

Compliance

Compliance is the ability of the lungs and thorax to expand. It is defined as the change in volume per unit change in the respiratory pressure. Determination of compliance is useful as it is the measure of stiffness of lungs. Stiffer the lungs, less is the compliance.

If lungs are removed from thorax, the expansibility (compliance) of lungs alone is doubled. It is because of the absence of the inactivity and the restriction exerted by the structures of thoracic cage, which interfere with expansion of lungs.

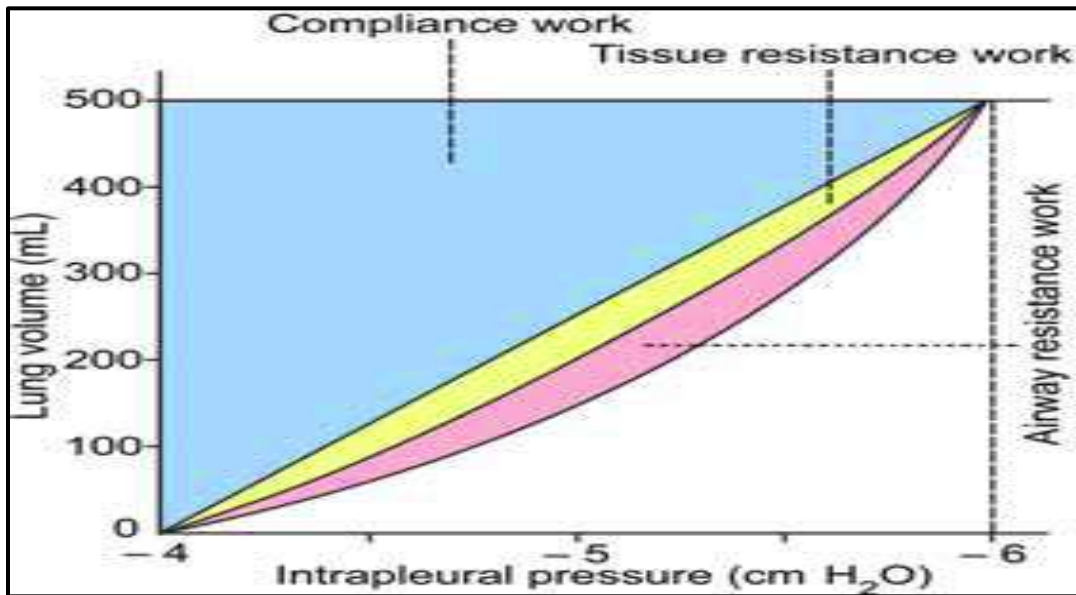
The work of breathing

It is the work done by the respiratory muscles during breathing to overcome the resistance in the thorax and respiratory tract.

During the respiratory processes, inspiration is active process and the expiration is a passive process. So, during quiet breathing, the respiratory muscles perform the work only during inspiration and not during expiration.

The energy obtained during the work of breathing is utilized to overcome three types of resistance:

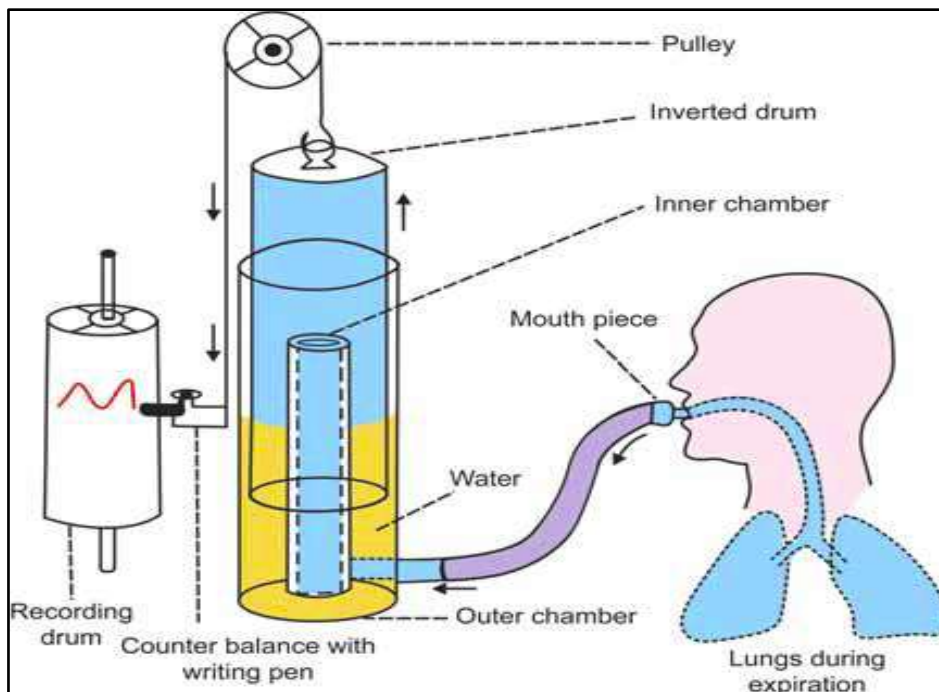
1. Airway resistance
2. Elastic resistance of lungs and thorax (compliance work).
3. Non-elastic viscous resistance (tissue resistance work).



Work of breathing

Pulmonary function tests

Pulmonary or lung function tests are useful in assessing the functional status of the respiratory system. These tests involve measurement of lung volumes and capacities.



Spirometer: During expiration, the air enters the spirometer from lungs. The inverted drum moves up and the pen draws a downward curve on the recording drum.

The air in lung is classified into two divisions:

1. Lung volumes.
2. Lung capacities.

Pulmonary function tests are carried out mostly by using spirometer. The graphical recording of lung volumes and capacities is called spirogram.

Lung volume

Lung volumes are the static volumes of air breathed by an individual. The lung volumes are of four types:

1. Tidal volume (TV)

Tidal volume is the volume of air breathed in and out of lungs in a single normal quiet respiration. Tidal volume signifies the normal depth of breathing.

Normal value = 500 mL (0.5 L).

2. Inspiratory reserve volume (IRV)

Inspiratory reserve volume is an additional volume of air that can be inspired forcefully after the end of normal inspiration.

Normal value = 3300 mL (3.3 L).

3. Expiratory reserve volume (ERV)

Expiratory reserve volume is the additional volume of air that can be expired out forcefully, after normal expiration.

Normal value = 1000 mL (1 L).

4. Residual volume (RV)

Residual volume is the volume of air remaining in the lungs even after forced expiration. Normally, lungs cannot be emptied completely even by forceful expiration. Some quantity of air always remains in the lungs even after the forced expiration.

Normal value = 1200 mL (1.2 L).

Lung capacity

Lung capacities are the combination of two or more lung volumes. Lung capacities are of four types:

1. Inspiratory capacity (IC)

Inspiratory capacity is the maximum volume of air that is inspired after normal expiration. It includes tidal volume and inspiratory reserve volume.

$$IC = TV + IRV = 500 + 3300 = 3800 \text{ mL.}$$

2. Vital capacity (VC)

It is the maximum volume of air that can be expelled out forcefully after a deep (maximal) inspiration. Vital capacity includes inspiratory reserve volume, tidal volume and expiratory reserve volume.

$$VC = IRV + TV + ERV = 3300 + 500 + 1000 = 4800 \text{ mL.}$$

3. Functional residual capacity (FRC)

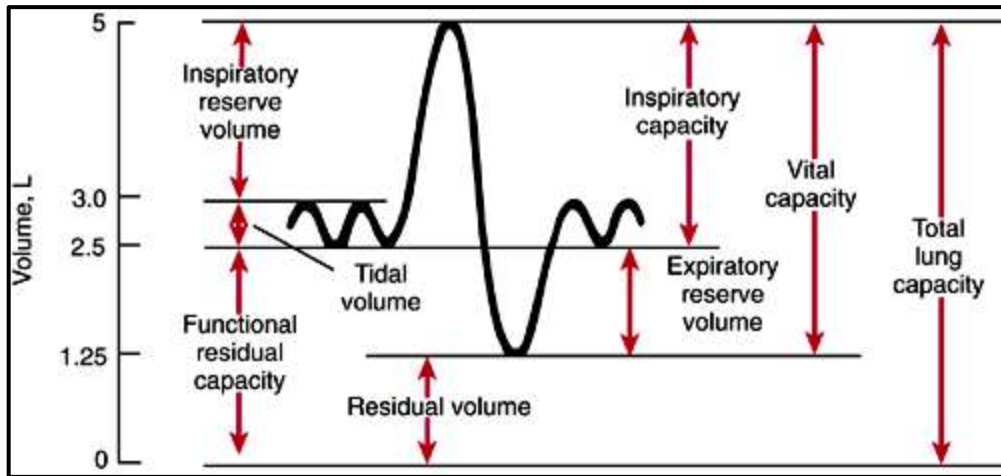
It is the volume of air remaining in the lungs after normal expiration (after normal tidal expiration). Functional residual capacity includes expiratory reserve volume and residual volume.

$$FRC = ERV + RV = 1000 + 1200 = 2200 \text{ mL.}$$

4. Total lung capacity (TLC)

Total lung capacity is the volume of air present in the lungs after a deep (maximal) inspiration. It includes all the volumes.

$$TLC = IRV + TV + ERV + RV = 3300 + 500 + 1000 + 1200 = 6000 \text{ mL.}$$



Respiratory volume and capacity

Ventilation

Pulmonary ventilation

It is the volume of air moving in and out of lungs per minute in quiet breathing. It is also called respiratory minute volume (RMV).

Normal value and calculation

Normal value of pulmonary ventilation is 6 L/minute. It is the product of tidal volume (TV) and the rate of respiration (RR). It is calculated by the formula:

$$\begin{aligned}
 \text{Pulmonary ventilation} &= \text{Tidal volume} \times \text{Respiratory rate} \\
 &= 500 \text{ mL} \times 12/\text{minute} \\
 &= 6,000 \text{ mL} = 6 \text{ L/minute.}
 \end{aligned}$$

Factors affecting pulmonary ventilation:

1. Surface tension of alveolar fluid (Surfactant)
2. Lung compliance:
 - a. Elasticity.
 - b. Surface tension
3. Airway resistance.

Alveolar ventilation

Alveolar ventilation is the amount of air utilized for gaseous exchange every minute.

Alveolar ventilation is different from pulmonary ventilation. In pulmonary ventilation, 6 L of air moves in and out of lungs in every minute. But the whole volume of air is not utilized for exchange of gases. The volume of air subjected for exchange of gases is the alveolar ventilation. The air trapped in the respiratory passage (dead space) does not take part in gaseous exchange.

Normal value of alveolar ventilation is 4,200 mL (4.2 L)/ minute

Dead space

Dead space is defined as the part of the respiratory tract, where gaseous exchange does not take place. The air present in the dead space is called dead space air.

Dead space is of two types:

- I. Anatomical dead space.
- II. Physiological dead space.

Physiological Dead Space

Physiological dead space includes anatomical dead space plus two additional volumes:

1. The air in the alveoli, which are nonfunctioning. In some of the respiratory diseases, alveoli do not function because of dysfunction or destruction of alveolar membrane
2. The air in the alveoli, which do not receive adequate blood flow. Gaseous exchange does not take place during inadequate blood supply.

Normal value and measurement of dead space

Under normal conditions, the physiological dead space is equal to anatomical dead space. It is because, all the alveoli are functioning and all alveoli receive adequate blood flow in normal conditions. *The volume of normal dead space is 150 ml.*

In respiratory disorders, which affect the pulmonary blood flow or the alveoli, the dead space increases. It is associated with reduction in alveolar ventilation. *The dead space is measured by single breath nitrogen washout method.*

Nervous mechanism of respiration

Respiration is a reflex process. But it can be controlled voluntarily also. Voluntary arrest of respiration (voluntary apnea) is possible only for a short period of about 40 seconds. However, by practice, breathing can be withheld for a long period. At the end of that period, the person is forced to breathe. Though, normally, the quiet regular breathing takes place because of regulatory mechanisms.

Respiration is regulated by two mechanisms:

A. Nervous or neural mechanism: Nervous mechanism that regulates respiration includes respiratory centers, afferent nerves and efferent nerves.

► **Respiratory centers** are group of neurons, which control the rate, rhythm and force of respiration. These centers are bilaterally situated in reticular formation of brainstem. The respiratory centers receive afferent impulses from different parts of the body and, modulate the movements of thoracic cage and lungs accordingly through efferent nerve fibers.

B. Chemical mechanism:

The chemical mechanism of respiratory regulation is operated through the chemoreceptors which give response to chemical changes in blood such as:

1. Hypoxia {decreased partial pressure of O_2 in blood (PO_2)}
2. Hypercapnea {increased partial pressure of CO_2 in blood (PCO_2)}
3. Increased hydrogen ion concentration.

Types of Chemoreceptors

Chemoreceptors are classified into two groups:

1. Central chemoreceptors: The chemoreceptors are present in the brain, situated in medulla oblongata, close to dorsal respiratory group of neurons. The main stimulant for the central chemoreceptors is the increased hydrogen ion concentration.

If hydrogen ion concentration increases in the blood, it cannot stimulate the central chemoreceptors because, the hydrogen ions from blood cannot cross the blood-brain barrier and blood cerebrospinal fluid barrier.

On the other hand, if carbon dioxide increases in the blood, it can easily cross the blood-brain barrier and blood cerebrospinal fluid barrier and enter the interstitial fluid of brain or the cerebrospinal fluid. There, the carbon dioxide combines with water to form carbonic acid. Since carbonic acid is unstable, it immediately dissociates into hydrogen ion and bicarbonate ion.

The hydrogen ions stimulate the central chemoreceptors. Chemoreceptors in turn send stimulatory impulses to dorsal respiratory group of neurons causing increased ventilation (increased rate and force of breathing). Because of this, the excess carbon dioxide is washed out and the respiration is brought back to normal.

2. Peripheral chemoreceptors: Chemoreceptors present in the carotid and aortic region are called peripheral chemoreceptors. Reduction in partial pressure of oxygen is the most potent stimulant for the peripheral chemoreceptors; but these receptors are mildly sensitive to the increased partial pressure of carbon dioxide and increased hydrogen ion concentration.

The relationship between oral health and respiratory disease

- ▶ The relationship between oral health and systemic conditions, including the association between poor oral hygiene, periodontal disease, and respiratory disease, has been increasingly debated over recent decades. Oral bacteria and, especially, periodontal pathogens have been implicated as important agents with regard to causing other illnesses including respiratory diseases
- ▶ Four possible mechanisms to explain the biological plausibility of an association between oral conditions and nosocomial respiratory infections have been described:

1. Oral pathogens directly aspirated into the lungs.

The most common respiratory pathogens are found within the dental plaque inside the oral cavity. These bacteria, once established in the mouth, can be aspirated into the lungs and cause infection.

2. Salivary enzymes associated with periodontal disease modify respiratory tract mucosal surfaces and promote adhesion and colonization by respiratory pathogens, with consequent aspiration into the lungs thereby causing infection.

3. Hydrolytic enzymes from periodontopathic bacteria may destroy the salivary film that protects against pathogenic bacteria. This may reduce the ability of mucins to adhere to pathogens, thus leaving them free to adhere to mucosal receptors in the respiratory tract.

4. The presence of a large variety of cytokines and other biologically active molecules continually released from periodontal tissues and peripheral mononuclear cells, in case of untreated periodontitis, may alter the respiratory epithelium and promote colonization by respiratory pathogens, thereby resulting in infection.