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I Semester

Textbook of **Applied**

Anatomy & Physiology

for BSc Nursing Students

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Chapter

2.2

Physiology of Respiration

CHAPTER OUTLINE

INTRODUCTION

The word respiration has been derived from the Latin word *respirare*, which means to *breathe*. The primary role of the respiratory system is to provide O_2 to the tissues for metabolic needs and remove the CO_2 formed by them. An adult body consumes about 250 mL of O_2 and produces about 200 mL of CO_2 per minute. Respiration entails two processes: The external respiration and internal respiration.

The *internal respiration* or tissue respiration refers to utilization of O_2 and production of CO_2 by the tissue.

The *external respiration* includes supply of O_2 to the tissues from the environment and excretion of CO_2 released by the tissues into the atmosphere. The process of external respiration involves three major events:

1. ***Pulmonary ventilation***, i.e., exchange of gases between the environment and lungs. It includes mechanics of respiration.
2. ***Pulmonary diffusion*** refers to transfer of gases from alveoli to the blood by diffusion across the respiratory membrane.
3. ***Transport of gases*** from the blood to the body cells and back.

PULMONARY VENTILATION

Pulmonary ventilation, as defined above, refers to the process of exchange of gases between the environment and lungs. Inflation and deflation of the lung occurring with each breath ensure this regular exchange of gases. The aspects and concept related to pulmonary ventilation, which need deliberation include:

- Mechanism of breathing
- Pressure and volume changes during respiratory cycle
- Lung volumes and capacities
- Pulmonary elastance and compliance.

MECHANISM OF BREATHING

Pulmonary ventilation is accomplished by two processes:

1. Inspiration
2. Expiration

Inspiration

Inspiration refers to inflow of atmospheric air into the lungs. This obviously occurs when the intrapulmonary pressure falls below the atmospheric air pressure.

It is an active process, normally produced by contraction of the *inspiratory muscles.* During tidal inspiration (quiet breathing) the diaphragm and external intercostal muscles contract and cause increase in all the three dimensions of thoracic cavity.

Role of Diaphragm

In tidal inspiration (quiet breathing) 70–75% of expansion of chest is caused due to contraction of diaphragm. The diaphragm is a dome-shaped, musculotendinous partition between thorax and abdomen. The convexity of this dome is directed toward the thorax. When the diaphragm contracts the following changes occur:

- The dome becomes flattened and the level of diaphragm is lowered *increasing the vertical diameter* of the thoracic cavity (Fig. 2.2-1A). During quiet breathing, the descent of diaphragm is about 1.5 cm and during forced inspiration, it increases to 7 cm.
- The descent of diaphragm causes rise in intra-abdominal pressure which is accommodated by the reciprocal relaxation of the abdominal wall musculature.
- Contraction of diaphragm also lifts the lower ribs causing thoracic expansion laterally and anteriorly (the bucket handle and pump handle effect, respectively) (Figs 2.2-1B and C).

Role of External Intercostal Muscles

When the external intercostal muscles contract, the ribs are elevated causing lateral and anteroposterior enlargement of thoracic cavity due to the socalled bucket handle and pump handle effects, respectively.

Role of Laryngeal Muscles

The abductor muscles of the larynx contract during inspiration pulling the vocal cords apart.

IMPORTANT TO KNOW

Forced inspiration is characterized by:

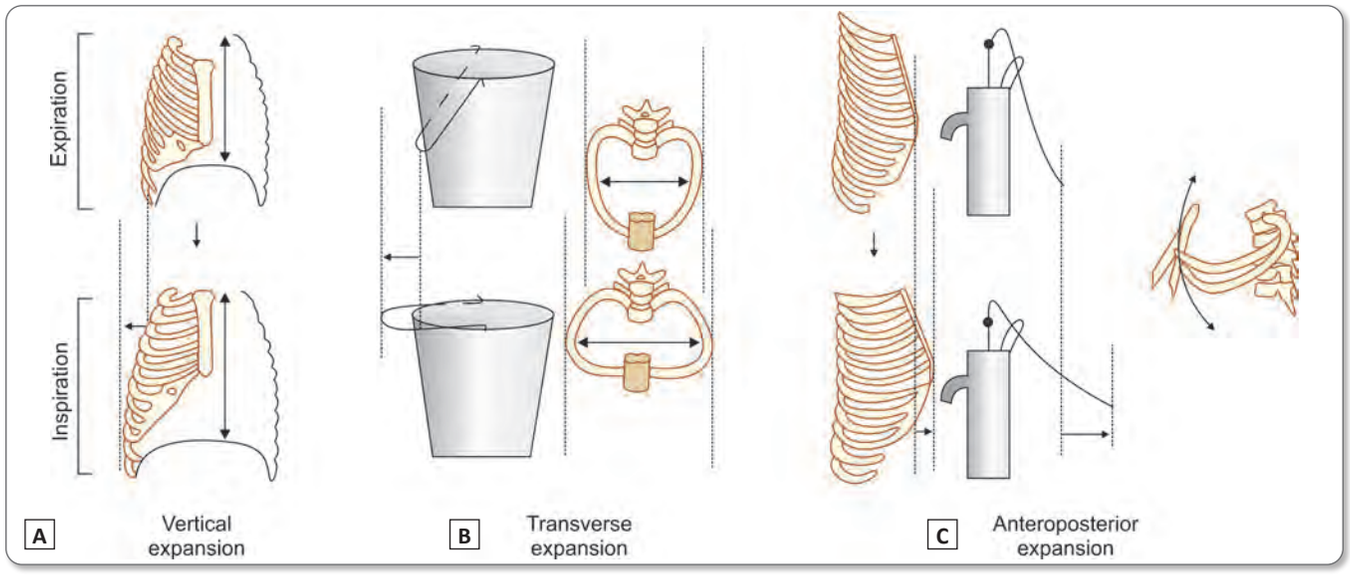
- *Forceful contraction of diaphragm* leading to descent of diaphragm by 7–10 cm as compared to 1–1.5 cm during quiet inspiration.
- *Forceful contraction of external intercostal muscles* causing more elevation of ribs leading to more increase in transverse and anteroposterior diameter of thoracic cavity.
- *Contraction of accessory muscles of inspiration like:* Sternocleidomastoid muscle, anterior serrati muscles, and scaleni muscles that cause further expansion of thorax.

Expiration

Expiration refers to outflow of air from the lungs into the atmosphere. This obviously occurs when the intrapulmonary pressure rises above the atmospheric air pressure. Expiration in quiet breathing is largely a passive phenomenon and is brought about by the:

- Elastic recoil of the lungs
- Decrease in size of the thoracic cavity due to relaxation of diaphragm and external intercostal muscles.

Forced expiration is required when respiration is increased during exercise or in the presence of severe respiratory disease. It is an active process caused by:



Figs 2.2-1A to C: Mechanism of increase in diameter of thoracic cavity: A. Increase in vertical diameter (descent of diaphragm); B. Increase in transverse diameter (bucket handle effect); C. Increase in anteroposterior diameter (pump handle effect)

Contraction of abdominal muscles causes:

- Downward pull on the lower ribs and thus decreases the anteroposterior diameter of the thoracic cavity.
- Fixation of the lower ribs so that internal intercostal muscles act more effectively.

Contraction of the internal intercostal muscles tend to pull all the ribs downward reducing anteroposterior diameter (because of falling of pump handle effect) as well as the transverse diameter (because of action of ribs like falling of bucket handle) of the thoracic cavity.

Pressure and Volume Changes during Respiratory Cycle

Intrapulmonary Pressure Changes during Respiratory Cycle (Fig. 2.2-2)

The movement of air in and out of the lungs depends primarily on the pressure gradient between the alveoli and the atmosphere (i.e., *transairway pressure*). Intrapulmonary or alveolar pressure is the air pressure inside the lung alveoli. Intrapulmonary pressure changes during respiratory cycle are as follows (Fig. 2.2-2):

At end-expiration and end-inspiration, i.e., when the glottis is open and there is no movement of air, pressure in all parts of the respiratory tree are equal to atmospheric pressure, the intrapulmonary pressure is considered to be 0 mm Hg (760 mm Hg atmospheric pressure).

During inspiration in quiet breathing, the pressure in the alveoli decreases to about –1 mm Hg, which is sufficient to suck in about 500 mL of air into the lungs within 2 seconds period of inspiration. At the end-inspiration, the intrapulmonary pressure again becomes zero.

During expiration in quiet breathing, the elastic recoil of the lungs causes the intrapulmonary pressure to swing slightly to the positive side (+1 mm Hg) which forces the 500 mL of inspired air out of the lungs during 2–3 seconds of expiration. At the end-expiration, once again the alveolar pressure regains the atmospheric pressure (0 mm Hg or 760 mm Hg).

Forceful expiration against closed glottis (Valsalva's maneuver) may produce intrapulmonary pressure of as much as 100 mm Hg.

Intrapleural (Pleural) Pressure Changes during Respiratory Cycle

Pleural pressure is the pressure of fluid in the space between the visceral pleura and parietal pleura. Intra-pleural pressure changes during respiratory cycle are as follows (Fig. 2.2-2):

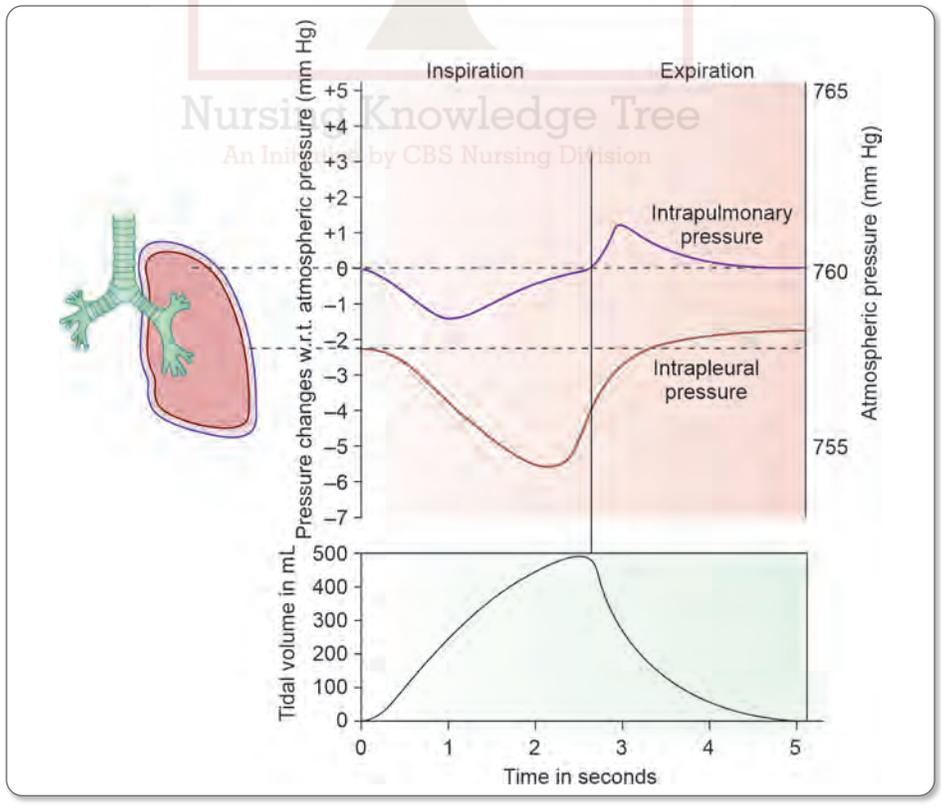


Fig. 2.2-2: Pressure and volume change during respiratory cycle

Normal pleural pressure when the respiratory muscles are completely relaxed and the airways are open, is about –2.5 mm Hg atmospheric pressure. It is the result of balance of two opposite forces, the recoil tendency of the lungs and the recoil tendency of thoracic cage.

During inspiration due to expansion of the chest wall, the pleural pressure becomes still more negative (–6 mm Hg) and pulls the surface of lungs with greater force creating negative intrapulmonary pressure.

At the end of inspiration, the inspiratory muscles relax and the recoiling force of lungs begins to pull the chest wall back to expiratory position. At end-expiratory position where the recoil force of the lungs and recoil force of thoracic cage balance, the pleural pressure returns back to –2.5 mm Hg.

IMPORTANT TO KNOW

Lung Volume Changes during Respiratory Cycle

- *During tidal inspiration*, the volume of air in the lungs increases by 500 mL (tidal volume).
- *During tidal expiration*, the elastic forces compress the gas in the lungs which starts flowing out and at the end of expiration the volume of air in the lungs decreases by 500 mL (Fig. 2.2-2).

Lung Volumes and Capacities

Lung Volumes

The maximum volume to which a lung can be expanded has been divided into four nonoverlapping volumes (Fig. 2.2-3).

Tidal volume (TV)

It is the volume of air inspired or expired with each breath during normal quiet breathing. It is approximately 500 mL in normal adult male.

Inspiratory reserve volume (IRV)

It is the extra volume of air that can be inhaled by a maximum inspiratory effort over and beyond the normal tidal volume. It is about 3000 mL in a normal adult male.

Expiratory reserve volume (ERV)

It is the extra volume of air that can be exhaled by maximum forceful expiration over and beyond the normal tidal volume (i.e. after the end of normal passive expiration). It is approximately 1100 mL in a normal adult male.

Residual volume (RV)

It is the volume of the air that still remains in the lungs after the most forceful expiration. It is about 1200 mL in a normal adult male.

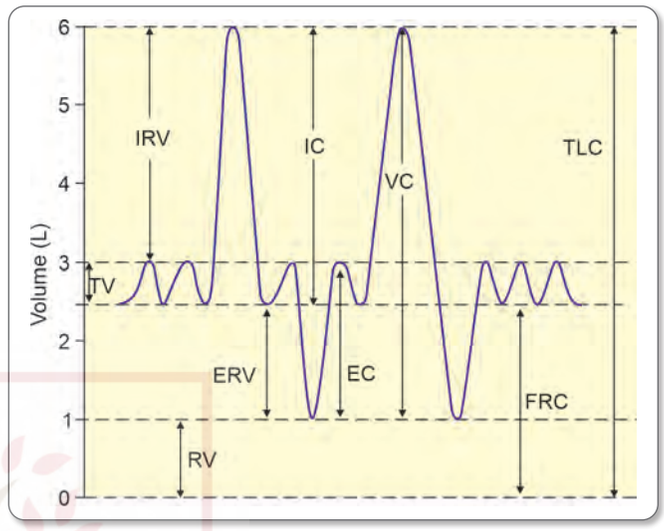


Fig. 2.2-3: A spirogram showing various lung volumes and capacities.

Abbreviations: IRV, inspiratory reserve volume; IC, inspiratory capacity; VC, vital capacity; TLC, total lung capacity; ERV, expiratory reserve volume; EC, expiratory capacity; FRC, functional residual capacity; RV residual volume TV, tidal volume

Lung Capacities

Lung capacities are combination of two or more pulmonary volumes and include the following (Fig. 2.2-3):

Inspiratory capacity (IC)

This is the maximum volume of the air that can be inspired after normal tidal expiration. Therefore, it equals the tidal volume plus inspiratory reserve volume (TV + IRV) and is approximately 3500 mL in a normal adult male.

Expiratory capacity (EC)

It is the maximum volume of air that can be expired after normal tidal inspiration. It equals tidal volume plus expiratory reserve volume (TV + ERV) and is approximately about 1600 mL in a normal adult male.

Functional residual capacity (FRC)

It is the volume of air remaining in the lungs after normal tidal expiration. Therefore, it equals the expiratory reserve volume plus the residual volume (ERV + RV) and is about 2300 mL in a normal adult male.

Significance of functional residual capacity (FRC)

The FRC (RV + ERV) of about 2300 mL represents the air that remains in the lungs most of the time. Even after the most forceful expiration about 1200 mL (residual volume) air is always present in the lungs. This has several advantages:

Continuous exchange of gases is possible due to presence of some air always in the lungs, and thereby concentration of O_2 and CO_2 in blood are maintained constant. Without FRC, pO_2 would have risen to 150 mm Hg during inspiration, and reduced nearly to zero during expiration, which is maintained at about 100 mm Hg due to the FRC.

Breath-holding is made possible due to the FRC.

Dilution of toxic inhaled gases occurs due to the reserve of 2300 mL of air in the lungs (FRC) most of the time.

Load on respiratory mechanism and left ventricle would have been much more if there was no FRC.

Vital Capacity (VC)

This is the maximum amount of air a person can expel from the lungs after the deepest possible inspiration. Therefore, it equals tidal volume plus inspiratory reserve volume plus expiratory reserve volume (TV + IRV + ERV) and is about 4600 mL in a normal adult male. Vital capacity depends on various factors. These include:

Size of the thoracic cavity. VC is more in males (2.6 L/m^2 body surface area) because of large chest size and more muscle power than females.

Age. In old age VC is decreased due to decrease in elasticity of the lungs.

Strength of respiratory muscles. In swimmers and divers VC is more because of the increased strength of the respiratory muscles.

Gravity. In standing position, VC is more than in sitting and lying position because of:

- Increased size of thoracic cavity in standing (diaphragm moves down)
- Reduced pulmonary blood flow due to decreased venous return.

In pregnancy VC is reduced due to pushing up of diaphragm and reduced capacity of thoracic cavity.

In ascites (accumulation of fluid in the abdominal cavity) VC is reduced due to same reason as in pregnancy.

Pulmonary diseases like pulmonary fibrosis, emphysema, respiratory obstruction, pulmonary edema, pleural effusion and pneumothorax are associated in decreased VC.

Timed Vital Capacity (TVC) or Forced Vital Capacities

Forced vital capacity is the volume of the air that can be expired rapidly with a maximum force following a maximum inspiration. The volume of air expired can be timed by recording the vital capacity on a spirograph moving at the known speed. Components of TVC or FVC (Fig. 2.2-4) are:

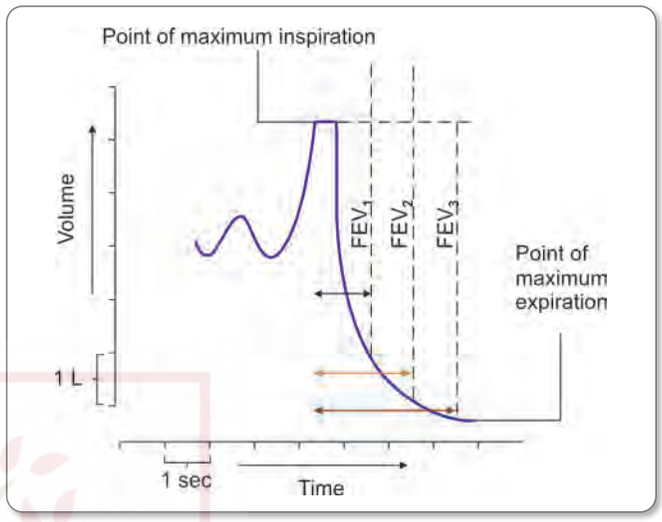


Fig. 2.2-4: Components of timed vital capacity.
Abbreviations: FEV, forced expiratory volume

Forced expiratory volume in 1 sec (FEV_1). It represents the volume expired in the first second of a FVC. FEV_1% is the per cent of FVC expired in one second (i.e. $FEV_1\% = FEV_1/FVC \times 100$). Normally FEV_1% is about 80% of the FVC (Fig. 2.2-5A). Estimation of FEV_1 is the most commonly used screening test for airway diseases.

Clinical application. FEV_1 is useful in distinguishing between restrictive and obstructive lung diseases:

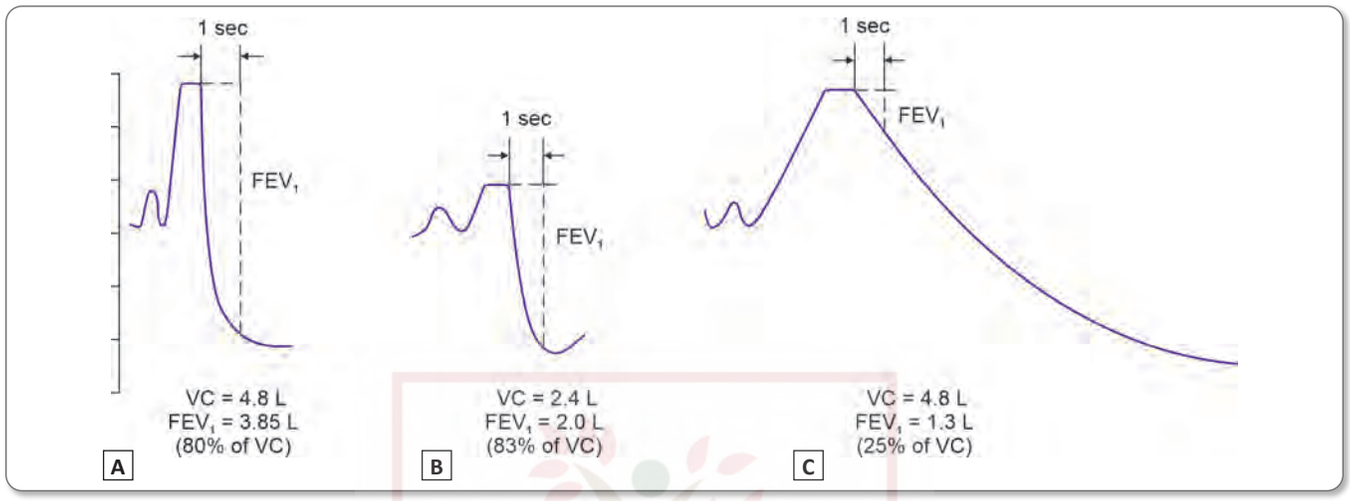
- Patients with *restrictive lung disease* (e.g. kyphoscoliosis and ankylosing spondylitis) have a reduced FVC but are able to achieve relatively high flow rates; therefore their FEV_1% exceeds 80% (Fig. 2.2-5B).
- Patients with *obstructive lung disease* (e.g. bronchial asthma) have low flow rates as a result of high airway resistance. Therefore, their FEV_1% is abnormally low (Fig. 2.2-5C).

Forced expiratory volume in 2 sec (FEV_2): It represents the volume of air expired in first 2 sec. of FVC; FEV_2% is about 90% of FVC under normal condition.

Forced expiratory volume in 3 sec. (FEV_3). It represents the volume of air expired in first 3 sec. Normally FEV_3% is 98–100% of FVC.

Total Lung Capacity (TLC)

It is the volume of air present in the lungs after the maximal inspiration. It equals the vital capacity plus the residual volume (VC + RV) and is about 5800 mL in a normal adult male.



Figs 2.2-5A to C: Forced expiratory volume in first sec (FEV_1) component of timed vital capacity. A. In normal subject; B. In a patient with restrictive lung disease; C. In a patient with restrictive lung disease

IMPORTANT TO KNOW

Minute Ventilation (MV) or Pulmonary Ventilation (PV): It is the volume of air inspired or expired per minute. It equals the tidal volume multiplied by respiratory rate (TV × RR). The TV at rest averages 500 mL (0.5 L), and the normal respiratory rate is 12–15 breaths/min; therefore normal minute volume is 6–7.5 L/min.

Maximum breathing capacity (MBC) or maximum voluntary ventilation or maximum ventilation volume (MVV): It is the maximum volume of air that can be ventilated on command during a given interval. Normal adult male can attain a maximum ventilation volume (MVV) of 80–170 L/min (average 100 L/min).

MVV is profoundly reduced in patients with emphysema, airway obstruction and very poor respiratory muscle strength.

Measurement of static lung volumes and capacities

All volumes and capacities except residual volume, functional residual capacity and total lung capacity are recorded by spirometer. Functional residual capacity is determined by nitrogen washout method or helium dilution method and then residual volume and total lung capacity are calculated.

Recording of lung volumes and capacities are the important lung function tests.

PULMONARY ELASTANCE AND COMPLIANCE

Pulmonary Elastance

Elastance refers to *recoil* (retractive) tendency of a structure. Both the thoracic cage and lungs have elastance.

Elastance or recoil tendency of the thoracic cage refers to constant tendency of the thoracic cage to expand (to pop outward). The elastance of the thoracic cage is because of the fact that the chest wall is an elastic structure, which is normally kept partially pulled inward. The elastic property of the thoracic cage is because of the elastic nature of ribs, muscles and tendons.

Elastance of lungs. Elastance or recoil tendency of the lungs refers to the constant tendency of the lungs to collapse. The recoil forces in the lungs are generated by:

Tissue forces. These are due to the presence of many elastic tissues such as smooth muscle, elastic and collagen in the lung parenchyma which are kept under constant stretch in the inflated lungs.

Surface forces. These are generated at the alveolar surface lined by fluid (*alveolar surface tension*) due to which the alveoli become progressively smaller and collapse.

Alveolar Surface Tension

Alveolar surface tension is generated because of the unbalanced attraction of the liquid molecules at the surface of alveolar membrane.

Pulmonary Surfactant

Pulmonary surfactant is a complex mixture of several phospholipids, proteins and ions. It is secreted by type II alveolar epithelial cells (granular pneumocytes). The presence of pulmonary surfactant in the fluid lining the alveoli reduces the surface tension markedly.

NURSING IMPLICATIONS AND APPLICATIONS

Respiratory distress syndrome (RDS) of newborn or the *hyaline membrane disease* occurs in the newborn babies (especially premature) due to inadequate formation of surfactant resulting in an elevated alveolar surface tension. In this condition it is extremely difficult to expand the lungs. Respiratory work is greatly increased and there is inadequate exchange of gases due to alveolar instability, pulmonary edema, and collapse of alveoli (atelectasis) in many areas. This results in severe respiratory insufficiency and the infant may die.

Pulmonary Compliance

- Compliance (C) refers to change in lung volume (ΔV) per unit change in transpulmonary pressure (ΔP), i.e.,

$$C = \frac{\Delta V}{\Delta P}$$

  Transpulmonary pressure is the difference in the pressure between alveolar pressure and pleural pressure.
- Compliance expresses the distensibility (expansibility) of the lung and chest wall.
- Normal value of compliance for the lungs and chest wall combined is 0.13 L/cm of H_2O and for the lung alone it is 0.22 L/cm of H_2O.

PULMONARY DIFFUSION

Pulmonary diffusion refers to transfer of gases from alveoli to capillary blood across the respiratory membrane.

To understand the intricacies of diffusion of gases across the respiratory membrane it is essential to have knowledge about the following related aspects and concepts:

- Pulmonary perfusion, i.e., pulmonary blood flow *Chapter 2.1 see page 68*).
- Physics of gas diffusion and gas partial pressures.
- Alveolar ventilation, i.e., the rate at which new air reaches the gas exchange area of the lungs.
- Alveolar ventilation-perfusion ratio.
- Diffusion of gases through the respiratory membrane.

PHYSICS OF GAS DIFFUSION AND GAS PARTIAL PRESSURES

Some of the important aspects concerning physics of gas diffusion and gas partial pressures are:

Gas Pressure

The gas molecules have a kinetic energy so they are in a continuous random motion. These molecules bounce against each other and/or against the walls of container and exert a pressure. The gas pressure (P) exerted is denoted by the equation:

$$P = \frac{nRT}{V}$$

where
P = Pressure of gas
n = Number of molecules of gas
T = Absolute temperature
V = Volume of gas
R = Gas constant

Partial Pressure

According to *Dalton's law* of partial pressure, the total pressure exerted by a mixture of gases is equal to the sum of the partial pressure of all gases present in the mixture. Thus, the partial pressure (p) refers to the pressure exerted by any one gas present in a mixture of gases. Hence, the partial pressure (p) of a gas can be calculated by multiplying its fractional concentration by the total pressure. For example, environmental air which has atmospheric pressure (at sea level) of about 760 mm Hg is a mixture of 21% oxygen (O_2), and 79% nitrogen (N_2). Therefore, the partial pressure (p) of O_2 and N_2 respectively will be:

$$pO_2 = 760 \times \frac{21}{100} = 160 \text{ mm Hg}$$

$$pN_2 = 760 \times \frac{79}{100} = 600 \text{ mm Hg}$$

Water Vapor Pressure

The atmospheric air entering the respiratory passages during inspiration is humidified by the water vapors from the conducting passages. By the time the atmospheric air reaches the alveoli, it is saturated with water vapors. Thus, in the alveolar air, besides O_2 and N_2, water vapors also exert their partial pressure. Vapor pressure of water is dependent upon its temperature. At body temperature (37°C) the vapor pressure of water in alveolar air is 47 mm Hg.

ALVEOLAR VENTILATION

Alveolar ventilation is the volume of the fresh air, which reaches the gas exchange area of the lung every minute. During inspiration some of the air inhaled never reaches the gas exchange areas but instead fills the non-gas exchange areas (conducting zone) of the respiratory tract called the *dead space*, which is equal to about 150 mL.

During expiration out of 500 mL of tidal volume 150 mL of the alveolar expired air remains in the conducting passages. Therefore, of 500 mL air entering the lungs only

350 mL/breath is the fresh air, which contributes to alveolar ventilation. Thus, alveolar ventilation can be calculated as:

Alveolar ventilation (VA) = Respiratory rate × (Tidal volume – Dead space volume).

With a normal tidal volume of 500 mL, a normal dead space of 150 mL, and a respiratory rate of 12 breaths per minute, alveolar ventilation equals 12 × (500–350), or 4200 mL/min.

ALVEOLAR VENTILATION-PERFUSION RATIO

Alveolar ventilation-perfusion ratio (VA/Q) is the ratio of alveolar ventilation per minute to quantity of blood flow to alveoli per minute. Normally, alveolar ventilation (VA) is 4.2–5.0 L/min, and the pulmonary blood flow (equal to cardiac output) is approximately 5 L/min. So, the normal VA/Q is about 0.84–0.9. At this ratio, maximum oxygenation occurs.

Alveolar Air

Volume of air which is available for exchange of gases in the alveoli per breath is called alveolar air, which is equivalent to tidal volume minus dead space, i.e., (500–150) or 350 mL.

Composition of Alveolar Air

Alveolar air composition is considerably different than that of atmospheric air because of the following reasons:

- Water vapors dilute the other gases in the inspired air.
- Alveolar air is renewed very slowly by atmospheric air.
- Oxygen is constantly being absorbed from the alveolar air.
- Carbon dioxide is constantly diffusing from the pulmonary blood to alveoli.

Composition of Expired Air

As shown in Table 2.2-1, the composition of expired air is different than that of alveolar air. This is because of the fact that the expired air is a combination of dead space air and alveolar air.

DIFFUSION OF GASES THROUGH THE RESPIRATORY MEMBRANE

Respiratory Unit and Respiratory Membrane

Each respiratory unit is composed of a respiratory bronchiole, alveolar ducts, atria and alveoli. There are about 300 million respiratory units in the two lungs. Gas exchange occurs through the membranes of all the structures forming a respiratory unit, not merely in the alveoli themselves.

Respiratory membrane or pulmonary membrane or the alveolocapillary membrane is the name given to the tissues,

TABLE 2.2-1: Composition and partial pressure of gases in atmospheric air, humidified air, alveolar air and expired air

Partial pressure (mm Hg) and concentration (percentage) of various gases				
Gas air	**Atmospheric air**	**Humidified air**	**Alveolar air**	**Expired**
N_2	597.0 (78.62%)	563.4 (74.09%)	569.0 (74.9%)	566.0 (74.5%)
O_2	159.0 (20.84%)	149.3 (19.67%)	104.0 (13.6%)	120.0 (15.7%)
CO_2	0.3 (0.04%)	0.3 (0.04%)	40.0 (5.3%)	27.0 (3.6%)
H_2O	3.7 (0.5%)	47 (6.20%)	47.0 (6.2%)	47.0 (6.2%)
Total	760.0 (100%)	760 (100%)	760 (100%)	760 (100%)

which separate the capillary blood from the alveolar air. The exchange of gases between the capillary blood and alveolar air requires diffusion through this membrane.

Structure of Respiratory Membrane

It consists of the following layers (Fig. 2.2-6):

- Layer of pulmonary surfactant and fluid lining the alveolus
- Layer of alveolar epithelial cells
- Basement membrane of the alveolar epithelial cells
- A very thin interstitial space between the epithelial and endothelial cells
- Basement membrane of capillary endothelial cells
- Layer of capillary endothelial cells.

Surface area of the total respiratory membrane is about 70 m^2 in the normal adult.

Diffusion and Equilibration of Gases Across the Respiratory Membrane

Diffusion of O_2

The normal alveolar pO_2 is 104 mm Hg, whereas the blood entering the pulmonary capillary normally has a pO_2 of 40 mm Hg. Pressure gradient therefore is 64 mm Hg in the beginning. After dissolving in the respiratory membrane, the O_2 molecules diffuse into the blood. As O_2 diffuses from alveoli to blood, the pO_2 of blood becomes the same as in alveolar air (104 mm Hg), the gradient becomes zero and no diffusion occurs (Figs 2.2-7A and B). By the time blood passes to one-third of distance in capillary the pO_2 of blood equals that of alveoli.

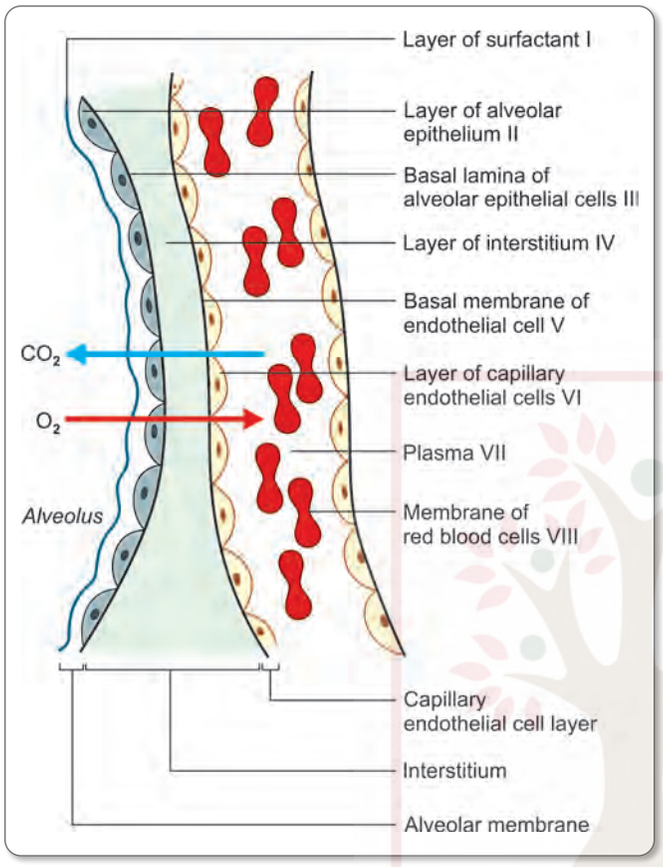
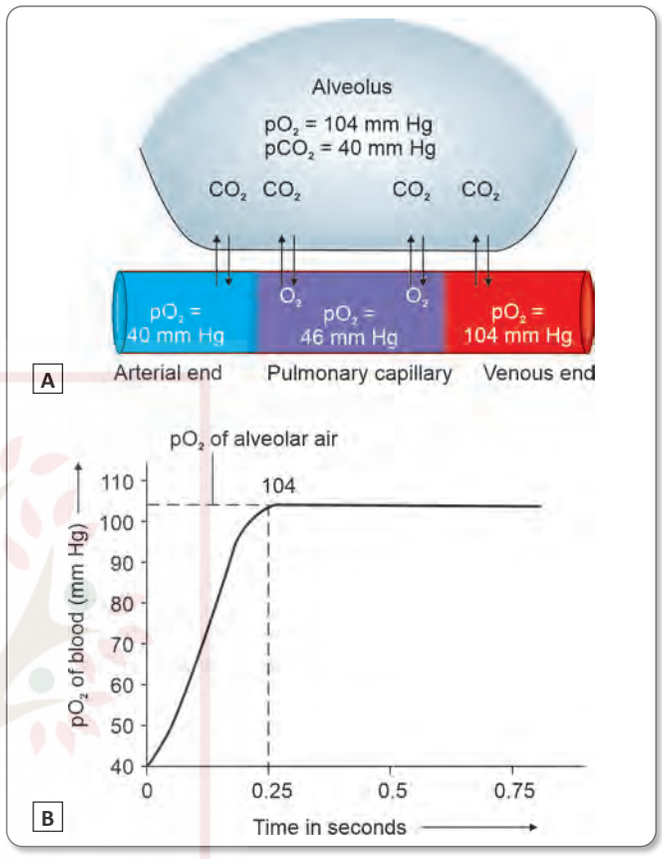


Fig. 2.2-6: Layers of respiratory membrane



Figs 2.2-7A and B: A. Diffusion of oxygen across respiratory membrane; B. Leading to progressive increase in capillary pO_2

Diffusion of CO_2

It occurs from blood to alveoli because pCO_2 is higher in blood than in alveolar air. The average pCO_2 in the pulmonary capillary blood is 46 mm Hg as opposed to 40 mm Hg in the alveoli. Therefore pressure gradient in the beginning is 6 mm Hg and time integrated pressure gradient calculated for CO_2 (in a manner similar to O_2) across the respiratory membrane is only 1 mm Hg. Although the pressure gradient for CO_2 is only one-tenth of the O_2 diffusion gradient, CO_2 diffuses almost 20 times more rapidly than O_2 because of higher diffusion coefficient.

Effect of Alveolar Ventilation-perfusion Ratio on Pulmonary Gas Exchange

- Optimum gas exchange across the respiratory membrane occurs when the alveolar ventilation-perfusion ratio (VA/Q) is normal, i.e., between 0.8 and 1.
- Both, a decrease as well as an increase, in VA/Q ratio reduce the gas exchange.

IMPORTANT TO KNOW

Diffusion Capacity of Lung

Diffusion capacity (DC) of the lung is quantitative expression of the ability of the respiratory membrane to exchange a gas between the alveoli and the pulmonary blood. It is defined as the volume of gas (V gas) that diffuses through the respiratory membrane of lung each minute for a pressure gradient of 1 mm Hg.

TRANSPORT OF GASES

TRANSPORT OF OXYGEN

Transport of oxygen from the lungs to tissues occurs due to constant circulation of blood and diffusion of O_2 that occurs in the direction of concentration gradient which is represented by O_2 tension (pO_2) differences as given below:

- Alveolar air pO_2: 104 mm Hg
- Arterial blood pO_2: 95 mm Hg

- Venous blood pO_2: 40 mm Hg
- Tissue interstitial fluid pO_2: 40 mm Hg

Uptake of Oxygen by Pulmonary Blood

As mentioned above, pO_2 of pulmonary arterial blood is about 40 mm Hg and that of alveolar air is 104 mm Hg. Therefore, due to this great concentration gradient oxygen readily diffuses from the alveoli into the blood.

Transport of Oxygen in Arterial Blood

Arterial blood contains about 20 mL of O_2 and venous blood about 15 mL of O_2 per 100 mL. Thus, about 5 mL of O_2 is transported per 100 mL of blood from lungs to the tissue cells. Oxygen is transported into the blood in two forms:

- As dissolved form
- In combination with hemoglobin.

Oxygen Transport in Dissolved Form

The solubility of O_2 in water (plasma) is so little that at pO_2 value of 100 mm Hg, out of the 20 mL of O_2 present in 100 mL of blood, only 0.3 mL is in dissolved form and rest is combined with hemoglobin (as oxyhemoglobin).

Oxygen Transport in Combination with Hemoglobin

Oxygenation of hemoglobin. After entering into the blood from the alveolar air, most of the oxygen combines with hemoglobin to form a *loose and reversible* combination. This process is called *oxygenation* (not oxidation) and converts deoxyhemoglobin into oxyhemoglobin. Each molecule of hemoglobin can combine with as many as *four O_2 molecules.*

Oxygen-carrying capacity of hemoglobin. One gram of hemoglobin can bind with maximum of 1.34 mL of O_2. Thus 100 mL of blood with hemoglobin level of 15 gm% can carry 1.34×15, or 20.1 mL of oxygen. Practically 100 mL of the arterial blood carries about 19.8 mL of oxygen out of which about 19.5 mL as oxyhemoglobin and 0.3 mL as dissolved form in plasma.

Oxygen-hemoglobin dissociation curve

Oxygen-hemoglobin dissociation curve refers to the curve obtained when the relation between the pO_2 and the percentage of hemoglobin saturation is plotted (Fig. 2.2-8). The O_2-Hb dissociation curve shows that percentage saturation of hemoglobin increases with the increase in pO_2 of arterial blood. However the relation is not linear but *sigmoid or S-shaped.* Several factors affect the affinity for hemoglobin for O_2 and thus shift the O_2-Hb dissociation curve to either right or left.

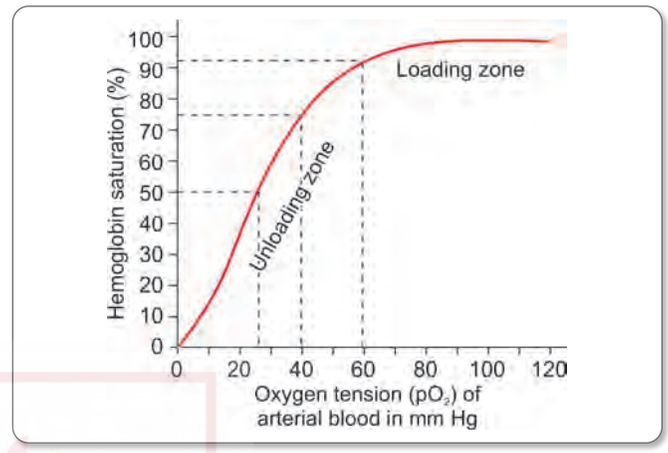


Fig. 2.2-8: Normal oxygen-hemoglobin dissociation curve

Shift to right

Shift to right of O_2-Hb dissociation curve signifies decreased affinity of hemoglobin for O_2 (Fig. 2.2-8). Thus at every level of pO_2, the oxygen saturation of hemoglobin is somewhat lower than the normal curve leading to more offloading of O_2.

Causes: The factors causing right shift are:

An increase in pCO_2 shifts the curve to right, this phenomenon is known as *Bohr's effect* (Fig. 2.2-9A).

A decrease in pH of blood, as occurs in the tissues, also shifts the O_2-Hb dissociation curve to the right.

Effect of temperature. An increase in the temperature of blood shifts the curve to right.

Effect of diphosphoglycerate. The red blood cells are rich in 2, 3-diphosphoglycerate (2, 3-DPG) which is a highly charged anion that binds to β-chain of deoxygenated adult hemoglobin.

$$HbO_2 + 2, 3\text{-DPG} \rightarrow Hb\ 2, 3\text{-DPG} + O_2$$

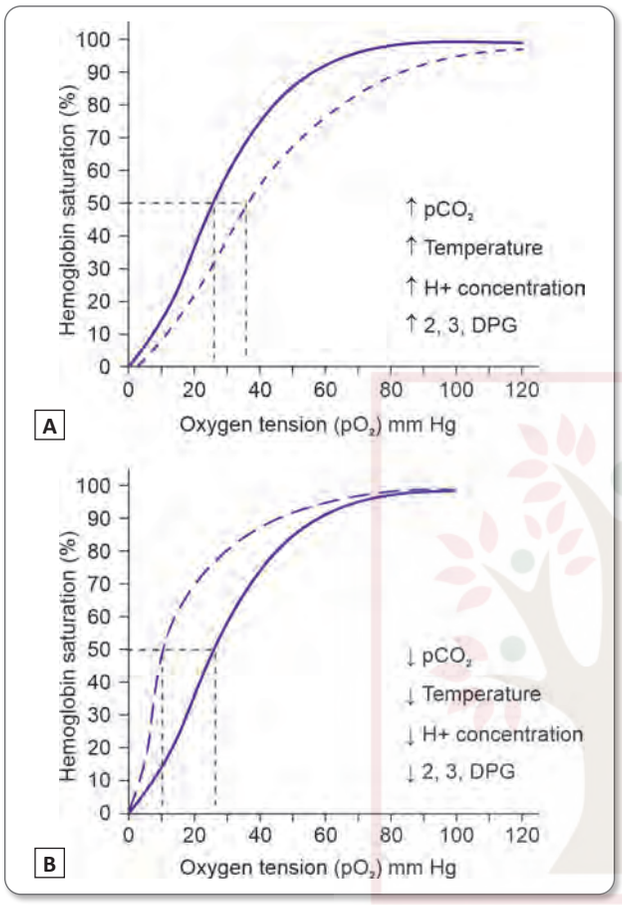
Thus, an increase in the concentration of 2, 3-DPG decreases the affinity of Hb for O_2 and shifts the normal O_2-Hb dissociation curve to the right. Causes of increased levels of 2, 3-DPG are anemia, exposure to chronic hypoxia at high altitude and certain pulmonary diseases.

Advantages versus disadvantages of right shift are:

- Right shift is advantageous in the tissues where greater O_2 is released from hemoglobin (at the same pO_2).
- However, right shift is disadvantageous in the lungs because (at the same pO_2) blood takes up less oxygen.

Shift to left

Shift to left of O_2-Hb dissociation curve signifies increased affinity of hemoglobin for O_2 (Fig. 2.2-9B). Thus, at every pO_2 level the oxygen saturation of hemoglobin is somewhat greater than in normal curve.



Figs 2.2-9A and B: Right A, and left B, shift of oxygen-hemoglobin dissociation curve

Causes for left shift of the curve are:

- Decreased pCO_2 of blood
- Increased pH of blood
- Decreased temperature
- Fetal hemoglobin.

Advantages versus disadvantages of left shift are:

Left shift of the curve has limited advantage because though it allows greater uptake of O_2 at lungs (at same pO_2), it decreases the release of O_2 to the tissues (at same pO_2).

Effect of carbon monoxide on O_2 transport

- Carbon monoxide (CO) interferes with O_2 transport because it has about 200 times the affinity for hemoglobin as compared to oxygen.
- CO combines with Hb at the same site on its molecule as O_2 and forms the *carboxyhemoglobin*, and thus decreases the functional hemoglobin concentration.

Release of Oxygen in Tissues at Rest

Oxygen Delivery

It represents the amount of O_2 that is presented to body cells per minute. It is equal to the arterial O_2 content multiplied by cardiac output. Since 100 mL of arterial blood at pO_2 of about 100 mm Hg contains about 20 mL of oxygen, thus with a cardiac output of about 5 L/min the normal oxygen delivery to the entire body is about 1 L/min. The oxygen delivery to the tissues decreases with either decrease in arterial O_2 content or decrease in cardiac output.

Oxygen Consumption

When the arterial blood with approximate pO_2 100 mm Hg reaches the tissues with tissue fluid pO_2 40 mm Hg, because of pressure gradient, about 5 mL of O_2 diffuses from the tissue capillaries to the interstitial fluid out of 100 mL of blood (containing ~ 19 mL O_2) every minute. Thus, oxygen consumption of the whole body at rest with a cardiac output of 5 L/min is about $\frac{5 \times 5000}{100}$ or 250 mL of O_2 per minute.

Utilization Coefficient

Utilization coefficient refers to the percentage of oxygen consumed out of oxygen delivered to the tissue, i.e.

$$\text{Coefficient of utilization} = \frac{\text{Oxygen consumed/min}}{\text{Oxygen delivered/min}}$$

So, at rest coefficient of utilization of whole body

$$\frac{250 \text{ mL/min}}{1000 \text{ mL/min}} \times 100 = 25\%$$

TRANSPORT OF CARBON DIOXIDE

Transport of carbon dioxide from tissue cells to the lungs occurs due to *constant circulation* of blood and diffusion of CO_2 that occurs at various sites in the direction of concentration gradient which is represented by CO_2 tension (pCO_2) differences as given below:

- Intracellular pCO_2 : 46 mm Hg
- Interstitial fluid pCO_2 : 45 mm Hg
- Arterial blood pCO_2 (in tissue capillaries) : 40 mm Hg
- Venous blood pCO_2 : 45 mm Hg
- Alveolar air pCO_2 : 40 mm Hg

From the above pCO_2 levels, it is clear that:

Diffusion of CO_2 from the cells into the interstitial fluid occurs along a tension gradient of 1 mm Hg.

Diffusion of CO_2 in blood. From the interstitial fluid the CO_2 diffuses into the capillaries at a tension gradient of 5 mm Hg.

Transport of CO_2 in the blood occurs in three forms:

1. In dissolved state (7%)
2. In bicarbonate form (70%)
3. In carbamino compound form (23%).

Release of CO_2 in lungs. From the venous blood that is supplied to pulmonary capillaries the CO_2 diffuses across the respiratory membrane into the alveoli along a tension gradient of 5 mm Hg.

Carbon dioxide dissociation curve

- Carbon dioxide dissociation curve is obtained by plotting the relationship between pCO_2 and total CO_2 content of the blood (Fig. 2.2-10).
- The graph shows that relationship between the two is nearly linear over wider range of pCO_2 (if compared with O_2-Hb dissociation curve, which is sigmoid shaped).

Rate of total CO_2 transport

In resting conditions each 100 mL of blood transports about 4 mL of CO_2 from the tissues to the lungs. Thus, with an average cardiac output of 5 L/min, a total of (4 × 5000) /100 or 200 mL of CO_2 is transported/min.

During exercise the amount of CO_2 transported increases depending upon the severity of exercise. In severe exercise, as much as 4 L of CO_2 may be transported per minute.

Respiratory quotient

Definition. Respiratory quotient (RQ) refers to the ratio of the rate of CO_2 excretion and rate of O_2 consumption per minute. It is also called *respiratory exchange ratio.*

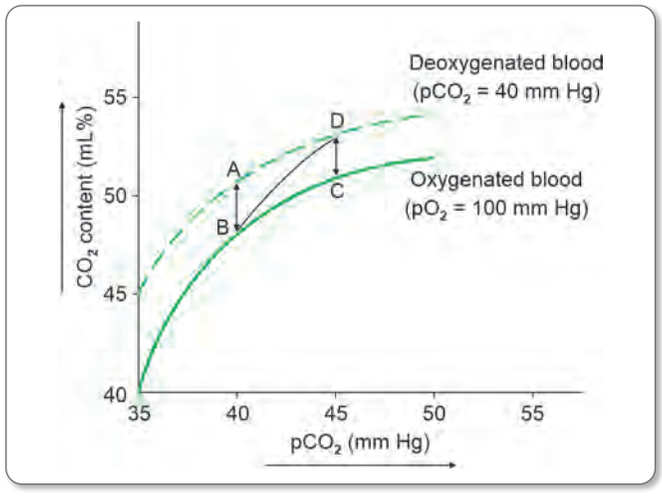


Fig. 2.2-10: Carbon dioxide dissociation curve for oxygenated (solid line) and for deoxygenated blood (dotted line) to demonstrate Haldane's effect

Normal value. Normally, the rate of CO_2 excretion is 4 mL/100 mL/min, and rate of O_2 consumption is 5 mL/100 mL/min. So, respiratory quotient = 4/5, or 0.8.

REGULATION OF RESPIRATION

Respiration is regulated by a complex integration of neural control mechanisms which are modified by certain respiratory reflexes and chemical control mechanisms.

NEURAL REGULATION OF RESPIRATION

The involuntary neural control system regulates respiration by several groups of neurons situated bilaterally in medulla and pons which include medullary respiratory centers, pontine respiratory centers, and reticular activating system (RAS).

Medullary Respiratory Centers

The medullary respiratory centers include two groups of neurons: the dorsal respiratory group (DRG) and ventral respiratory group (VRG) which generate the basic respiratory rhythm (Fig. 2.2-11A).

Pontine Respiratory Centers

The pontine centers include the apneustic center (APN) and pneumotaxic center (PNC), both of which modify the activity of medullary respiratory centers.

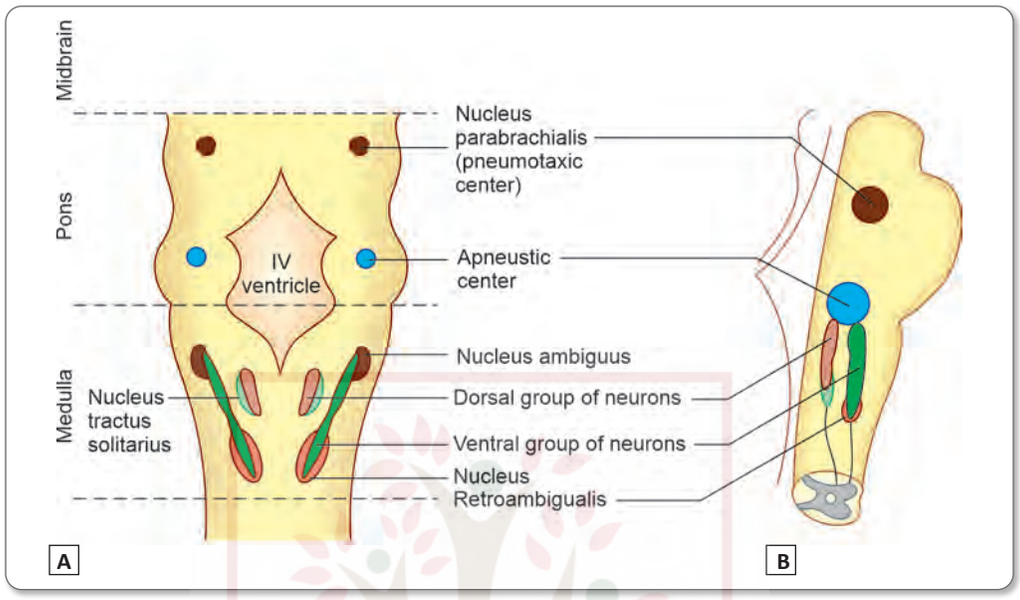
Apneustic center (APN). Refers to a group of inhibitory neurons located bilaterally in the lower part of pons (Figs 2.2-11A and B). It sends signals to neurons of DRG. This increases the tidal volume and duration of inspiration, resulting in a deeper and more prolonged inspiratory effort termed *apneusis.* However, normally the apneustic center is inhibited by impulses carried by the vagus nerves and also by the activity of pneumotaxic center.

Pneumotaxic center (PNC). The pneumotaxic centers are located bilaterally in the upper pons. They inhibit the apneustic center (APN). Therefore, stimulation of PNC shortens inspiration, leading to shallow and more rapid respiratory pattern.

Thus, though rhythm of respiration resides in DRG neurons in medulla, PNC and APN control these neurons to regulate the depth and rate of respiration.

Reticular Activating System

The reticular activating system (RAS) stimulates the respiratory centers to increase respiratory drive. During sleep, RAS activity diminishes, decreasing respiratory drive, which diminishes alveolar ventilation and results in a slight elevation of arterial CO_2 tension.



Figs 2.2-11A and B: Medullary and pontine respiratory centers. A. Front view; B. Lateral view

CHEMICAL REGULATION OF RESPIRATION

The chemical factors regulating respiration are pCO_2, pO_2 and pH of blood. These factors influence respiration in such a way that their own blood levels are maintained constant. The chemical mechanism of regulation operates through chemoreceptors.

Chemoreceptors

Chemoreceptors are the sensory nerve endings which are highly sensitive to changes in pCO_2, pO_2 and pH of blood. These are of two types:

1. Peripheral chemoreceptors
2. Central chemoreceptors.

Peripheral Chemoreceptors

Location. Peripheral chemoreceptors include the carotid and aortic bodies (Fig. 2.2-12).

Carotid body is located on either side near the bifurcation of common carotid artery.

Aortic bodies, two or more in number, are located near the arch of aorta.

Functions. The peripheral chemoreceptors respond to lowered pO_2, increased pCO_2 and increased H^+ concentration in the arterial blood. The afferent impulses from the chemoreceptors stimulate the dorsal respiratory group (DRG) neurons, which lead to an increased rate and depth of respiration called *hyperventilation.* The peripheral chemoreceptors are the only sites that detect changes in pO_2.

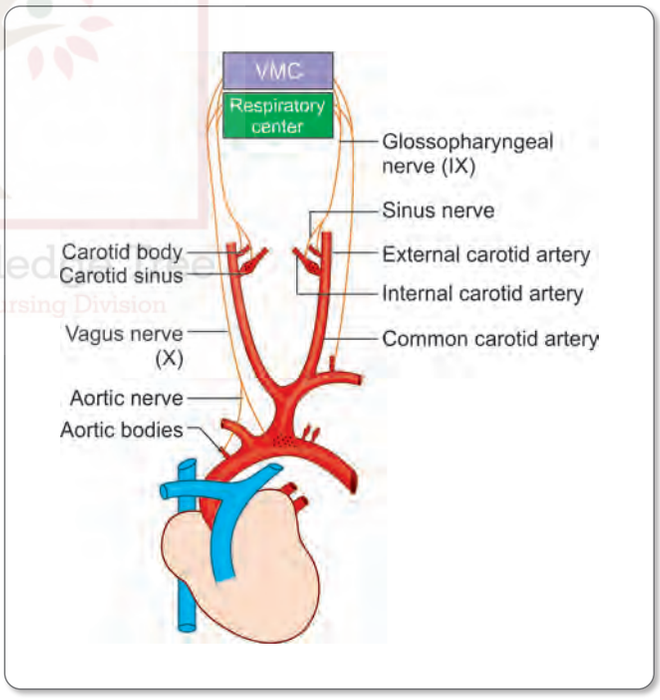
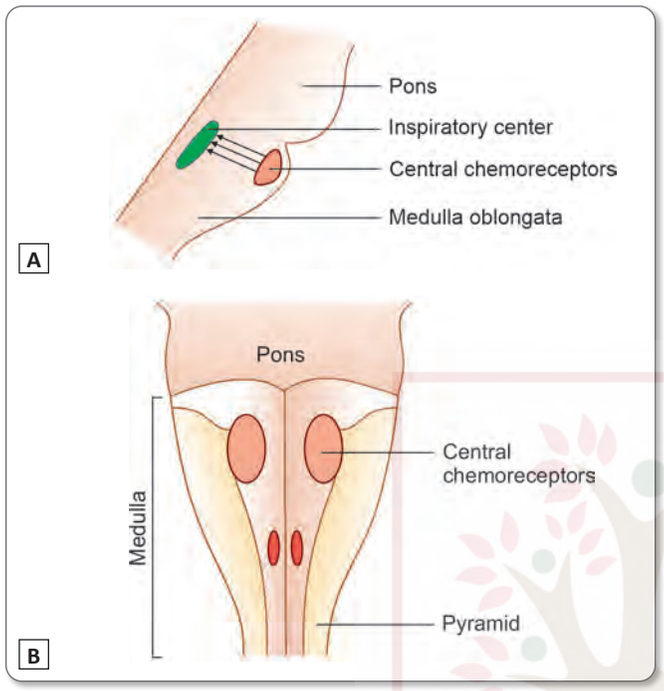


Fig. 2.2-12: Location of carotid and aortic bodies (peripheral chemoreceptors)

Central Chemoreceptors

Location. Central chemoreceptors are the cells (neurons) that lie just beneath the ventral surface of the medulla oblongata and are therefore also called medullary receptors (Figs 2.2-13A and B).



Figs 2.2-13A and B: Location of central chemoreceptors in medulla: A. Lateral view; B. Front view

Stimulation characteristics of central chemoreceptors are:

- They respond to H^+ concentration in the surrounding interstitial fluid and cerebrospinal fluid.
- The magnitude of stimulation is directly proportional to the local H^+ concentration, which in turn parallels arterial pCO_2.

OTHER FACTORS THAT INFLUENCE RESPIRATION

Afferent impulses from the receptors other than the chemoreceptors, i.e., from non-chemical receptors include the following impulse (Fig. 2.2-14):

Afferent Impulses from Pulmonary Stretch Receptors (Hering-Breuer Reflex)

The Hering-Breuer inspiratory inhibitory reflex is initiated when the stretch receptors located in the smooth muscles of the bronchi and bronchioles are stimulated by inflation of the lungs. The impulses are then sent through *vagii nerves* to pontomedullary respiratory centers to inhibit respiration. This reflex is weakest in humans. It does not play any regulatory

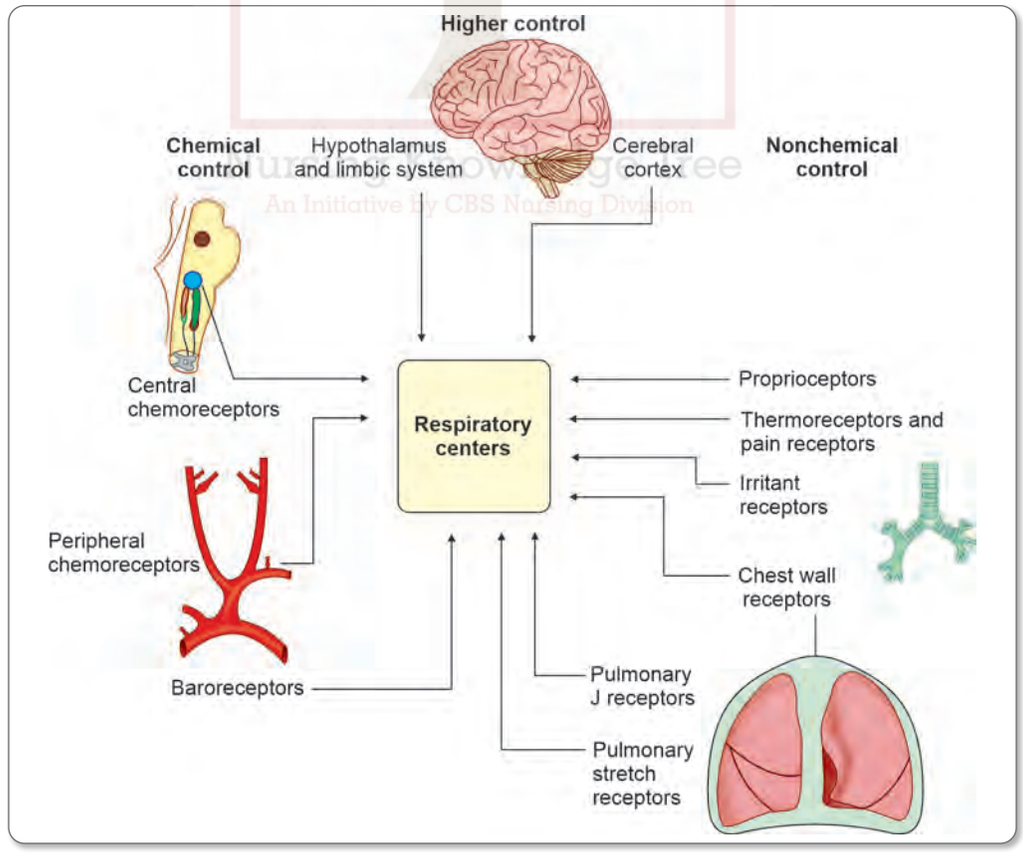


Fig. 2.2-14: Afferent impulses to respiratory centers

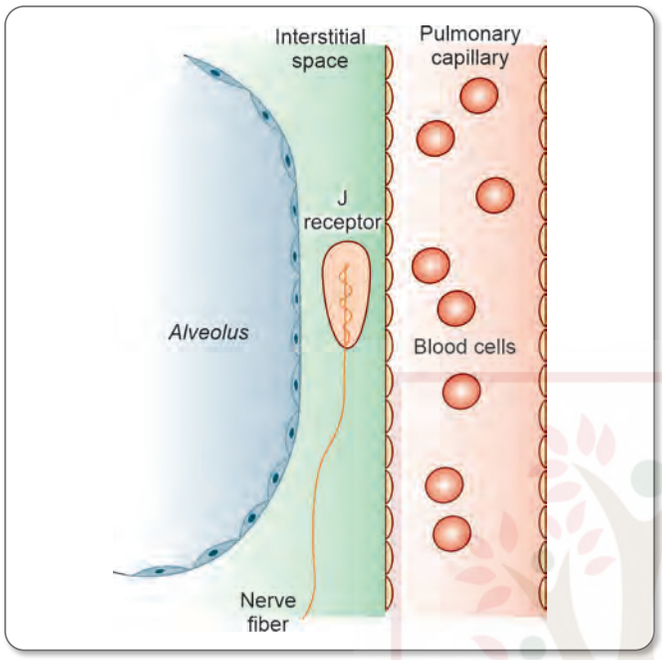


Fig. 2.2-15: Location of J receptors

role in tidal respiration. This reflex is initiated only when the tidal volume is more than 1–1.5 L. Thus, the reflex tends to limit the tidal volume.

Afferent Impulses from J-receptors

Afferent impulses from J-receptors constitute the J-reflex. The J-receptors were discovered by an Indian physiologist AS Paintal in 1954. The name J-receptors (juxtapulmonary capillary receptors) was given to them because of their location very close to the pulmonary capillaries (Fig. 2.2-15).

These receptors are primarily sensitive to increase in the content of interstitial fluid between the capillary endothelium and alveolar epithelium, therefore, they are stimulated in conditions, like pulmonary congestion, pulmonary edema, pneumonia, hyperinflation of lungs and microembolism in pulmonary capillaries.

Afferent Impulses from Proprioceptors

Proprioceptors are the receptors present in the muscles, tendons and joints and are stimulated during change in the position of different parts of the body. This reflex helps in increasing ventilation during exercise. The pediatricians employ this reflex for initiating first breath in the newborn by slapping.

Afferent Impulses from Thermoreceptors

Thermoreceptors are those receptors which are stimulated by a change in the body temperature. When warm receptors are stimulated, the impulses are conveyed to cerebral cortex via somatic afferent nerves. Cerebral cortex in turn stimulates the respiratory centers to produce hyperventilation.

Respiration helps to maintain body temperature, as some amount of heat is lost in the expired air. In dogs, panting is one of the major mechanisms of thermoregulation.

RESPIRATORY ADJUSTMENTS TO STRESSES IN HEALTH

Respiratory adjustments to stresses in health illustrate the integrated operation of the respiratory regulatory mechanisms. The stresses faced by respiration requiring adjustments in day-to-day life include:

- Respiratory adjustments during exercise.
- Respiratory adjustments at high altitude.
- Respiratory adjustments to high atmospheric pressure.

RESPIRATORY ADJUSTMENTS DURING EXERCISE

Exercise is the most frequently faced stress in day-to-day life.

Respiratory Responses to Exercise

Increase in Pulmonary Ventilation

The pulmonary ventilation increases linearly with the increases in intensity of exercise (O_2 consumption) until the anaerobic threshold is reached.

Mechanism of increased pulmonary ventilation

There are several factors which can account for the marked increase in pulmonary ventilation occurring in severe exercise are:

Neural control mechanisms play main role in increasing pulmonary ventilation during exercise.

Chemical mechanism does not play the main role in exercise hyperpnea, as the alveolar and arterial pO_2 and pCO_2 are well maintained during exercise however:

Accentuations of the normal oscillations in pO_2 and pCO_2 synchronous with respiration might stimulate the carotid body chemoreceptors and explains part of exercise hyperpnea.

Acidosis produced due to accumulation of lactic acid during severe exercise (above the aerobic threshold level) is responsible for increase in pulmonary ventilation.

Increase in Oxygen Uptake in the Lungs

The oxygen uptake by blood in the lungs increases from 250 mL/min at rest to about 4 L/min during heavy exercise. This is made possible by:

- Increased pulmonary perfusion
- Increased alveolar capillary pO_2 gradient
- Increased pulmonary diffusion capacity.

RESPIRATORY ADJUSTMENTS AT HIGH ALTITUDE

At high altitude barometric pressure is low and so the partial pressure of O_2 is also low, however, the amount of O_2 in the atmosphere is same as it is at the sea level. When a person is exposed to high altitude, particularly by rapid ascent, the different systems of the body cannot cope with the lowered O_2 tension and the effects of hypoxia start. The changes in the body at high altitude are produced mainly by:

- Hypoxia produced by low pO_2
- Expansion of gases due to fall in atmospheric pressure.

Hypoxia at High Altitude

The effects of hypoxic hypoxia produced by decreasing pO_2 at high altitude depend upon:

- The level of altitude
- Rate at which hypoxia develops, i.e., hypoxia occurs due to rapid ascent (acute hypoxia) or slow ascent (subacute hypoxia)
- Duration of exposure to hypoxia, i.e., whether short-term stay or long-term stay (chronic hypoxia).

Clinical Syndromes Caused by High Altitude

The three specific entities (clinical syndromes) which produce effects of low pO_2 at high altitude are:

- High altitude pulmonary edema (HAPO),
- Acute mountain sickness
- Chronic mountain sickness.

High altitude pulmonary edema. High altitude pulmonary edema (HAPO) usually occurs as an effect of rapid ascent at high altitude (above 10,000 ft). It is usually seen in individuals who engage in heavy physical work during first 3 –4 days after rapid ascent to high altitude due to sympathetic stimulation caused by hypoxia.

Acute mountain sickness. The characteristics of acute mountain sickness are headache, nausea, vomiting, irritability, insomnia, and breathlessness.

Chronic mountain sickness. Chronic mountain sickness (Monge's disease) occurs in some long-term residents of high altitude who develop extreme polycythemia, cyanosis, malaise, fatigue and exercise intolerance.

Physiological Compensatory Responses to High Altitude Hypoxia

Two types of physiological compensatory responses known to occur in individuals exposed to high altitude hypoxia are accommodation and acclimatization.

Accommodation refers to immediate reflex adjustments of the respiratory and cardiovascular system to hypoxia. These are:

Hyperventilation. Hyperventilation occurs due to stimulation of peripheral chemoreceptors by low O_2 tension in the arterial blood. The hyperventilation improves arterial pO_2 and reduces pCO_2.

Increase in 2, 3-diphosphoglycerate (2, 3-DPG) concentration occurs in RBCs in response to hypoxia.

Acclimatization refers to changes in the body tissues in response to long-term exposure to hypoxia. Following changes occur in the tissues:

Increase in red blood cell count (polycythemia). Occurs due to release of renal erythropoietin. This increase in RBC count leads to:

- Increase in hemoglobin concentration
- Increase in hematocrit value.

Increase in pulmonary ventilation. There is gradual increase in ventilation to an average of above five times as that of normal.

Cardiovascular changes in the form of tachycardia, and increased force of contraction of the heart.

Pulmonary hypertension. It occurs in response to pulmonary vasoconstriction.

Increase in total lung capacity and diffusion capcity of lungs.

Cellular and tissue acclimatization occur by:

- Increase in oxidative enzymes concentration
- Increase in mitochondrial density
- Increase in capillary density.

RESPIRATORY ADJUSTMENTS TO HIGH ATMOSPHERIC PRESSURE

Respiratory adjustments to high atmospheric pressure form a part of physiological problems faced by the body while going under the sea.

Physiological Problems under Depth

If the appropriate preventive measures are not taken, then the individuals working at depth in sea will have to face the following physiological problems:

Physiological Problems due to Effect of High Pressure on Respiratory Gases

Air under high atmospheric pressure is breathed under the sea. At high atmospheric pressure of air the partial pressure of oxygen (pO_2), nitrogen (pN_2) and carbon dioxide (pCO_2) is also increased producing the following physiological problems.

Effects of increased po_2 (oxygen toxicity)

Oxygen toxicity may be acute or chronic.

Acute oxygen toxicity occurs on exposures to four atmosphere pressure of oxygen (pO_2 in lungs about 3000 mm Hg).

1. *Nervous complications of acute oxygen poisoning* include disorientation, dizziness, convulsions and even coma.

2. *Irritation of airways* in the form of nasal congestion, sore throat, substernal discomfort, sneezing, coughing and bronchoconstriction

3. *Pulmonary edema and atelectasis* begin to develop after 12 hours of exposure.

4. *Bronchopneumonia* may be initiated when exposure is continued for >24 hours.

Effects of increased pN_2 (nitrogen narcosis)

Nitrogen narcosis is characterized by:

Euphoric symptoms. The individual becomes jovial and carefree. These are followed by impairment of mental functions and intelligence, individual becomes drowsy and has poor muscular coordination.

Physiological Problems of Ascent

The two physiological problems which occur when an individual ascends back to sea level after sufficient exposure to high atmospheric pressure in the deep sea are:

1. Decompression sickness
2. Air embolism.

Decompression sickness

Decompression sickness is also known as Caisson's disease, dysbarism, compressed air sickness, the bends, and diver's palsy. When the individual ascends rapidly to sea level nitrogen is decompressed and escapes from the tissues at a faster rate. Being gas it forms bubbles while escaping rapidly from the tissues. The gas bubbles block the blood vessels producing tissue ischemia and sometimes the tissue death.

Symptoms produced by escaping gas bubbles constitute the *decompression sickness*. These are:

- *Pain in joints and muscles* of legs or arms
- *Sensation of numbness*, tingling or pricking (paraesthesia) and itching
- *The chokes*—shortness of breath
- *Paralysis* of muscles
- *Coronary ischemia* or myocardial infarction
- *Neurological symptoms* like dizziness and unconsciousness may occur.

Air embolism

Air embolism is another physiological problem which may occur during rapid ascent from a depth below the sea level.

Manifestations of air embolism include chest pain, tachypnea, systemic hypotension and hypoxemia. In severe cases, air emboli may travel to the systemic circulation, block the blood flow to some vital organs and may even result in death.

DISTURBANCES OF RESPIRATION

From the physiological viewpoint, disturbances of respiration can be discussed under the following headings:

- Abnormal respiratory patterns
- Disturbances related to respiratory gases.

ABNORMAL RESPIRATORY PATTERNS

Eupnea refers to normal respiratory pattern, which implies a normal rate, rhythm and depth of respiration. Various abnormal respiratory patterns (Fig. 2.2-16) can be produced by changes in the environment or diseases affecting the respiratory system, cardiovascular system, or brain. The common altered patterns of respiration are:

Tachypnea refers to increase in the rate of respiration.

Bradypnea means decrease in the rate of respiration.

Polypnea is used to denote the rapid but shallow breathing resembling panting in dogs. In this, the rate of respiration is increased but the force does not change significantly.

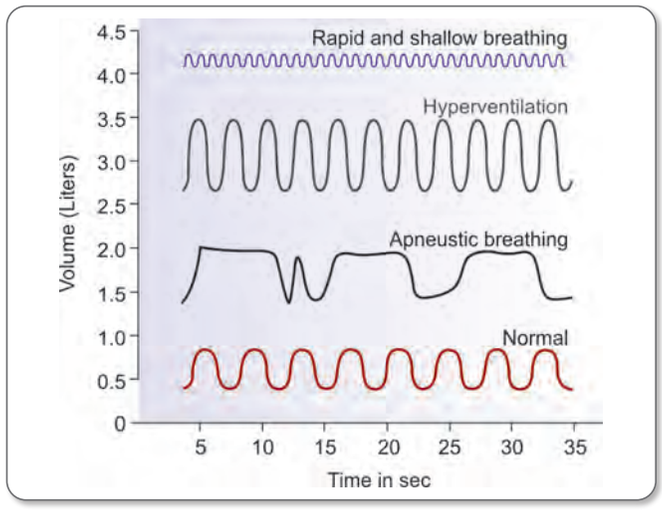


Fig. 2.2-16: Various abnormal respiratory patterns

Hypoventilation term is used to describe a decrease in rate and force of respiration.

Hyperventilation refers to increase in rate as well as force of respiration.

Hyperpnea signifies a marked increase in pulmonary ventilation due to increase in rate and/or force of respiration.

Dyspnea. When hyperpnea involves 4–5 fold increase in pulmonary ventilation, an unpleasant sensation or discomfort is felt. This type of respiration is called dyspnea.

Periodic breathing refers to a respiratory pattern characterized by alternate periods of respiratory activity and apnea.

Apnea refers to temporary cessation of breathing. Depending upon the cause apnea may be of the following types:

- *Voluntary apnea* refers to temporary arrest of breathing due to voluntary control of respiration. It is also called breath-holding.
- *Apnea after hyperventilation* occurs due to reduced stimulation of respiratory center owing to CO_2 wash caused by hyperventilation.
- *Deglutition apnea* occurs reflexly during swallowing (about 0.5 sec). There is closure of glottis (the opening between vocal cords). This effect prevents aspiration of fluid or food into the lungs
- *Breath-holding attacks* are attacks of brief period of apnea which occur in infants and young children and are generally precipitated by emotional distress.
- *Adrenaline apnea* occurs after injection of high doses of adrenaline.
- *Sleep apnea* refers to cessation of breathing for a brief period (10 sec) during sleep in normal individual.

DISTURBANCES RELATED TO RESPIRATORY GASES

Respiratory disturbances related to respiratory gases include:

- Hypoxia
- Hypercapnia
- Hypocapnia
- Asphyxia
- Carbon monoxide poisoning.

Hypoxia

The term hypoxia is used to denote deficiency of oxygen supply at the tissue level.

Causes. Hypoxia can occur because of any one or more of the following defects:

- Decreased oxygen tension (pO_2) of the arterial blood
- Decreased oxygen carrying capacity of the blood
- Decreased rate of blood flow to the tissue
- Decreased utilization of oxygen by tissue cells.

Types. Depending upon the mechanism of occurrence there are four types of hypoxia:

1. Hypoxic hypoxia
2. Anemic hypoxia
3. Stagnant hypoxia
4. Histotoxic hypoxia.

Hypoxic hypoxia occurs due to decreased oxygen tension (pO_2) of the arterial blood, hence also called *arterial hypoxia*. Therefore, in this condition O_2 carrying capacity of blood, rate of blood flow to tissue, and utilization of O_2 by tissues is normal.

Anemic hypoxia occurs due to the decreased O_2 carrying capacity of blood due to decreased hemoglobin content.

Stagnant hypoxia occurs due to decreased blood flow to the tissues so that in spite of normal pO_2 and hemoglobin, adequate O_2 is not delivered to the tissues.

Histotoxic hypoxia occurs due to decreased ability of the tissues themselves to utilize the oxygen. So, strictly speaking, it is not a true hypoxia, because O_2 supply to the tissues is adequate, e.g. as in cyanide poisoning.

Hypercapnia

Hypercapnia refers to increase in arterial pCO_2 (normal value 40 mm Hg). When hypercapnia is the primary problem, it is associated with respiratory acidosis since an increase in CO_2 promptly generates excess H^+ through:

$$H_2O + CO_2 \xrightarrow{\text{Carbonic anhydrase}} H_2CO_3 \rightarrow H^+ + HCO_3^-$$

Causes. Hypercapnia occurs due to:

Defective elimination of CO_2 as occurs in:

- Reduced pulmonary ventilation
- Reduced effective alveolar ventilation

Accidental inhalation of CO_2 in persons working in breweries and refrigeration plants.

Hypocapnia

Hypocapnia, i.e., reduced pCO_2 is usually associated with *respiratory alkalosis*, since decrease in CO_2 promptly drives the following reaction in backward direction, resulting in a decrease in H^+ concentration.

$$H_2O + CO_2 \xleftarrow{\text{Carbonic anhydrase}} H_2CO_3 \leftarrow H^+ + HCO_3^-$$

Causes. Hypocapnia occurs due to hyperventilation.

Asphyxia

Asphyxia refers to a condition in which hypoxia (decreased pO_2) is associated with hypercapnia (increased pCO_2).

Causes. Asphyxia can be general or local.

Local asphyxia occurs due to complete obstruction or ligation of blood vessel.

General asphyxia can be chronic or acute.

- *Chronic asphyxia* may occur in patient with cor pulmonale, i.e., right ventricular failure due to lung diseases.
- *Acute asphyxia* occurs due to sudden blockage in airways. Common causes are strangulation, drowning, acute tracheal obstruction (due to entry of food or due to choking) and paralysis of diaphragm, as in acute poliomyelitis.

Carbon Monoxide Poisoning

Carbon monoxide (CO) is a dangerous gas present in exhaust of gasoline engines, coal mines, gases from deep wells and underground drainage systems.

Toxic effects: Carbon monoxide produces anemic hypoxia and derangement of cellular metabolic system.

ARTIFICIAL RESPIRATION AND CARDIOPULMONARY RESUSCITATION

ARTIFICIAL RESPIRATION

Artificial respiration (AR) alone is required as an emergency life-saving procedure:

When there is sudden stoppage of breathing as seen in:

- Drowning
- Electrocution
- Anesthetic accidents
- Carbon monoxide poisoning
- Strangulation
- Accidents.

IMPORTANT TO KNOW

Artificial respiration *may also be needed when breathing is expected to stop gradually* as in paralysis of muscles in:

- Poliomyelitis
- Diphtheria
- Ascending paralysis.

It is important to note that the tissues of brain, particularly cerebral cortex, develop irreversible damage if oxygen supply is stopped for 5 minutes. So, the resuscitation must be started quickly without any delay, before the development of cardiac failure.

Methods of Artificial Respiration

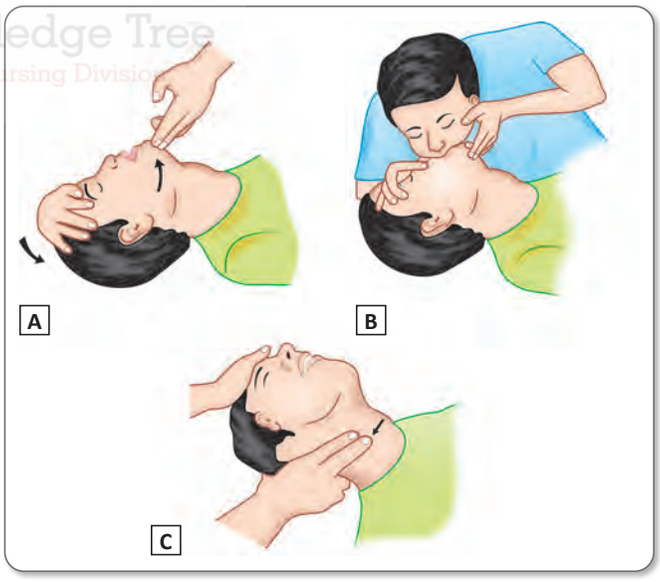
Mouth-to-mouth Breathing Method

Mouth-to-mouth breathing (exhaled air ventilation) is very useful because:

- It can be applied quickly without waiting for the availability of any aid.
- It is simple and effective measure of resuscitation.
- It can be applied in all age groups.
- It is the only technique capable of producing adequate ventilation.
- It also works by expanding the lungs.

Procedure of mouth-to-mouth breathing is as:

- To begin with patient's neck is extended by placing one hand under the neck and lifting it and pressing the forehead with the other hand (Fig. 2.2-17A). This prevents the flaccid tongue from falling back into the pharynx.
- Then the patient's nostrils are closed by the thumb and index finger of the hand (Fig. 2.2-17B).
- The resuscitator then takes a deep breath and exhales air into the patient's airway after tightly placing his mouth over patient's mouth, and noting the expansion of the chest at the same time. The volume of the air exhaled must be twice the normal tidal volume. This expands the patient's lungs.
- Then the resuscitator removes his mouth from that of the patient, allowing expiration to occur passively due to the elastic recoil of the lungs and chest (Fig. 2.2-17C).
- The above procedure is repeated 12–16 times per minute till spontaneous breathing returns, or till the patient is shifted to a hospital.



Figs 2.2-17A to C: Mouth-to-mouth breathing: A. The neck is extended by placing one hand under the neck and pressing the forehead with other hand; B. Nostrils are closed with thumb and index finger and resuscitator exhales into the patient's airway by tightly placing his mouth over the patients mouth; C. Allows the patient to exhale passively by unsealing nose and mouth

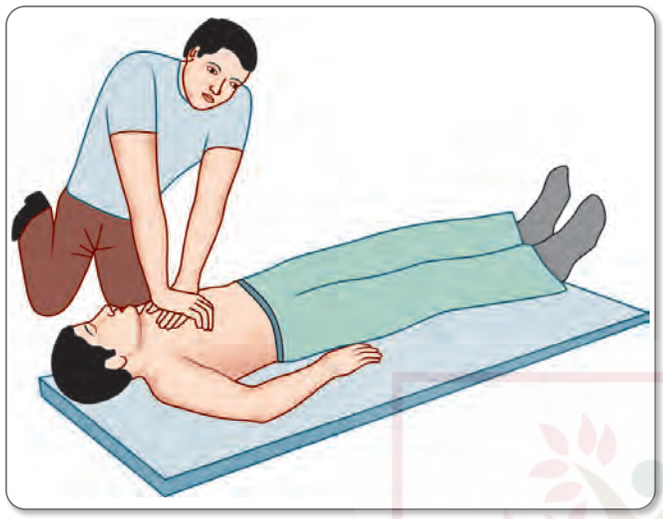


Fig. 2.2-18: Procedure showing external cardiac massage

CARDIOPULMONARY RESUSCITATION

Cardiopulmonary resuscitation (CPR) is required in some patients when heart and respiration both stop. Breathing usually stops before the heart stops, so artificial respiration should be started immediately.

Emergency Plan of Cardiopulmonary Resuscitation

The following plan called ABC of CPR has proved useful in reviving such patients:

A *Airway care*

B *Breathing* by artificial respiration (AR) method.

C *Cardiac massage* is required when carotid pulse cannot be felt (Fig. 2.2-18).

Conventional CPR is performed by 30 chest compression followed by 2 breaths (one cycle). The depth of compression should be minimum 2 inches for adults. For effective CPR rescuer should perform 5 cycles within 2 minutes.

NURSING IMPLICATIONS AND APPLICATIONS

A nurse must know and learn the methods of artificial respiration and cardiopulmonary resuscitation.

PULMONARY FUNCTION TESTS

Pulmonary function tests can be classified into the following groups:

- Ventilatory function tests
- Tests of diffusion
- Tests of ultimate purpose of respiration
- Tests during exercise.

VENTILATORY FUNCTION TESTS

Ventilatory function tests are meant for assessment of the expansion of lungs and chest wall; and for assessment of restrictive and obstructive ventilatory defects. The assessment of ventilatory functions can be accomplished by measurement of various lung volumes and capacities by spirometry.

Spirometry refers to recording of volume changes during various clearly defined breathing maneuvers. It can be performed using a simple spirometer, a modified spirometer called respirometer or computerized spirometer.

Simple spirometer (Fig. 2.2-19) is made of metal. It consists of the following parts:

- *Outer chamber* or container which is filled with water.
- *Floating drum* or a gas bell, with 6 L capacity, floats in the water in an inverted manner. It is attached to a chain which passes over a pulley bearing a balancing weight and a writing needle (pen). The needle (pen) moves with the movement of the floating drum. The floating drum is thus counterpoised and has very little inertia and friction.
- *Inner chamber* is open at the top end which lies above the water level in outer chamber and is connected to a tube at the bottom end. At the end of tube, a mouthpiece is attached through which the subject is made to respire.
- *Kymograph* is a recording drum on which the movements of the needle are recorded.

TESTS OF DIFFUSION

Pulmonary diffusion refers to transfer of gases from alveoli to capillary blood across the respiratory membrane. The

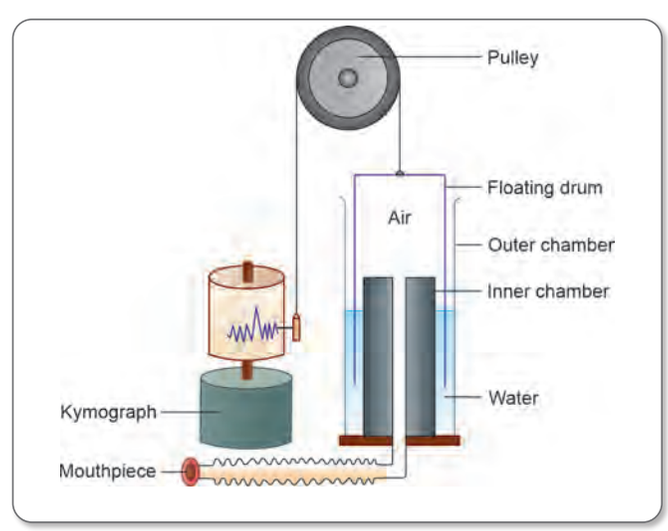


Fig. 2.2-19: Simple spirometer

exchange of gases in the lungs was earlier believed to be dependent merely on the ability of the gases to diffuse across the respiratory membrane. This term led to the use of term *diffusion capacity.* Nowadays, the term *transfer factor,* rather than diffusion capacity, is used.

TESTS OF ULTIMATE PURPOSE OF RESPIRATION

Since the ultimate purpose of respiration is to supply O_2 from atmosphere to tissues and removal of CO_2 from the tissues into the atmosphere; so, the estimation of arterial blood pO_2, pCO_2 and pH (blood gas analysis) are most fundamental of all the pulmonary function tests.

ASSESS YOURSELF

Short and Long Answer Questions

1. **Describe briefly:**
   - Mechanism of tidal respiration
   - Factors affecting vital capacity
   - Transport of oxygen from lungs into the tissues
2. **Define the following:**
   - Functional residual capacity
   - Pulmonary elastance
   - Diffusion capacity
3. **Draw diagram to show:**
   - Hemoglobin–oxygen dissociation curve.
   - Layers of respiratory membrane
4. **Differentiate between;**
   - Intrapleural and intrapulmonary pressure
   - Expired and alveolar air
   - Pulmonary and alveolar ventilation
   - Peripheral and central chemoreceptors
5. **Write notes on:**
   - Surfactant
   - Hypoxia
   - Carbon monoxide poisoning
   - Decompression sickness
   - Oxygen toxicity

Multiple Choice Questions

1. **True statement for role of diaphragm in breathing:**
   a. During expiration dome gets flattened
   b. Descent raises intra-abdominal pressure
   c. Descent raises intrapleural pressure
   d. Descent raises intrapulmonary pressure
2. **The true statement about expiration in quite breathing:**
   a. Intrapulmonary pressure becomes positive
   b. Intrapulmonary pressure becomes negative
   c. Intrapleural pressure is at atmospheric
   d. Intrapleural pressure becomes negative
3. **During tidal expiration there is:**
   a. Contraction of external intercostals muscles
   b. Decrease in thoracic cavity
   c. Relaxation of internal intercostals muscles
   d. Contraction of internal intercostals muscles
4. **Vital capacity is equal to:**
   a. TV + IRV
   b. TV + IRV + ERV
   c. TV + IC
   d. TV + IC + EC
5. **In which of the following condition vital capacity increases:**
   a. Gravity
   b. Old age
   c. In swimmers
   d. Pregnancy
6. **The normal value of FEV_1 is:**
   a. 98–100%
   b. 90%
   c. 80%
   d. 70%
7. **The partial pressure of oxygen (pO_2) of atmospheric air is:**
   a. 600 mm Hg
   b. 160 mm Hg
   c. 47 mm Hg
   d. 4 mm Hg
8. **Hemoglobin–oxygen dissociation curve shifts to the left when:**
   a. Increased pCO_2
   b. Acidic pH
   c. Decrease pO_2
   d. Decrease in body temperature
9. **The peripheral chemoreceptors are most sensitive to:**
   a. Increased pCO_2
   b. Decrease pO_2
   a. H^+ concentration
   b. HCO_3^- concentration
10. **J receptors mainly respond to:**
    a. Increased pCO_2
    b. Decrease pO_2
    c. H^+ concentration
    d. Increased content of interstitial fluid
11. **During exercise, pulmonary ventilation increases because of:**
    a. Hypoxia
    b. Acidosis
    c. Increased pulmonary perfusion
    d. Oxygen debt
12. **At high altitude, hyperventilation occurs due to:**
    a. Increased in level of 2–3 diphosphoglycerate
    b. Polycythemia
    c. Pulmonary hypertension
    d. Increase in oxidative enzymes

13. **Which of the following is not the feature of decompression sickness:**
    a. Numbness
    b. Myocardial infarction
    c. Pain in joints
    d. Increased total lung capacity
14. **Oxygen carrying capacity is low but blood flow to the tissues is normal in:**
    a. Anemic hypoxia
    b. Hypoxic hypoxia
    c. Histotoxic hypoxia
    d. Stagnant hypoxia
15. **Asphyxia occurs due to:**
    a. Carbon monoxide poisoning
    b. Nitrogen narcosis
    c. Strangulation
    d. Chronic heart failure
16. **Which ventilatory function is not done by spirometry:**
    a. Vital capacity
    b. Timed vital capacity
    c. Functional residual capacity
    d. Maximum breathing capacity

ANSWER KEY

1.	b	**2.**	a	**3.**	b	**4.**	b	**5.**	c	**6.**	c	**7.**	b	**8.**	d
9.	b	**10.**	d	**11.**	b	**12.**	a	**13.**	d	**14.**	a	**15.**	c	**16.**	c

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