Chapter 14: Breathing and Exchange of Gases

Exercises — Exercises

Q1 Define vital capacity. What is its significance?

SOLUTION

Vital Capacity (VC) is the maximum volume of air a person can breathe in after a forced expiration, or equivalently, the maximum volume of air a person can breathe out after a forced inspiration.

It is the sum of Tidal Volume (TV) + Inspiratory Reserve Volume (IRV) + Expiratory Reserve Volume (ERV).

Significance:

1. Vital capacity is an index of the maximum ventilation capacity of the lungs — the greater the VC, the more efficiently a person can ventilate the lungs during strenuous exercise.
2. It is measured by a spirometer and used clinically to assess pulmonary function and detect restrictive or obstructive lung diseases (e.g., fibrosis, emphysema).
3. Athletes and physically fit individuals generally have a higher vital capacity, reflecting stronger respiratory muscles and greater lung compliance.
4. A reduced vital capacity may indicate conditions like pleural effusion, pneumonia, or muscular weakness affecting respiration.

Answer: Vital Capacity = TV + IRV + ERV; it measures the maximum usable ventilation range and is a key clinical indicator of lung health and physical fitness.

Q2 State the volume of air remaining in the lungs after a normal breathing.

SOLUTION

After a normal (quiet) expiration, the volume of air remaining in the lungs is the Functional Residual Capacity (FRC).

FRC = Expiratory Reserve Volume (ERV) + Residual Volume (RV)

FRC = approximately 1000–1100 mL (ERV) + 1100–1200 mL (RV) = approximately 2100–2300 mL.

Note: The Residual Volume (RV) alone (about 1100–1200 mL) remains even after a forcible expiration, but after a normal expiration the lungs retain the FRC (ERV + RV).

Answer: After normal expiration, Functional Residual Capacity (FRC = ERV + RV) remains in the lungs — approximately 2100–2300 mL.

Q3 Diffusion of gases occurs in the alveolar region only and not in the other parts of respiratory system. Why?

SOLUTION

The respiratory tract is divided into two functional parts:

1. Conducting part: External nostrils → nasal chamber → pharynx → larynx → trachea → bronchi → bronchioles → terminal bronchioles. This portion merely transports air; it does not participate in gas exchange.

2. Exchange (respiratory) part: Alveoli and alveolar ducts — this is where actual diffusion of O2 and CO2 occurs.

Reasons why diffusion is restricted to alveoli:

- Alveoli are thin-walled (simple squamous epithelium), highly vascularised sacs surrounded by a dense network of pulmonary capillaries, providing an enormous surface area (~70 m2 in adults) for diffusion.
- The diffusion membrane at the alveolus is extremely thin (< 1 mm), consisting of: (i) thin squamous epithelium of alveoli, (ii) basement substance, and (iii) endothelium of alveolar capillaries — allowing rapid passive diffusion.
- In contrast, the walls of trachea, bronchi and bronchioles are thick, composed of cartilage, smooth muscle, and connective tissue, and lack the intimate capillary network required for gas exchange.
- The conducting airways are lined with ciliated pseudostratified columnar epithelium (not thin squamous epithelium), presenting a thick barrier unsuitable for diffusion.
- Therefore, all conditions for efficient gas exchange — thin membrane, large surface area, rich blood supply, and maintained partial pressure gradients — exist only at the alveolar level.

Answer: Alveoli alone have the thin diffusion membrane, vast vascularised surface area and maintained partial pressure gradients needed for gas exchange; the conducting airways have thick walls, no capillary network and serve only air transport.

Q4 What are the major transport mechanisms for CO2? Explain.

SOLUTION

Carbon dioxide produced at the tissues is transported to the alveoli by three mechanisms:

1. As bicarbonate ions (HCO3-) — approximately 70%:
CO2 diffuses from tissues into RBCs and plasma. Inside RBCs, the enzyme carbonic anhydrase catalyses:
CO2 + H2O --> H2CO3 --> HCO3- + H+
HCO3- moves out into plasma in exchange for Cl- (chloride shift). At the alveoli, the reaction reverses (carbonic anhydrase again), regenerating CO2 which diffuses out.

2. As carbamino-haemoglobin — approximately 20-25%:
CO2 binds directly to haemoglobin (at amino groups) to form carbamino-haemoglobin. This binding is favoured by high pCO2 and low pO2 (tissue conditions). At the alveoli, low pCO2 and high pO2 cause dissociation and CO2 is released.

3. Dissolved in plasma — approximately 7%:
A small fraction of CO2 remains dissolved as a gas in the blood plasma and is carried in this state to the alveoli.

Every 100 mL of deoxygenated blood delivers approximately 4 mL of CO2 to the alveoli.

Answer: CO2 is transported as: (1) bicarbonate HCO3- (~70%, via carbonic anhydrase in RBCs), (2) carbamino-haemoglobin (~20-25%, bound to Hb), and (3) dissolved in plasma (~7%).

Q5 What will be the pO2 and pCO2 in the atmospheric air compared to those in the alveolar air?
(i) pO2 lesser, pCO2 higher
(ii) pO2 higher, pCO2 lesser
(iii) pO2 higher, pCO2 higher
(iv) pO2 lesser, pCO2 lesser

SOLUTION

From Table 14.1 in the chapter:
- Atmospheric air: pO2 = 159 mm Hg, pCO2 = 0.3 mm Hg

- Alveolar air: pO2 = 104 mm Hg, pCO2 = 40 mm Hg

Comparing atmospheric air to alveolar air:
- pO2: atmospheric (159) > alveolar (104) — so atmospheric pO2 is HIGHER
- pCO2: atmospheric (0.3) < alveolar (40) — so atmospheric pCO2 is LESSER

Therefore, in atmospheric air: pO2 is higher and pCO2 is lesser compared to alveolar air.
The correct answer is (ii).

Answer: (ii) pO2 higher, pCO2 lesser — atmospheric air has pO2 = 159 mm Hg vs alveolar 104 mm Hg, and pCO2 = 0.3 mm Hg vs alveolar 40 mm Hg.

Q6 Explain the process of inspiration under normal conditions.

SOLUTION

Inspiration is the process by which atmospheric air is drawn into the lungs. It is an active process requiring muscular contraction.

Steps of inspiration:
1. Contraction of the diaphragm: The dome-shaped diaphragm contracts and flattens, increasing the thoracic volume in the antero-posterior (top-to-bottom) axis.
2. Contraction of external intercostal muscles: These muscles contract, lifting the ribs and sternum upward and outward, increasing the thoracic volume in the dorso-ventral axis.
3. Increase in thoracic volume: The combined effect increases the overall volume of the thoracic cavity.
4. Increase in pulmonary volume: Since the lungs are enclosed in the airtight thoracic cavity, the increase in thoracic volume causes a corresponding increase in pulmonary (lung) volume.
5. Fall in intra-pulmonary pressure: The expanded lung volume causes the intra-pulmonary pressure to decrease below atmospheric pressure (negative pressure in the lungs relative to atmosphere).
6. Air flows in: Air from outside, which is at higher atmospheric pressure, rushes into the lungs through the respiratory tract to equalise the pressure — this is inspiration.

Under forced inspiration, the accessory muscles of the neck and abdomen provide additional help. A healthy person breathes 12-16 times per minute at rest.

Answer: Contraction of diaphragm + external intercostals increases thoracic and pulmonary volume, reducing intra-pulmonary pressure below atmospheric pressure, causing air to flow into the lungs.

Q7 How is respiration regulated?

SOLUTION

Respiration is regulated primarily by the neural system through specialised centres in the brain:

1. Respiratory rhythm centre (in medulla oblongata):
   - The primary centre responsible for maintaining the basic respiratory rhythm.
   - It sets the rate and depth of breathing to meet the metabolic demands of the body.

2. Pneumotaxic centre (in pons region of brain):
   - Moderates the function of the respiratory rhythm centre.
   - Neural signals from this centre can reduce the duration of inspiration, thereby increasing respiratory rate when needed.

3. Chemosensitive area (adjacent to rhythm centre in medulla):
   - Highly sensitive to CO_2 and H^+ ions (not significantly to O_2).
   - An increase in CO_2 or H^+ activates this area, which signals the rhythm centre to increase the rate and depth of breathing to eliminate excess CO_2.

4. Peripheral chemoreceptors (in aortic arch and carotid artery):
   - Receptors here can detect changes in pCO_2 and H^+ concentration in the blood.
   - They send signals to the respiratory rhythm centre, which then makes adjustments to restore normal levels.

Note: The role of oxygen (pO_2) in regulating respiratory rhythm is relatively insignificant compared to CO_2 and H^+.

Answer: Respiration is regulated by the respiratory rhythm centre (medulla), modulated by the pneumotaxic centre (pons) and a chemosensitive area (medulla) sensitive to CO_2/H^+, and peripheral receptors in the aortic arch and carotid artery.

Q8 What is the effect of pCO2 on oxygen transport?

SOLUTION

High pCO2 reduces oxygen transport by haemoglobin — this is known as the Bohr effect.

Mechanism:
- Haemoglobin (Hb) binds O2 primarily based on pO2, but pCO2, H+ concentration, and temperature are important modifying factors.
- When pCO2 is high (as in the tissues), CO2 reacts with water to form H2CO3, which dissociates to give H+ ions, lowering pH.
- High H+ concentration and high pCO2 reduce the affinity of haemoglobin for oxygen — the oxygen dissociation curve shifts to the right (Bohr effect).
- This promotes dissociation of oxyhaemoglobin, releasing O2 to the metabolically active tissues.

At the alveoli:
- pCO2 is low (40 mm Hg in alveoli vs 45 mm Hg in tissues), so H+ is low, pH is higher, and Hb has a higher affinity for O2.
- O2 readily binds to Hb to form oxyhaemoglobin.

Summary: High pCO2 favours O2 release (dissociation of oxyhaemoglobin); low pCO2 favours O2 binding. This ensures efficient O2 delivery where it is most needed (actively respiring tissues).

Answer: High pCO2 (and the associated rise in H+) decreases Hb's affinity for O2, promoting O2 release at tissues (Bohr effect); low pCO2 at alveoli increases Hb affinity, promoting O2 uptake.

Q9 What happens to the respiratory process in a man going up a hill?

SOLUTION

As a person goes up a hill (higher altitude), the following changes occur in the respiratory process:

1. Decrease in atmospheric pressure: At higher altitudes, the total atmospheric pressure falls, and consequently the partial pressure of oxygen (pO2) in the air is lower.
2. Reduced O2 availability: The lower pO2 means less oxygen is available to diffuse across the alveolar membrane into the blood.

3. Decreased pO2 in alveoli: Less O2 reaches the alveoli, so alveolar pO2 falls.
4. Reduced oxyhaemoglobin formation: Less O2 is loaded onto haemoglobin, leading to lower blood oxygen saturation.
5. Compensatory increase in breathing rate (hyperventilation): The body tries to compensate for low O2 by increasing the rate and depth of breathing to bring in more air.
6. Increased heart rate: To deliver more oxygenated blood to tissues.
7. Symptoms of altitude sickness: The person may experience breathlessness, dizziness, nausea, and headache (symptoms of hypoxia) if the body cannot compensate adequately.
8. Long-term acclimatisation: Over days, the body increases production of RBCs and haemoglobin (erythropoiesis) to improve O2 carrying capacity, and the 2,3-BPG levels in RBCs increase, shifting the O2 dissociation curve to the right for better O2 unloading.

Answer: At higher altitude, pO2 is lower; the person breathes faster and deeper (hyperventilation) to compensate. Persistent low pO2 causes hypoxia; long-term acclimatisation involves increased RBC/Hb production.

Q10 What is the site of gaseous exchange in an insect?

SOLUTION

In insects, the site of gaseous exchange is the tracheal system — a network of tubes called tracheae and tracheoles.

Details:
- Insects have a network of tracheal tubes (tracheal respiration) that branch extensively throughout the body.
- The tracheae open to the outside through small pores called spiracles located on the body surface.
- The tracheae branch repeatedly into finer tubes called tracheoles, which penetrate directly into the tissues and cells.
- Gaseous exchange (O2 in, CO2 out) occurs by simple diffusion at the tips of the tracheoles, which are in direct contact with the cells.
- This system bypasses the circulatory system — oxygen is delivered directly to cells without the need for a respiratory pigment in the blood.
- Therefore, the tracheoles (and specifically the tracheole tips/ends) are the actual sites of gaseous exchange in insects.

Answer: In insects, gaseous exchange occurs at the tracheoles — fine terminal branches of the tracheal tube system — where O2 and CO2 diffuse directly between air and body cells.

Q11 Define oxygen dissociation curve. Can you suggest any reason for its sigmoidal pattern?

SOLUTION

Definition:

The oxygen dissociation curve is a graph that plots the percentage saturation of haemoglobin with oxygen (on the Y-axis) against the partial pressure of oxygen — pO2 (on the X-axis). It represents the relationship between pO2 and the degree to which haemoglobin is bound to oxygen.

Sigmoidal (S-shaped) pattern — reasons:

1. Cooperative binding (allosteric effect): Haemoglobin is a tetramer with four subunits, each carrying one haem group. When the first O2 molecule binds to one haem, it causes a conformational change in the haemoglobin molecule that increases the affinity of the remaining three haem groups for oxygen.
2. Progressive increase in affinity: The binding of each successive O2 molecule makes it progressively easier for the next O2 to bind. This cooperative binding results in a slow initial rise (at low pO2), then a rapid steep rise (at moderate pO2), and finally a plateau (at high pO2 when all haem groups are saturated).
3. The steep portion of the curve (pO2 between 20-60 mm Hg) corresponds to the range of tissue pO2 — small changes in pO2 at tissues cause large amounts of O2 to be released, ensuring efficient O2 delivery.
4. The plateau at high pO2 (alveolar range) ensures near-complete saturation of haemoglobin in the lungs.
5. The combination of these properties — slow start, rapid mid-section, and flat top — produces the characteristic sigmoid (S-shaped) curve.

Answer: The oxygen dissociation curve plots % Hb saturation vs pO2; its sigmoidal shape arises from cooperative binding — each O2 bound increases Hb's affinity for the next, producing a slow start, rapid mid-rise, and plateau.

Q12 Have you heard about hypoxia? Try to gather information about it, and discuss with your friends.

SOLUTION

Hypoxia is a condition in which the body or a region of the body is deprived of an adequate supply of oxygen, even though blood flow may be maintained.

Types of hypoxia:
1. Hypoxic hypoxia (altitude hypoxia): Caused by low pO2 in the inhaled air, as at high altitudes. The alveolar and arterial pO2 falls, reducing O2 loading onto haemoglobin.
2. Anaemic hypoxia: Caused by reduced haemoglobin concentration or defective haemoglobin (e.g., sickle-cell anaemia, carbon monoxide poisoning — CO binds Hb with 250x the affinity of O2, forming carboxyhaemoglobin).
3. Stagnant (circulatory) hypoxia: Caused by poor blood flow (e.g., heart failure, shock), so O2 is not delivered adequately to tissues even if blood is well-oxygenated.
4. Histotoxic hypoxia: Cells are unable to utilise O2 even when delivered, e.g., cyanide poisoning (blocks cytochrome oxidase in the electron transport chain).

Effects of hypoxia:
- At high altitude: breathlessness, increased heart rate, headache, nausea, fatigue, and in severe cases, pulmonary or cerebral oedema.
- Chronic hypoxia leads to erythrocytosis (increased RBC production via erythropoietin from kidneys), increased breathing rate, and cardiovascular adaptations.

Significance: Understanding hypoxia is important in aviation medicine, mountaineering, anaesthesia, and treatment of heart/lung diseases.

Answer: Hypoxia is oxygen deficiency in tissues; types include hypoxic (low pO2), anaemic (reduced Hb), stagnant (poor blood flow), and histotoxic (cells cannot use O2). Causes range from altitude to CO poisoning; body compensates by increasing breathing rate and RBC production.

Q13 Distinguish between
(a) IRV and ERV
(b) Inspiratory capacity and Expiratory capacity.
(c) Vital capacity and Total lung capacity.

SOLUTION

(a) IRV (Inspiratory Reserve Volume) vs ERV (Expiratory Reserve Volume):
- IRV: The additional volume of air that can be inspired by a forcible (maximum) inspiration beyond a normal tidal inspiration. Approximately 2500-3000 mL.
- ERV: The additional volume of air that can be expired by a forcible (maximum) expiration beyond a normal tidal expiration. Approximately 1000-1100 mL.
- IRV is always greater than ERV. IRV is measured during forcible inspiration; ERV during forcible expiration.

(b) Inspiratory Capacity (IC) vs Expiratory Capacity (EC):
- IC: Total volume of air a person can inspire after a normal expiration = TV + IRV (approximately 3000-3500 mL).
- EC: Total volume of air a person can expire after a normal inspiration = TV + ERV (approximately 1500-1600 mL).
- IC is always greater than EC, as IRV > ERV.

(c) Vital Capacity (VC) vs Total Lung Capacity (TLC):
- VC: Maximum volume of air a person can breathe in after a forced expiration (or breathe out after a forced inspiration) = ERV + TV + IRV. Does NOT include Residual Volume.
- TLC: Total volume of air the lungs can accommodate at the end of a forced inspiration = VC + RV (i.e., ERV + TV + IRV + RV).
- TLC is always greater than VC by the Residual Volume (RV, approximately 1100-1200 mL). RV cannot be expelled even after a forced expiration, so it is not part of VC but is part of TLC.

Answer: (a) IRV = extra air inspired forcibly (~2500-3000 mL); ERV = extra air expired forcibly (~1000-1100 mL). (b) IC = TV+IRV; EC = TV+ERV. (c) VC = ERV+TV+IRV (no RV); TLC = VC+RV.

Q14 What is Tidal volume? Find out the Tidal volume (approximate value) for a healthy human in an hour.

SOLUTION

Tidal Volume (TV):

Tidal Volume is the volume of air inspired or expired during a normal, quiet (resting) respiration. It is approximately 500 mL (0.5 L) per breath in a healthy adult.

Tidal volume in one hour:

- Normal breathing rate = 12-16 breaths per minute. Taking the average = approximately 15 breaths per minute.
- Breaths in one hour = 15 breaths/min x 60 min = 900 breaths/hour.
- Tidal volume per hour = 500 mL x 900 = 450,000 mL = 450 litres per hour.

Alternatively, using the standard calculation from the text:

- A healthy man can inspire or expire approximately 6000-8000 mL per minute (tidal volume x breathing rate = 500 mL x 12-16 = 6000-8000 mL/min).
- Per hour = 6000-8000 mL/min x 60 min = 360,000-480,000 mL = 360-480 litres per hour.
- Approximate value: ~360-480 litres per hour (commonly stated as approximately 420-450 litres/hour).

Answer: Tidal Volume = ~500 mL per breath. At 12-16 breaths/min, a healthy human breathes approximately 360-480 litres of air per hour (~450 L/hr using 15 breaths/min).