Questions & Answers in PHYSIOLOGY

(for MBBS, BDS, BMS, BHMS)

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Foreword
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JAYPEE

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CHAPTER 4

Respiratory System

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Q. 1. State the name and location of respiratory centers. Describe their role in normal breathing.

Ans.

Nucleus parabrachialis , Kolliker fuse nuclei	Pneumotaxic center:	upper pons
	Apneustic center	lower border of pons
Expiratory center near Nucleus Ambiguus and Nucleus Retroambiguus	Ventral respiratory group	Medulla
Inspiratory center near Nucleus Tractus Solitarius	Dorsal respiratory group	Medulla

PreBotzinger complex between Nucleus ambiguus and lateral reticular nucleus is believed to be the pacemaker for the respiratory group if neurons.

Nervous regulation of respiration

1. Automatic control of respiration by medulla and pons
2. Voluntary control of respiration by cortex.

1. Automatic control of respiration by medulla and pons

Medulla: The I neurons provide rhythmic drive to phrenic motor neurons. They receive vagal afferents from airways and chemoreceptors. The I neurons and E neurons are reciprocally innervated. The connection is inhibitory in nature. So, any impulse that stimulates one will inhibit the other. The medulla is the respiratory center or the switch box.

Pons: Neurons tonically active in the pons constitute the apneustic center. They activate I neurons. If vagus nerve are cut then arrest of respiration will occur or **Apneusis.** If this area is damaged respiration becomes slower and tidal volume greater,

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there are prolonged inspiratory spasms that resembles breath holding.

Pneumotaxic neurons inhibit apneustic center and inhibit apneusis. They are active both in expiration and inspiration. They produce rhythmic breathing by switching between inspiration and expiration.

The medulla discharge of I and E neurons are intrinsically automatic and spontaneous. These are modified by the neurons in the pons and vagi nerves.

2. Voluntary control of respiration

Respiration can be modified both in rate and depth at will for a specific period, e.g. breath holding. The pathway for such control is corticospinal tract originating in the cerebral cortex to end on spinal motor neurons innervating the respiratory group of neurons, by passing the medullary group of neurons.

Q. 2. Describe the role of pulmonary stretch receptor in regulation of respiration:

Pulmonary stretch receptors: The pulmonary stretch receptors **Ans.** In the lungs get stimulated during inspiration. These send inhibitory impulses via vagus to apneustic center.

The pneumotaxic center stimulates the expiratory center, which reciprocally inhibits the inspiratory center. This can be simulated by two men slapping each other.

Apneustic center gets inhibited and ceases to activate the inspiratory center as a result the inspiratory center stops discharging and expiration follows passively.

These are also J receptors or Juxtacapillary receptors, because of the nearness to pulmonary capillaries. They are responsive to pulmonary edema, hyperinflation of the lung or IV administration of capsaicin. They cause apnea followed by rapid breathing, bradycardia and hypotension. These receptors were brought into action at the time of Bhopal Gas tragedy. The

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Indian Physiologist JS Paintel discovered these receptors and they are named after him.

Q. 3. What is hypoxia? Describe the different types of hypoxia with examples.

Ans. Describe briefly the changes that will take place in a person ascending to a height of about 10,000 feet.

Hypoxia is defined as deficiency of oxygen at the tissue level

It is divided into:

1. **Hypoxic hypoxia:** The partial pressure of oxygen in the arterial blood is reduced. At high altitudes >10,000 feet, breathing in a closed space, airway obstruction, weakness or paralysis of respiratory muscles, depression of medulla by morphine. This occurs due to decrease in the partial pressure in air and consequently alveoli and blood.
2. **Ischemic or anemic hypoxia:** This occurs when hemoglobin concentration is low. The decrease in hemoglobin concentration in carbon monoxide poisoning occurs due to formation of carboxyhemoglobin. The CO-Hb affinity is twenty times more than that of oxygen-Hb. The individual is cherry red in color. The other causes are methemoglobinemia.
3. **Stagnant hypoxia:** This occurs when blood flow to the tissue is low. Hypoxia due to circulatory failure, hemorrhage, congenital heart failure, when deoxygenated hemoglobin exceeds 5gm% produces cyanosis.
4. **Histotoxic hypoxia:** In cyanide poisoning the cells are unable to utilise oxygen due to arrest of its cellular machinery of cytochrome oxidase.

High altitude

Hyperventilation occurs due to hypoxic hypoxia, which stimulates the peripheral chemoreceptors. The concentration of 2,3 DPG occurs, because of increase of pH in the cells.
(as deoxyhemoglobin binds more hydrogen ions than

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oxyhemoglobin). The person feels lazy, sleepy and may get a headache. These are due to central CNS depression by hypoxia.

Acclimatization of high altitude

1. Larger chest size
2. Increase in Red blood cells, circulation improves
3. Increased vascularity and improved oxygen utilization in tissues

HAPO: High altitude pulmonary edema Pulmonary vasoconstriction in response to hypoxia is associated by hypertension and causes edema. The person suffers from dyspnea. He needs rest, calcium channel blockers, oxygen and return to lower altitude. This may occur even in acclimatized individuals who exercise and is usually seen in unacclimatized individuals. It is also associated with nausea, vomitting, headache and cerebral edema.

Q. 4. Describe briefly:

Ans. a. Periodic breathing
b. Lung compliance
c. Role of chemoreceptos in the regulation of respiration
d. FEV1 its normal value and its clinical significance
e. Surfactant
f. Decompression sickness
g. Bohr effect
h. Chemical regulation of respiration
i. The respiratory changes in deep sea diving
j. Mechanism of inspiration and expiration.

a. Periodic breathing: The person has alternating spells of apnea and respiration.

During hyperventilation the CO_2 wash out occurs. The hypercapnic drive is lost and apnea or breathing ceases. This leads to a build in the partial pressure of carbon-dioxide to cause hyperventilation. The cycle repeats itself with breathing occurring only at intervals or periods.

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Cheyne's Stoke or Biot's breathing are examples of periodic breathing. Cheyne's stokes occurs in sleep, high altitude, uremia, where the central chemoreceptors have altered sensitivity and in congestive heart failure when the circulation slows down. Biot's breathing occurs in cases of head injury.

b. Lung Compliance: Δ V/ Δ P : Unit change in lung volume with unit change in pressure of the airway is termed as Lung Compliance or distensibility

Normal Value of lungs and thorax= 0.13L/cm of H_2O

Means that if the increase in pressure of airways by I cm of H_2O then the volume of lungs in the thoracic wall increases by 0.13L

Normal value of lungs only is =0.22 L/cm of H_2O

The changes in pressure are measured by a manometer in the esophagus and the changes in volume with the help of a spirometer.

Compliance decreases in conditions of fibrosis, scoliosis, pulmonary congestion. It is increased in emphysema. The compliance is a static measure of lung recoil.

c. Role of chemoreceptors in the regulation of respiration :

Central chemoreceptors are present in the ventral surface of medulla near the respiratory center. They are responsive to increase in H^+ concentration in brain interstitial fluid (parallels hypercapnia and hypoxia). They are inhibited during sleep, anesthesia and cyanide. They trigger an increase in rate and depth of respiration. It regulates the respiration from minute to minute. Provides late drive to increase respiration in response to increase in arterial pCO_2. Contributes to 80% increase in pulmonary ventilation.

The peripheral chemoreceptors are located in the carotid and aortic body. They are responsive to hypoxia, primarily. They send electrical signals via the glossopharyngeal and vagus

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(buffer nerve), that connect to the medullary group of respiratory neurons to increase rate of respiration.

d. FEV_1, its normal value and its clinical significance: Forced expiratory volume in one second is the amount of air that is expelled from the lungs in one second.

FEV1/FVC > 80% is considered as normal.

If FEV1/FVC < 80% it is indicative of an obstruction in the airway, e.g. asthma

On administration of bronchodilator the ratio returns to normal.

e Surfactant: The surfactant is Dipalmitoyl phosphatidylcholine (DPCC), phospholipids, glycine, lipids, proteins and carbohydrate. It is secreted by Pneumocyte II cell and lines the alveoli. It serves the following function.

1. Helps keep the alveoli dry. Air exchange takes place across dry surfaces. Surfactant prevents pulmonary edema by Starling forces, causing an inward suction force. This is the difference from systemic circulation where interstitial fluid is present, its absent in the lungs.
2. Reduces surface tension of the alveoli.
3. Prevents alveoli from collapsing

P= 2T/R To keep Pressure (P) constant when alveolar radius decrease the surfactant reduces the tension, Thus preventing the alveoli from collapsing.

Surfactant is important at birth. When the infants cries to take in first breath, the lung expands, surfactant prevents them from collapsing again.

Deficiency of surfactant is the cause of hyaline membrane disease or Respiratory distress syndrome (RDS). The alveoli are collapsed or atelectasis. Adminstration of bovine surfactant helps, insulin, thyroid and glucocorticoid accelerates maturation of surfactant.

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f. Decompression sickness: This is also called Caissons disease or Bend's disease or Dysbarism or Divers disease.
It is cause by the increased pN_2 in blood. Nitrogen is five times more soluble in fat than water and passes through cell membrane very slowly. During diving the Nitrogen gas dissolves in the blood. Due to high pN_2 **Nitrogen narcosis** is produced. This is characterized by euphoria or sense of well being, impairment of mental functions or symptoms resembling alcoholic intoxication.

When the diver suddenly comes to the surface, dissolved N_2 comes out in the fluid like bubbles.

Bubbles press the nerve to produce sensory and motor disturbances. Loss of sensations, itching, paresthesias are the symptoms. **Divers palsy** may also be produced in which the diver is paralysed. Bubbles block pulmonary capillaries to produce dyspnea called **Chokes.** In the heart it leads to myocardial infarction. In joints and muscles it causes severe pain called **Bends.**

Treatment and Prevention

Diver should come to the surface slowly. The subject may be recompressed by putting him in a recompression chamber that redissolves N_2. The body can then slowly get rid of the nitrogen. The pain, dyspnea and paresthesias disappear.

Nitrogen narcosis can be avoided by breathing **O_2-Helium mixture** instead of N_2.

g. Bohr effect: Bohr effect is the unloading of O_2 due to loading of CO_2. The decrease of affinity of pO_2 for hemoglobin occurs when the pH of the blood falls. The other factors that cause the O_2-dissociation curve to shift to right are high altitude, increased body temperature, anaemia, exercise, hormones like thyroid, androgen and growth hormone. These increase the 2,3 DPG levels.

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h. **Chemical regulation of respiration:** Central chemoreceptors are present in the ventral surface of medulla near the respiratory center. They are responsive to increase in H^+ concentration in brain interstitial fluid (parallels hypercapnia and hypoxia). They are inhibited during sleep, anesthesia and cyanide. They trigger an increase in rate and depth of respiration. It regulates the respiration from minute to minute. Provides late drive to increase respiration in response to increase in arterial pCO_2. Contributes to 80% increase in pulmonary ventilation.

Location:
There are two or more aortic bodies near the arch of aorta. Two carotid bodies one on each side near bifurcation of common carotid artery.
Innervation: Aortic body by aortic nerve (X nerve), carotid bodies by carotid sinus nerve (IX nerve).
Stimulated by: Increase arterial pCO_2, decrease arterial pO_2, cyanide, nicotine and lobeline. Mainly stimulated by hypoxia, vascular stasis, asphyxia
Influence on respiration:

- Increase rate and depth of respiration, hypertension, and tachycardia.
- Regulates the respiration from breath to breath.
- Provides initial drive to increase respiration in response to increase in arterial pCO_2
- Contributes to 20% increase in pulmonary ventilation

The peripheral chemoreceptors are located in the carotid and aortic body. They are responsive to hypoxia, primarily. They send electrical signals via the glossopharyngeal and vagus (buffer nerve), that connect to the medullary group of respiratory neurons to increase rate of respiration.

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Pulmonary and myocardial chemoreceptors

They are located in pulmonary and myocardial coronary blood vessels. It is innervated by vagus nerve. Stimulated by injecting veratridine or nicotine in these vessels. The effect of stimulation is bradycardia, hypotension, apnea followed by tachypnea. This is termed as **Pulmonary chemoreflex or Coronary chemoreflex or Bezold Jarisch reflex.**

This is seen in MI.

i. **The respiratory changes in deep sea diving:**
   At 33 feet in the sea an increase of one atmosphere pressure occurs. The lungs become small and this can produce serious damage.

The high atmospheric pressure increases partial pressures of Nitrogen and oxygen to equalize the high pressure of water on chest wall and abdomen. This may produce **Oxygen toxicity and Caisson's disease.**

Oxygen toxicity causes retrolental fibroplasia, respiratory nasal congestion, sore throat, sneezing, coughing, tachypnea, bronchoconstriction, and bronchopneumonia.

These complications may be prevented by SCUBA (Self contained underwater breathing apparatus) and by breathing oxygen-helium mixtures.

j. **Mechanism of inspiration and expiration:** The I neurons send excitatory impulses to the pneumotaxic center. This discharges inhibitory impulses to apneustic center.

The pulmonary stretch receptors in the lungs get stimulated during inspiration. These send inhibitory impulses via vagus to apneustic center.

The pneumotaxic center stimulates the expiratory center, which reciprocally inhibits the inspiratory center. *This can be simulated by two men slapping each other.*

Apneustic center gets inhibited and ceases to activate the inspiratory center as a result the inspiratory center stops discharging and expiration follows passively.

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The E neurons discharge only in exercise or voluntary expiration.

Inspiration is an active process in which thorax is enlarged by rib movements (pump handle and bucket handle) and descent of diaphragm (as a result of discharge of $C_{3,4,5}$).

The chests expand, pulling the parietal pleura outwards , the intrapleural pressure falls and the visceral pleura pulls the lungs outward. Air enters as the lung expands.

Accessory muscles of inspiration are scaleni and sternocleidomastoid.

Expiration is a passive process. The lungs collapse after inspiration. Forceful expiration is brought about by contraction of anterior abdominal wall muscles , upward movement of diaphragm and internal intercostal muscles contraction. Accessory muscles of expiration are adductor muscles of vocal cord.

Q.5. Describe the transport of carbondioxide by the blood. What is the role of carbondioxide in the regulation of respiration?

Ans. Transport of carbondioxide by blood

1. Carbondioxide is transported as bicarbonate ion.
2. Carbaminohemoglobin is formed with combination with hemoglobin
3. Carbondioxide is dissolved in the plasma.

Role of carbondioxide in regulation of respiration.

CO_2 stimulates respiration. Carbondioxide dissolves in the CSF after diffusing the blood brain barrier.

$$CO_2 + H_2O \xrightarrow{CA} HCO_3^- + H^+.$$

CA Carbonic Anhydrase

The H^+ ion stimulates the central chemoreceptor in the medulla to produce increase in rate of respiration.

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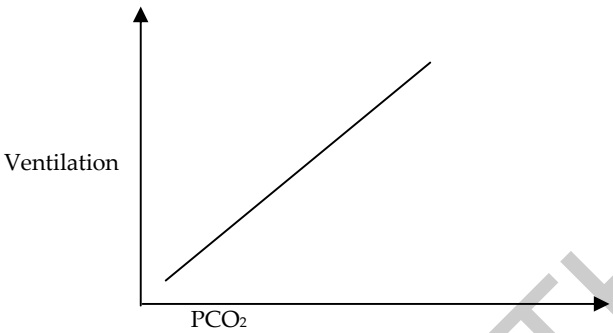

Q. 6. Describe how oxygen is transported in the blood and factors that influence its delivery to the tissues. Enumerate the factors affecting the shift of oxygen dissociation curve.

Ans. Vehicles for the transport of Oxygen

1. Plasma
2. Hemoglobin
3. Blood

Factors that influence delivery to tissues

1. Amount of O_2 entering the lungs
2. The adequacy of pulmonary gas exchange
3. Blood flow to the tissues (constriction of vascular bed and the cardiac output)
4. Capacity of the blood to carry oxygen (Hemoglobin amount and affinity of Hb to oxygen).

Factors affecting the shift of oxygen dissociation curve.

Shift to Right

1. pH of the blood falls due to increase in CO_2 or any acid.
2. High altitude.
3. Increased body temperature.
4. Increase in 2,3 DPG levels.

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5. Anaemia, exercise
6. Hormones like thyroid, androgen and growth hormone.

Shift to Left

1. Alkalosis
2. Decrease in body temperature
3. Carbonmonoxide poisoning
4. Fetal hemoglobin
5. Myoglobin.

Q. 7. Define Vital Capacity. Describe Timed vital capacity. What is the difference in timed vital capacity in normal individual and individual with obstructive lung disease?

Ans. Vital capacity: IRV+TV+ERV. Is the maximal volume of air that can be expelled out of the lungs after maximal inspiration. Its normal value in males is 4.8 liters and 3.2 liters in females.

Timed Vital capacity is the maximum volume of air which can be breathed out as "forcefully" and rapidly as possible following a maximum inspiration.

Forced expiratory volume in one second is the amount of air that is expelled from the lungs in one second.

FEV1/FVC > 80% is considered as normal.

If FEV1/FVC < 80% it is indicative of an obstruction in the airway e.g. asthma.

On administration of bronchodilator the ratio returns to normal.

Q. 8. What are the various lung volumes and capacities, give their normal values

Ans. Tidal volume Is the volume of air that is breathed in or out of the lungs during quiet respiration. Normal value is 500 ml.

Inspiratory reserve volume: is the maximum volume of air which can be inspired after completing a normal tidal inspiration. Its normal value is 2000 to 3200 ml

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Inspiratory capacity is the sum of inspiratory reserve volume plus the tidal volume. Is the maximum volume of air that can be inspired after completing tidal expiration. Normal value is 2500 –3700 ml

Expiratory reserve volume is the maximum volume of air which can be expired after a normal tidal expiration. Normal value is 750 to 1000 ml.

Expiratory capacity TV+ ERV. is the maximum volume of air that can be expired after completing tidal inspiration. Normal value 1250 to 1500 ml.

Residual volume is the volume of air that remains in the lungs after normal expiration. Normal value is 1200 ml.

Closing volume is the lung volume above residual volume at which airways in the lower dependent parts in the lungs begin to close of because of the lesser transmural pressure of these areas.

Functional residual capacity ERV+RV. is the volume of air that remains in the lungs after normal tidal expiration. Normal value is 2450 to 2700 ml

Vital capacity IRV+TV+ERV. is the maximal volume of air that can be expelled out of the lungs after maximal inspiration. Its normal value in males is 4.8 liters and 3.2 liters in females.

Total lung capacity VC+RV. Is the volume of the air contained in the lungs after maximal inspiration. Normal is 6 liters.

Capacities are the sum of volumes.

Q. 9. With reference to peripheral chemoreceptors, mention their

i. Location of Nerve supply
ii. ii.specific stimuli and
iii. Influence on respiration.

Ans. Location: There are two or more aortic bodies near the arch of aorta. Two carotid bodies one on each side near bifurcation of common carotid artery.

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Innervation: Aortic body by aortic nerve (X nerve), carotid bodies by carotid sinus nerve (IX nerve).

Stimulated by: Increase arterial pCO_2, decrease arterial pO_2, cyanide, nicotine and lobeline. Mainly stimulated by hypoxia, vascular stasis, asphyxia.

Influence on respiration:

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Questions & Answers in PHYSIOLOGY

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This book has been written to help the students to understand concepts of physiology. They shall benefit by the ability to write answers in examinations.

1. Blood
2. Cardiovascular System (CVS)
3. Special Senses
4. Endocrinology
5. Central Nervous System (CNS)
6. Gastrointestinal System (GIT)
7. Kidney
8. Lungs.

Varun Malhotra did his MBBS from Maulana Azad Medical College, New Delhi; MD from University College of Medical Sciences, New Delhi; DNB (Physiology), and is a gold medalist in naturopathy and yoga. He is an author of *MCQ's in Physiology with Explanatory Answers* and course writer of Clinical Applied Physiology, with IGNOU. Presently, he is a Professor in Physiology at Vinayaka Missions Medical College, Salem, Tamil Nadu, India.

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