Respiratory system

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 (end expiratory position). It includes tidal volume and inspiratory reserve volume

IC= TV+IRV=500+3300- 3800 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 mL.

3. Functional residual capacity (FRC)

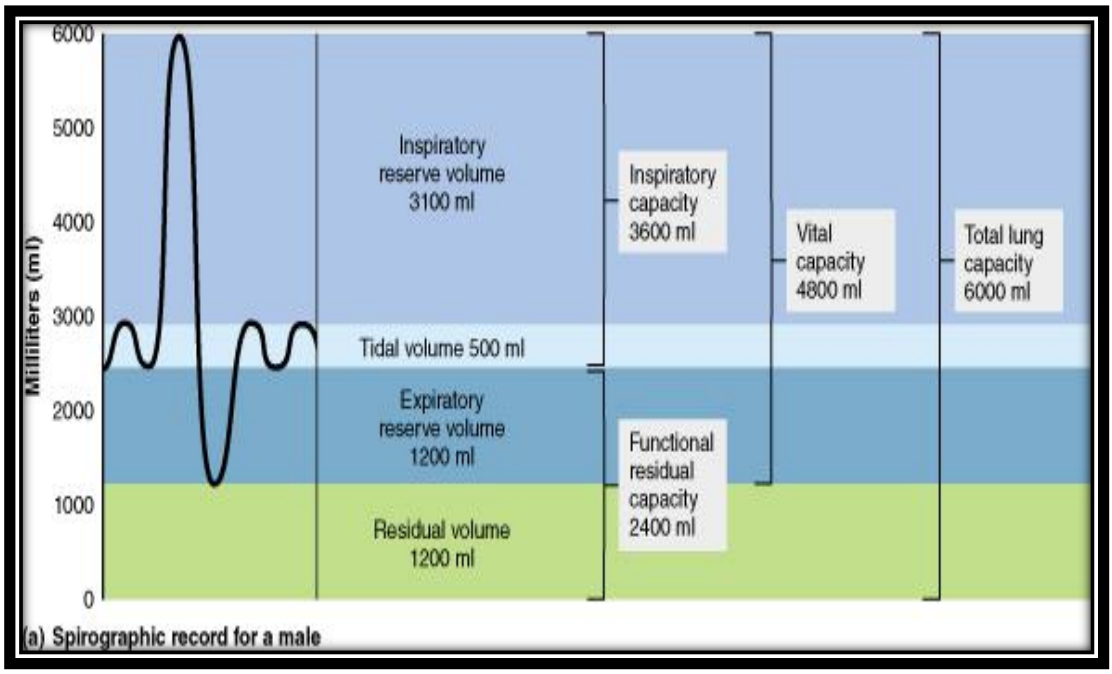
It is the volume of air remaining in the lungs after normal expiration (after normal tidal expiration). Functional residual capacity includes expiratory reserve volume and residual volume

FRC=ERV+RV=1000 +1200=2200 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 mL



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: **Pulmonary ventilation=Tidal volume x Respiratory rate**

=500 mL x 12/minute

=6,000mL=6 L/minute

Factors affecting pulmonary ventilation:

1. Surface tension of alveolar fluid

Surfactant (Pulmonary surfactant is a surface acting material that decreases the surface tension on the alveolar membrane).

2. Lung compliance

- Elasticity
- 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 the anatomical dead space volumes: plus two additional

1. The air in the alveoli, which are nonfunctioning. 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.

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. A considerable number of hypotheses have sought to explain the possible role of oral bacteria in the pathogenesis of respiratory diseases, and some clinical and epidemiological studies have found results favoring such an association.
- 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. 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 cases of untreated periodontal disease in high-risk individuals, the presence of a large variety of cytokines and other biologically active molecules continually released from periodontal tissues and peripheral mononuclear cells may alter the respiratory epithelium and promote colonization by respiratory pathogens, thereby resulting in infection.