receptors • Tyrosine kinase receptors • Cytokine receptors • Serine kinase receptors	• Cytoplasmic: Glucocorticoid, mineralocorticoid, estrogen, androgen, progesterone • Nuclear: Thyroid hormone, Vitamin D, Retinoic acid receptor (RAR, RXR).

BIG QUESTION 2: How do G-protein coupled receptors and their subtypes (Gs, Gi, Gq) mediate hormonal signaling pathways?

Broad Answer:

- G-protein coupled receptors (GPCRs) have α , β , and γ subunits. Upon hormone binding, the α -subunit activates intracellular effectors:
 - **Gs (Stimulatory)** → Activates Adenylyl Cyclase → $\uparrow$ cAMP
 - **Gi (Inhibitory)** → Inhibits Adenylyl Cyclase → $\downarrow$ cAMP
 - **Gq** → Activates Phospholipase C → IP₃ & DAG → $\uparrow$ Ca²⁺ and PKC activation
- Each subtype modulates specific second messengers and physiological responses depending on the hormone.





DETAILED QUESTIONS

Q2.1: What are the components and basic mechanism of GPCRs?

Answer:

- GPCRs are membrane-bound receptors associated with a heterotrimeric G-protein composed of α , β , and γ subunits.
- On hormone binding:
 - The α -subunit dissociates
 - It activates downstream effector enzymes (Adenylyl cyclase or Phospholipase C)
 - This leads to generation of second messengers (cAMP, IP₃, DAG)

Q2.2: What is the G_s pathway, and which hormones use it?

Answer:

Effector	G _s → Adenylyl Cyclase (AC)
Second Messenger	↑ cAMP
Downstream Target	Protein kinase A
Hormones	ACTH, TSH, LH, FSH, CRH, PTH, MSH, Glucagon, Calcitonin, ADH (V2 receptor), β -adrenergic

Q2.3: What is the G_i pathway, and which hormones use it?

Answer:

Effector	G _i → Inhibits Adenylyl Cyclase (AC)
Second Messenger	↓ cAMP
Hormones	Somatostatin, Angiotensin II, α 2-adrenergic, Acetylcholine (M2, M4)

Q2.4: What is the G_q pathway, and which hormones use it?

Answer:

Effector	G _q → Activates Phospholipase C (PLC)
Second Messenger	↑ IP ₃ → ↑ Ca ²⁺ ↑ DAG → Activates Protein Kinase C
Hormones	TRH, GnRH, GHRH, Oxytocin, ADH (V1 receptor), α 1-adrenergic, Acetylcholine (M1, M3, M5)

Q2.5: What is the final common pathway of GPCR-mediated signaling?

Answer:

- Hormone binds → Receptor activates G-protein
- α -subunit dissociates
- Activates either Adenylyl Cyclase (AC) or Phospholipase C (PLC)
- Second messengers generated (cAMP or IP₃/DAG)
- Kinase cascades initiated (PKA or PKC)
- Result: Alteration in gene expression or cell function.

BIG QUESTION 3: What are the other major membrane-bound hormone receptor classes besides GPCR, and how do they function? (Guanylyl, Tyrosine kinase, Cytokine, Serine kinase)

Broad Answer:

- Other major classes of membrane-bound hormone receptors include:
 - **Guanylyl cyclase receptors** → produce **cGMP** (e.g., ANF, NO)
 - **Tyrosine kinase receptors** → activate **RAS/RAF/MAPK** or **AKT/MAPK** pathways (e.g., insulin, IGF)
 - **Cytokine receptors** → activate **JAK-STAT** pathway (e.g., GH, PRL)
 - **Serine kinase receptors** → activate **SMAD** pathway (e.g., MIS, Activin)
- Each receptor type has a specific intracellular signaling mechanism without involving classical G-protein subunits.

DETAILED QUESTIONS

Q3.1: How do guanylyl cyclase receptors function, and what hormones use them?

Answer:

- These receptors contain intrinsic guanylyl cyclase activity.
- Upon hormone binding, they convert GTP to cGMP.
- cGMP acts as the second messenger.
- Examples:
 - Atrial natriuretic factor (ANF)
 - Nitric oxide (NO)

Q3.2: What are the pathways activated by tyrosine kinase receptors and the hormones involved?

Answer:

- Tyrosine kinase receptors auto-phosphorylate upon ligand binding.
- They activate two pathways:
 - **RAS/RAF/MAPK** → Used by Epidermal Growth Factor (EGF), Nerve Growth Factor (NGF)
 - **AKT/MAPK** → Used by Insulin, Insulin-like Growth Factor (IGF)

Q3.3: How do cytokine receptors signal and what hormones use this pathway?

Answer:

- Cytokine receptors lack intrinsic kinase activity.
- They are associated with intracellular **JAK (Janus kinase)** proteins.
- Ligand binding causes activation of JAK → phosphorylation of STAT (Signal Transducer and Activator of Transcription)

YOURWISH

- STAT translocates to the nucleus to affect gene expression.
- Examples:
- Growth Hormone (GH)
- Prolactin (PRL)

Q3.4: What is the signaling mechanism of serine kinase receptors, and what hormones act through them?

Answer:

- Ligand binding leads to phosphorylation of serine residues.
- This activates the **SMAD pathway**.
- Examples:
- Mullerian Inhibiting Substance (MIS)
- Activin

Q3.5: How do these non-PCR membrane receptors differ functionally from GPCRs?

Answer:

Feature	GPCRs	Non-GPCRs (e.g., Tyrosine Kinase)
Effector activation	Via G-protein	Direct kinase activity or associated kinases
Second messenger involvement	Yes (cAMP, IP ₃ , DAG)	May or may not use second messengers
Time of response	Rapid	Rapid to intermediate
Examples	LH, FSH, ACTH	Insulin, GH, ANF

BIG QUESTION 4: What are the actions, regulation, and physiological effects of growth hormone and IGF-1?

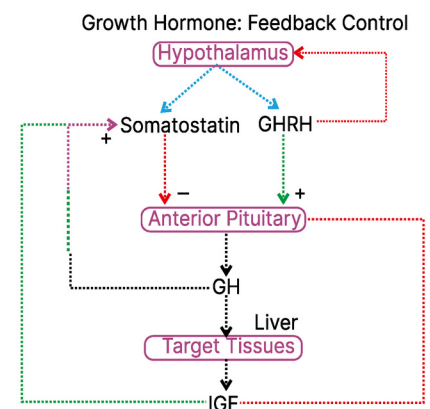
🔍 PYQ: NEET PG 2018

🔍 PYQ: AIIMS 2018

🔍 PYQ: FMGE 2024

Broad Answer:

- Growth hormone (GH) has both **direct** and **indirect** actions (via IGF-1).
- It plays a vital role in **metabolism, growth, and homeostasis**:
 - **Direct effects:** GH acts on liver and other tissues to alter metabolism
 - **Indirect effects:** GH stimulates IGF-1, promoting linear growth, protein synthesis, and organ growth



DETAILED QUESTIONS

Q4.1: What are the direct actions of GH on metabolism?

Answer:

- ↓ Glucose uptake → ↑ Blood glucose (anti-insulin effect)
- ↑ Lipolysis → ↑ Free fatty acids
- ↑ Protein synthesis in all tissues → ↑ Lean body mass
- ↑ Insulin resistance
- ↑ Na⁺, K⁺, and water retention

Q4.2: What are the indirect actions of GH via IGF-1?

Answer:

- IGF-1 (Insulin-like Growth Factor-1), primarily produced by liver in response to GH, mediates anabolic and growth-promoting effects:
 - o ↑ Protein synthesis in chondrocytes → ↑ Linear bone growth
 - o ↑ Protein synthesis in muscles → ↑ Lean body mass
 - o Anti-lipolysis
 - o ↑ Organ growth

Q4.3: How is growth hormone secretion regulated?

Answer:

- **Stimulators of GH secretion:**
 - o Hypoglycemia (e.g., fasting, exercise)
 - o Increased amino acids (e.g., protein meal)
 - o Stress (e.g., surgery, trauma)
 - o NREM sleep (stimulatory)
- **Inhibitors of GH secretion:**
 - REM sleep
 - Hyperglycemia
 - Somatostatin

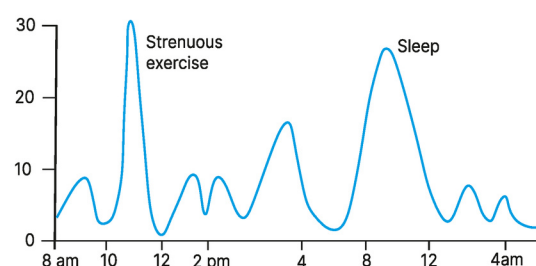
Q4.4: What is the pattern of GH secretion throughout the day?

Answer:

- GH secretion is **pulsatile** and **highest during NREM sleep**.
- GH secretion varies over 24 hours.
- A diagram showing GH peaks after NREM sleep is diagnostic.

Q4.5: Serum level of one hormone X is recorded throughout the day. Identify the hormone X from the picture.

- A. Growth hormone
- B. Cortisol
- C. Estrogen
- D. Insulin



Answer: A. Growth Hormone

- GH peaks during NREM sleep
- Estrogen, insulin, cortisol do not follow this pattern

Q4.6: What is the clinical relevance of GH in different physiological and pathological states?

Answer:

- GH deficiency → Dwarfism in children
- GH excess (before puberty) → Gigantism
- GH excess (after puberty) → Acromegaly

- GH therapy used in GH-deficient states
- IGF-1 testing often reflects GH activity due to its longer half-life

BIG QUESTION 5: How is ADH involved in water regulation, and how can different types of diabetes insipidus be differentiated?

Broad Answer:

- ADH (antidiuretic hormone) is secreted by the posterior pituitary and acts on V2 receptors of collecting duct cells, promoting water reabsorption.
- ADH secretion is primarily regulated by plasma osmolality and blood volume.
- **Diabetes insipidus (DI)** is classified into:
 - **Central DI:** Impaired ADH secretion
 - **Nephrogenic DI:** Impaired ADH response at V2 receptors
 - These can be differentiated using ADH challenge tests and urine osmolality responses.

DETAILED QUESTIONS

Q5.1: What is the mechanism of ADH action in water regulation?

Answer:

- ADH binds to **V2 receptors** on **collecting duct cells**
- Increases **aquaporin-2** channel insertion
- $\uparrow$ Water reabsorption $\rightarrow$ $\downarrow$ Urine output
- Secreted in response to $\uparrow$ Plasma osmolality or $\downarrow$ Blood volume

Q5.2: How is plasma osmolality related to ADH secretion?

Answer:

- Most potent stimulus: $\uparrow$ Plasma osmolality (>280 mOsmol/L)
- $\uparrow$ Osmolality $\rightarrow$ $\uparrow$ ADH secretion $\rightarrow$ $\uparrow$ Water reabsorption
- $\downarrow$ Osmolality $\rightarrow$ $\downarrow$ ADH $\rightarrow$ $\uparrow$ Urine volume

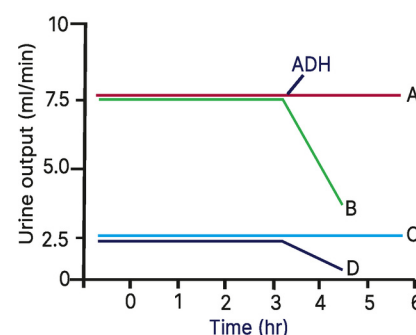
Q5.3: How does ADH affect urine output in central vs. nephrogenic DI?

Answer:

Type of DI	Problem Site	ADH Level	ADH Response	Urine Output after ADH Injection
Central	Hypothalamus/Pituitary	$\downarrow\downarrow$ (Low)	Normal	$\downarrow$ (Improves)
Nephrogenic	Kidney (V2 Receptor)	Normal/High	Defective	No change (Persists)

Q5.4: An experiment is conducted in which ADH is administered to four subjects (A to D) at hour 3. In the figure below, which line most likely reflects the response in central DI?

- A. A
- B. B
- C. C
- D. D

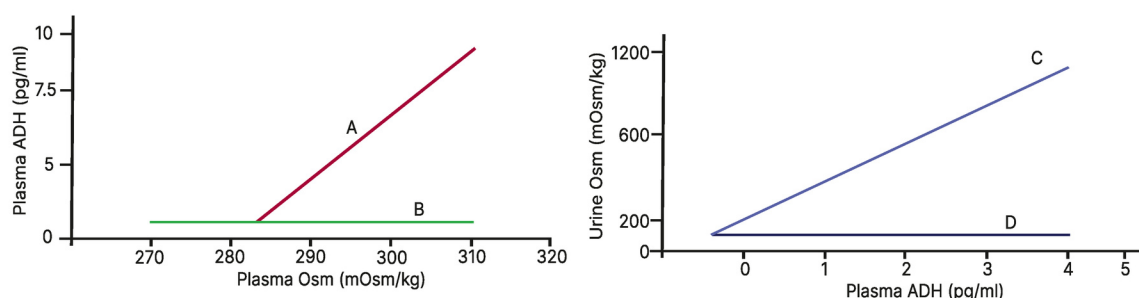


Answer: B, B

- ADH acts on V2 receptors in collecting ducts $\rightarrow \uparrow$ water reabsorption $\rightarrow \downarrow$ urine output.
- A & B: High urine output $\rightarrow$ likely DI.
- C & D: Low urine output $\rightarrow$ likely normal.
- Nephrogenic DI (A): ADH injection has no effect (V2 receptor defect) $\rightarrow$ urine output remains high.
- Central DI (B): Problem is lack of ADH secretion, but receptors are intact $\rightarrow$ ADH injection causes sharp $\downarrow$ in urine output.
- D: Normal - ADH causes moderate $\downarrow$ in urine output.
- So, B = Central DI (responds to ADH)

Q5.5: In the figure below, which lines most likely reflect the responses in nephrogenic DI?

- A. A and C
- B. A and D
- C. B and C
- D. B and D



Answer: B, A and D

- ADH secretion is stimulated by plasma osmolality > 280 mOsm/kg.
- Curve A: Normal or nephrogenic DI - ADH increases normally with rising osmolality.
- Curve B: Central DI - ADH fails to rise despite high osmolality (problem in secretion).
- Curve D: Nephrogenic DI - Urine osmolality remains low despite high ADH (V2 receptor defect).

Q5.6: How can you interpret urine volume and ADH curve analysis in suspected DI?

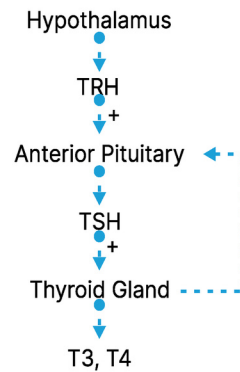
Answer:

- If ADH is low, and urine volume high $\rightarrow$ Central DI
- If ADH is high, but urine volume remains high $\rightarrow$ Nephrogenic DI
- ADH challenge test helps differentiate:
 - $\downarrow$ Urine output post-ADH $\rightarrow$ Central
 - No change $\rightarrow$ Nephrogenic

BIG QUESTION 6: How is thyroid hormone synthesized, transported, and regulated, and what are its physiological forms and actions?

Broad Answer:

- Thyroid hormones T4 (thyroxine) and T3 (triiodothyronine) are secreted by the thyroid gland.
 - T4 is the major product; T3 is the active form.
 - Most T4 is converted to T3 peripherally by deiodinase (mainly in liver, kidney).
 - Thyroid hormones are lipophilic and travel in blood bound to thyroxine-binding globulin (TBG), transthyretin (thyroxin binding prealbumin), and albumin.
 - T3 is more potent, has faster action, and binds better to thyroid hormone receptors than T4.
 - Their secretion is regulated via the hypothalamic-pituitary-thyroid axis.



DETAILED QUESTIONS

Q6.1: What is the major secretory product of the thyroid, and how is the active hormone formed?

Answer:

- T4 is the major secretion (80 µg/day; 93% of total).
- Converted to T3 in peripheral tissues (mainly liver and kidney) by deiodinase.
- T3 is the most biologically active form.

Q6.2: How are thyroid hormones transported in the plasma?

Answer:

Hormone	Binding Proteins	Main Carrier
T4	TBG > Transthyretin > Albumin	TBG
T3	Albumin > TBG	Albumin

- Thyroid hormones are **lipophilic**, hence require binding proteins for transport in plasma.

Q6.3: What are the differences between T3 and T4?

Answer:

PYQ: FMGE 2020, 2022

Feature	T4	T3
Secretion	More (80 µg/day; 93%)	Less (4 µg/day; 7%)
Plasma Concentration	8 ng/dL	0.15 ng/dL
Half-life	Longer (5-7 days)	Shorter (1 day)
Action	Slower	Faster
Potency	Less	3-5 times more potent
Receptor Binding Affinity	Lower	Higher

Q6.4: What regulates thyroid hormone secretion?

Answer:

- Regulated via negative feedback loop:
 - Hypothalamus secretes TRH → stimulates pituitary to release TSH → stimulates thyroid to release T4/T3.
 - ↑T4/T3 → ↓TRH and TSH via negative feedback.

BIG QUESTION 7: What are the types of hypothyroidism and hyperthyroidism, and how can they be differentiated clinically and biochemically?

Broad Answer:

- Thyroid disorders are classified based on the site of dysfunction:
 - Hypothyroidism:** Primary (thyroid), Secondary (pituitary), Tertiary (hypothalamus)
 - Hyperthyroidism:** Primary (thyroid), Secondary (pituitary), Tertiary (hypothalamus - rare)
- Diagnosis is based on hormone levels and response to stimulation tests like **TRH**.

DETAILED QUESTIONS

Q7.1: What are the features of primary hypothyroidism?

Answer:

- Problem lies in the **thyroid gland**.
- $\downarrow T3/T4 \rightarrow \downarrow$ negative feedback $\rightarrow \uparrow TSH$ and $\uparrow TRH$
- Example: **Hashimoto's thyroiditis**

Q7.2: How is secondary hypothyroidism characterized biochemically?

Answer:

- Problem lies in the **pituitary gland**.
- $\downarrow TSH \rightarrow \downarrow T3/T4$
- TRH increases but has no effect due to pituitary dysfunction.
- TSH does not rise** after TRH administration.

Q7.3: What is tertiary hypothyroidism and how is it diagnosed?

Answer:

- Problem lies in the **hypothalamus**.
- $\downarrow TRH \rightarrow \downarrow TSH \rightarrow \downarrow T3/T4$
- TSH rises** after TRH administration $\rightarrow$ confirms tertiary defect.

Q7.4: How are different types of hyperthyroidism classified and identified?

Answer:

 PYQ: FMGE 2021

 PYQ: NEET PG 2022

Type	Pathology	T4	TSH	TRH
Primary	Thyroid	$\uparrow\uparrow$	$\downarrow$	$\downarrow$
Secondary	Pituitary	$\uparrow$	$\uparrow\uparrow$	$\downarrow$
Tertiary (very rare clinically)	Hypothalamus	$\uparrow$	$\uparrow$	$\uparrow\uparrow$

Q7.5: A person presents with puffy skin and is lethargic. His TSH levels are low and are improved on administration of TRH. What is the most probable diagnosis?

- Hypothyroidism because of hypothalamus defect
- Diffuse thyroid enlargement
- Hyperthyroidism because of hypothalamus defect
- Hypothyroidism because of pituitary defect

Answer: A. Hypothyroidism because of hypothalamus defect

- Puffy skin + lethargy → hypothyroidism.
- TRH improves TSH → Tertiary hypothyroidism (hypothalamic defect).
- If it were secondary, TSH would not rise after TRH.

BIG QUESTION 8: How do thyroid hormone levels change during starvation, and what does this imply about T3 and rT3 roles?

Broad Answer:

- During starvation, the body downregulates active thyroid hormone (T3) to reduce metabolic rate and conserve energy.
 - o T3 decreases
 - o Reverse T3 (rT3) increases
 - o T4 remains relatively unchanged
- This adaptive mechanism reduces basal metabolic rate (BMR) to prevent energy loss.

DETAILED QUESTIONS

Q8.1: What is the major secretory product of the thyroid gland?

Answer:

- T4 is the major secretory product of the thyroid gland.

Q8.2: How are T3 and rT3 formed, and what are their respective roles?

Answer:

- T4 is converted to T3 (active form) and rT3 (inactive form) in peripheral tissues.
- T3 increases BMR
- rT3 does not increase metabolic rate (inactive)

Q8.3: What happens to thyroid hormone levels during starvation?

Answer:

- T3 decreases to reduce BMR
- rT3 increases as part of energy conservation
- T4 remains stable

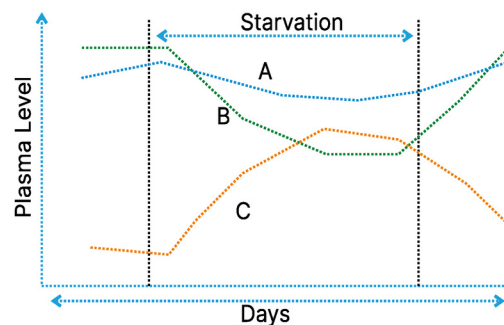
Q8.4: How is this shift beneficial during periods of starvation?

Answer:

- ↓ T3 prevents excessive energy use
- ↑ rT3 prevents conversion to metabolically active forms
 - o Overall adaptation to reduce metabolism during nutrient scarcity

Q8.5: The effect of different thyroid hormones in plasma during starvation is shown in the following curve. Curve B indicates which of the following?

- A. DIT
- B. rT3
- C. T3
- D. T4



Answer: C. T3

- Curve B: T3 - decreases during starvation
- Curve C: rT3 - increases during starvation
- Curve A: T4 - remains stable

BIG QUESTION 9: What are the zones of the adrenal gland, their hormonal outputs, and the regulation of cortisol secretion?

Broad Answer:

- The adrenal gland has a cortex and medulla, with the cortex further divided into three zones, each producing distinct hormones under different regulatory controls.
 - o **Zona Glomerulosa** → Mineralocorticoids (Aldosterone) → Regulated by **Angiotensin II**
 - o **Zona Fasciculata** → Glucocorticoids (Cortisol) → Regulated by **ACTH**
 - o **Zona Reticularis** → Sex steroids (DHEA, androstenedione) → Regulated by **ACTH**
 - o **Medulla** → Catecholamines (Epinephrine 90%, Norepinephrine 10%) → Regulated by **Sympathetic nervous system**

DETAILED QUESTIONS

Q9.1: What are the structural and hormonal divisions of the adrenal gland?

PYQ: AIIMS 2019

Answer:

PYQ: FMGE 2020

Layers (weight)	Hormone production	Main regulator
Cortex (72%)	Zona Glomerulosa (15%) • Mineralocorticoids: - o Aldosterone (main) - o Deoxycorticosterone	Angiotensin II
	Zona Fasciculata (50%) • Glucocorticoids (main) - o Cortisol - o Corticosterone • Sex steroids	ACTH
	Zona Reticularis (7%) • Sex steroid (main) - o DHEA - o Androstenedione (main) • Glucocorticoids	ACTH

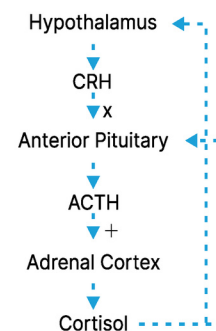
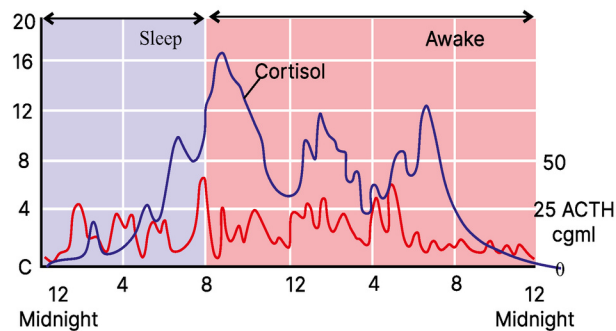
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Medulla (28%)	• Epinephrine (90%) • Norepinephrine (10%)	Sympathetic nervous system
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Q9.2: How is cortisol secretion regulated and what is its pattern?

Answer:

- Cortisol secretion is **pulsatile** and follows **diurnal variation**
- Within 24 hours of duration, secretion will peak at a point of time and secretion will decrease at another point of time
- **Peak secretion:** Morning (~8:30 AM)
- **Lowest level:** Midnight (~12:15 AM)
- ACTH regulates cortisol and follows the same pattern.



Q9.3: What is the feedback mechanism controlling cortisol secretion?

Answer:

- Cortisol exerts negative feedback on:
 - o Anterior pituitary → ↓ ACTH secretion
 - o Hypothalamus → ↓ CRH secretion

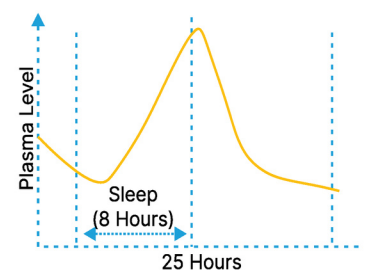
Q9.4: What is the clinical relevance of cortisol's diurnal variation?

Answer:

- Blood samples for cortisol should be collected in the early morning for accurate interpretation.
- Loss of diurnal rhythm (e.g., in Cushing's syndrome) is a diagnostic clue.

Q9.5: A 52 year old woman with a chief complaint of snoring is referred for a sleep study. As shown in the graph below, the concentration of a hormone varied over the 24 hour period of study. This diurnal variation in plasma level results from the secretion of which of the following hormones?

- Cortisol
- Oestrogen
- Insulin
- Thyroxine



Answer: A. Cortisol

BIG QUESTION 10: How is calcium homeostasis maintained through the actions of PTH, vitamin D, and calcitonin?

Broad Answer:

- Calcium homeostasis is regulated by three key hormones—**Parathyroid Hormone (PTH)**, **Vitamin D (Calcitriol)**, and **Calcitonin**.
- These hormones act on **bone**, **kidneys**, and **intestines** to maintain appropriate serum calcium and phosphate levels.

DETAILED QUESTIONS

Q10.1: What stimulates the secretion of PTH and what are its major actions?

Answer:

- **Stimuli for secretion:**
 - o ↓ Serum calcium
 - o ↑ Serum phosphate
 - o Hypomagnesemia
- **Actions of PTH:**
 - o Bone: ↑ Resorption → ↑ Ca^{2+} and PO_4^{3-} release
 - o Kidney:
 - ↑ Ca^{2+} reabsorption
 - ↓ PO_4^{3-} reabsorption
 - ↑ 1α -hydroxylase activity → activates Vitamin D
 - o Intestine (via Vitamin D): ↑ Ca^{2+} absorption
- **Net effect:**
 - o ↑↑ Serum Ca^{2+}
 - o ↓↓ Serum PO_4^{3-}

Q10.2: How does vitamin D contribute to calcium and phosphate regulation?

Answer:

- **Stimuli for secretion (activation):**
 - o ↓ Serum calcium
 - o ↓ Serum phosphate
- **Actions of Vitamin D (Calcitriol):**
 - o Bone: ↑ Resorption → ↑ Ca^{2+} and PO_4^{3-}
 - o Kidney:
 - ↑ Ca^{2+} reabsorption
 - ↑ PO_4^{3-} reabsorption
 - o Intestine:
 - ↑ Ca^{2+} and PO_4^{3-} absorption
- **Net effect:**
 - o ↑ Serum Ca^{2+}
 - o ↑ Serum PO_4^{3-}

Q10.3: What is the role of calcitonin in calcium homeostasis?

Answer:

- **Stimulus for secretion:**
 - o ↑ Serum calcium

- **Actions:**
 - o Bone: ↓ Osteoclastic activity → ↓ Resorption
 - o Kidney and intestine: Minor roles
- **Net effect:**
 - o ↓↓ Serum Ca^{2+}

BIG QUESTION 11: What is the role of leptin in puberty onset and how does it interact with the reproductive axis?

Broad Answer:

- Leptin, secreted by white adipose tissue, plays a **permissive role** in the onset of puberty.
- It regulates hypothalamic GnRH pulsatility and supports reproductive axis activation.
- While leptin alone does not initiate puberty, its presence is essential for normal hypothalamic-pituitary-gonadal (HPG) axis function.

DETAILED QUESTIONS

Q11.1: Where is leptin produced and what regulates its secretion?

Answer:

- **Produced by:** White adipose tissue
- **Secretion increases with:**
 - o Greater fat mass (obesity)
 - o Nutritional adequacy
 - o Energy sufficiency

Q11.2: How does leptin act on the hypothalamus to influence puberty?

Answer:

- Leptin exerts a permissive effect on GnRH secretion.
- It regulates circadian rhythms of gonadotropin secretion.
- Leptin increases pulsatile GnRH release, facilitating the activation of the HPG axis.

Q11.3: What are the central effects of leptin besides reproductive regulation?

Answer:

- Sends anorexigenic signals (promotes satiety) to the hypothalamus.
- Decreases fat storage.
- Stimulates thermogenesis to increase energy expenditure.

Q11.4: What happens in the absence of leptin?

Answer:

- Delayed or absent puberty
- Infertility
- Failure to activate GnRH pulsatility
- Seen in conditions of leptin deficiency or extreme undernutrition

Q11.5: Which hormone has permissive role in puberty:

- A. Leptin
- B. GnRH
- C. Insulin
- D. GH

Answer: A. Leptin

- Pulsatile secretion of GnRH hormone causes onset of puberty
- Before the onset of puberty, certain factors causes inhibition of GnRH hormones- Pulsatility is not seen

BIG QUESTION 12: How do the hypothalamic-pituitary-gonadal axes regulate reproductive function in males and females?

Broad Answer:

- The hypothalamic-pituitary-gonadal (HPG) axis regulates reproductive function through the coordinated release of hormones from the hypothalamus (GnRH), anterior pituitary (LH, FSH), and gonads (testosterone, estrogen, progesterone).
- In males, Sertoli and Leydig cells respond to FSH and LH, respectively.
- In females, theca and granulosa cells collaborate in steroid production, with hormonal feedback mechanisms governing ovulation and menstrual cycles.

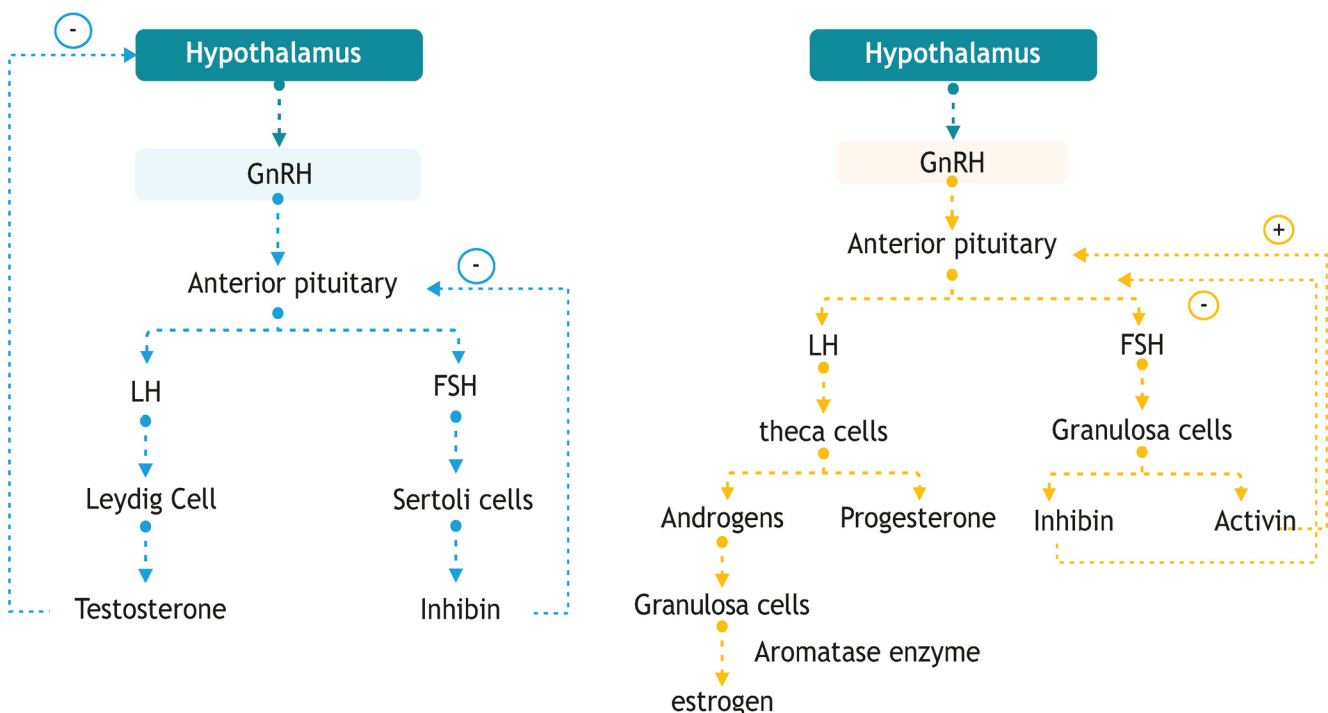
DETAILED QUESTIONS

Q12.1: What is the function of the hypothalamus in the HPG axis?

PYQ: INICET 2020

Answer:

- Secretes GnRH (Gonadotropin-releasing hormone) in a pulsatile manner.
- Stimulates anterior pituitary to secrete LH and FSH.



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Q12.2: What is the role of Sertoli and Leydig cells in male reproductive physiology?

Answer:

Cell Type	Key Features
Sertoli Cells	• Form blood-testis barrier • Respond to FSH and androgens • Nourish sperm • Produce Inhibin, ABP, AMH • Express aromatase to convert testosterone to estrogen
Leydig Cells	• Respond to LH • Synthesize testosterone • Contain 3β-HSD to convert androstenedione to testosterone • Acquire cholesterol via LDL/HDL receptors

Q12.3: How is testosterone synthesized and utilized in male reproductive physiology?

Answer:

- Synthesized by Leydig cells using cholesterol.
- Testosterone diffuses into seminiferous tubules.
- Converted to estradiol by aromatase in Sertoli cells.
- Supports spermatogenesis and development of secondary sexual characteristics.

Q12.4: How do theca and granulosa cells contribute to estrogen production in females?

Answer:

- Theca cells: Produce androgens under LH stimulation.
- Granulosa cells: Convert androgens to estrogens via aromatase under FSH stimulation.
- Both cell types are essential for estrogen and progesterone synthesis and ovarian function.

Q12.5: What is the timeline and hormonal control of ovulation?

Answer:

- Ovulation occurs ~14 days after menstruation onset in a 28-day cycle.
- Timelines relative to hormonal events:

Reference Point	Time to Ovulation
Estradiol rise	~83 hours
Estradiol peak	24-36 hours
LH surge onset	24-36 hours
LH peak	9-12 hours
Progesterone rise	~8 hours

- Feedback inhibition of LH by estrogen and progesterone helps regulate the menstrual cycle