LUNG VOLUMES AND CAPACITIES

1. The ventilation of the lungs can be divided between some arbitrary boundaries which separate different phases or zones of respiration. These boundaries are called ventilatory boundaries and these zones of respiration are classified into '*volumes*' and '*capacities*'.
2. The difference between a volume and a capacity is the simple fact that a capacity is the sum of more than one volumes.
3. The different volumes and capacities are: (Fig. 19.1)
   a. *Tidal volume* (500 mL)—This is the volume of air inspired or expired during normal breathing, i.e. the volume between the normal end-inspiratory and end-expiratory points.
   b. *Inspiratory reserve volume* (2–3 liters)—It is the volume of air that can be maximally (forcefully) inspired after a normal inspiration.
   c. *Expiratory reserve volume* (1300 mL)—It is the volume of air that can be maximally (forcefully) expired after a normal expiration.
   d. *Residual volume* (1200 mL)—It is the volume of air remaining in the lungs even after forceful expiration.
   e. *Inspiratory capacity* (2500–3500 mL)—Inspiratory reserve volume + Tidal volume.
   f. *Expiratory capacity* (1800 mL)—Expiratory reserve volume + Tidal volume.
   g. *Functional residual capacity* (2500 mL)—Expiratory reserve volume + Residual volume.

      It is the amount of air remaining in the lungs after a normal expiration.

      It is the '*resting respiratory level*' at which the lungs are under least tension after a normal expiration. At this point, forces expanding and collapsing the lungs are equal.
   h. *Vital capacity* (4–4.8 liters)—Inspiratory reserve volume + Tidal volume + Expiratory reserve volume (Vide infra).
   i. *Total lung capacity* (5–6 liters) – Vital capacity + Residual volume. It is the amount of air present in the lungs after a maximal inspiration.
4. a. Of all the volumes, *residual volume cannot be measured by a spirometer.* Along with this, the capacities incorporating residual volume, such as, functional residual capacity and total lung capacity cannot be measured by a spirometer.

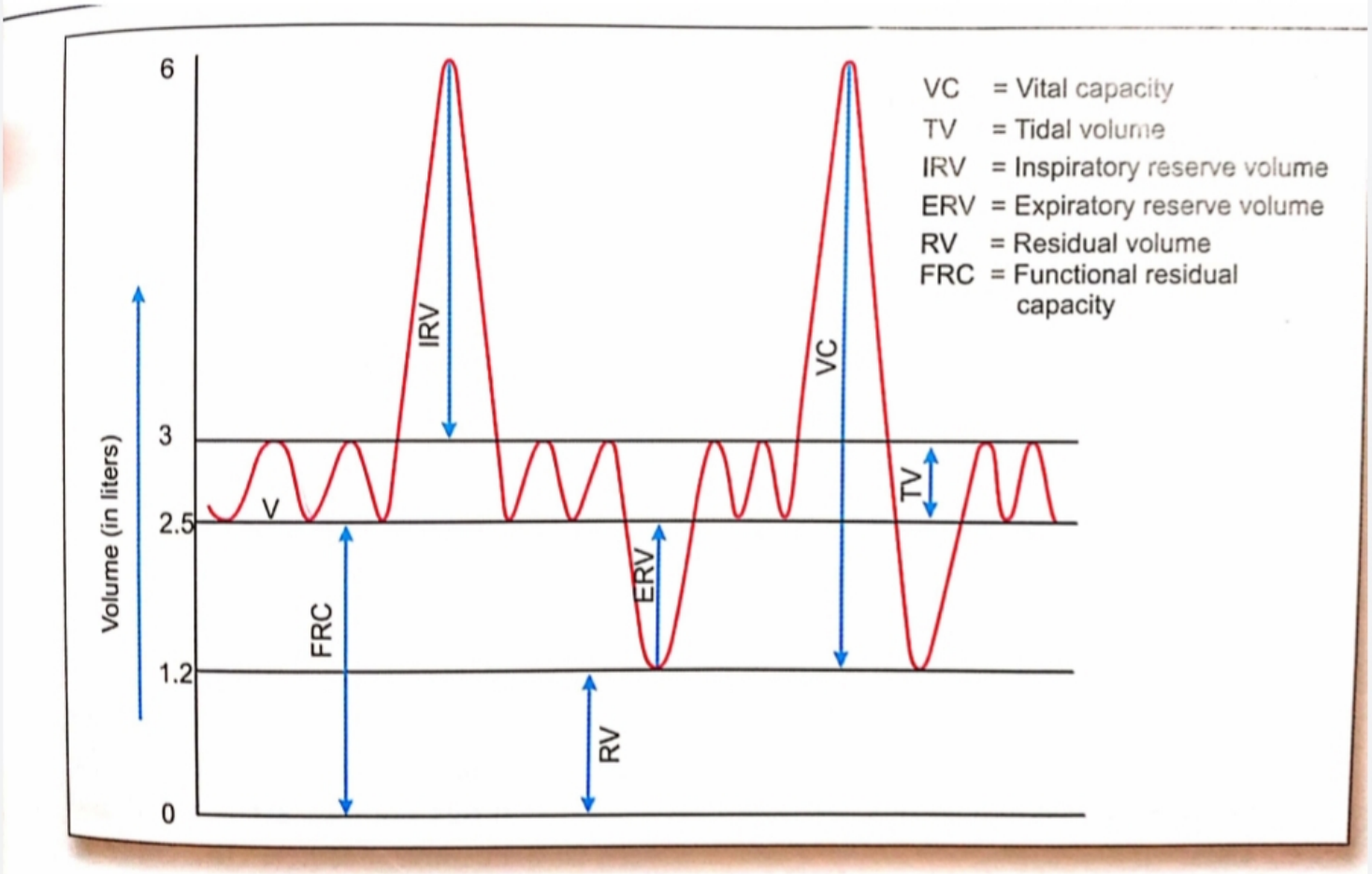


Fig. 19.1: Different volumes and capacities of lungs

well

The abbreviations those are commonly used in the spirometric measurements are given in the Table below :

Terms	Symbols	Descriptions
Vital capacity	VC	Maximal volume of air exhaled after forced inspiration (includes TV, IRV and ERV).
Tidal volume	TV	Volume of air inhaled or exhaled during quiet breathing
Inspiratory reserve volume	IRV	Maximal air that can be inhaled after a quiet inspiration.
Expiratory reserve volume	ERV	Maximal air that can be breathed out after quiet expiration.
Residual volume	RV	Volume of air remaining in lungs after full expiration.
Inspiratory vital capacity	IVC	Maximal volume of air inhaled after full expiration.
Forced expiratory volume, per time interval in seconds	FEV_t	Volume of air exhaled in a given period during a complete forced expiration (FVC).
Maximal expiratory flow rate	MEFR	Volume of air exhaled per second measured between 230 ml and 1,200 ml volumes of the forced expiratory Spirogram.
Maximal mid-expiratory flow	MMEF	Volume of air per second exhaled during middle half of expired volume of forced expiratory spirogram.
Maximal voluntary ventilation	MVV	Maximum breathing capacity—litre/minute. Subject can breathe with maximal voluntary effort (actual measurement for twelve seconds).

5. Vital capacity:
   a. It is the amount of air that can be forcefully expired after taking a maximal (forceful) inspiration.
   b. Normal value is 4–4.8 liters.
   c. Uses:
      - Vital capacity gives some idea about the physical fitness of an individual.
      - Vital capacity may help in identifying presence of disease although it has limited diagnostic value.
      - Vital capacity has prognostic value, as serial estimations of vital capacity may indicate the progress of disease.
   d. Factors influencing vital capacity:
      - *Age*—Low in extremes of age
      - *Sex*—Lower in females
      - *Posture*—Vital capacity is reduced in supine posture, compared to its value in standing posture. This is because,
        - In supine posture, the weight of the abdominal viscera, e.g. liver and spleen, pushes the diaphragm upwards. This restricts respiration.
        - Due to increased venous return in supine posture, the lungs contain extra blood and become heavier. This reduces respiratory excursions of lung and diminishes vital capacity.
      - *Training*—In athletes and sportsmen undergoing regular physical training, vital capacity is increased.
      - *Disease*—Vital capacity is decreased in various lung diseases.
6. Timed vital capacity: (Fig. 19.3)
   a. The time-related forceful expulsion of air, i.e. the amount of air forcefully expelled in 1, 2 or 3 seconds after a forceful inspiration is known as timed vital capacity.
   b. These are also known as 'Forced expiratory volume' in 1, 2 or 3 seconds or FEV_1, FEV_2, FEV_3 respectively.
   c. These, particularly FEV_1, have important diagnostic value when determined along with FVC or 'Forced vital capacity' (vital capacity performed forcefully).
      Normally, FEV_1 = 80% of FVC.

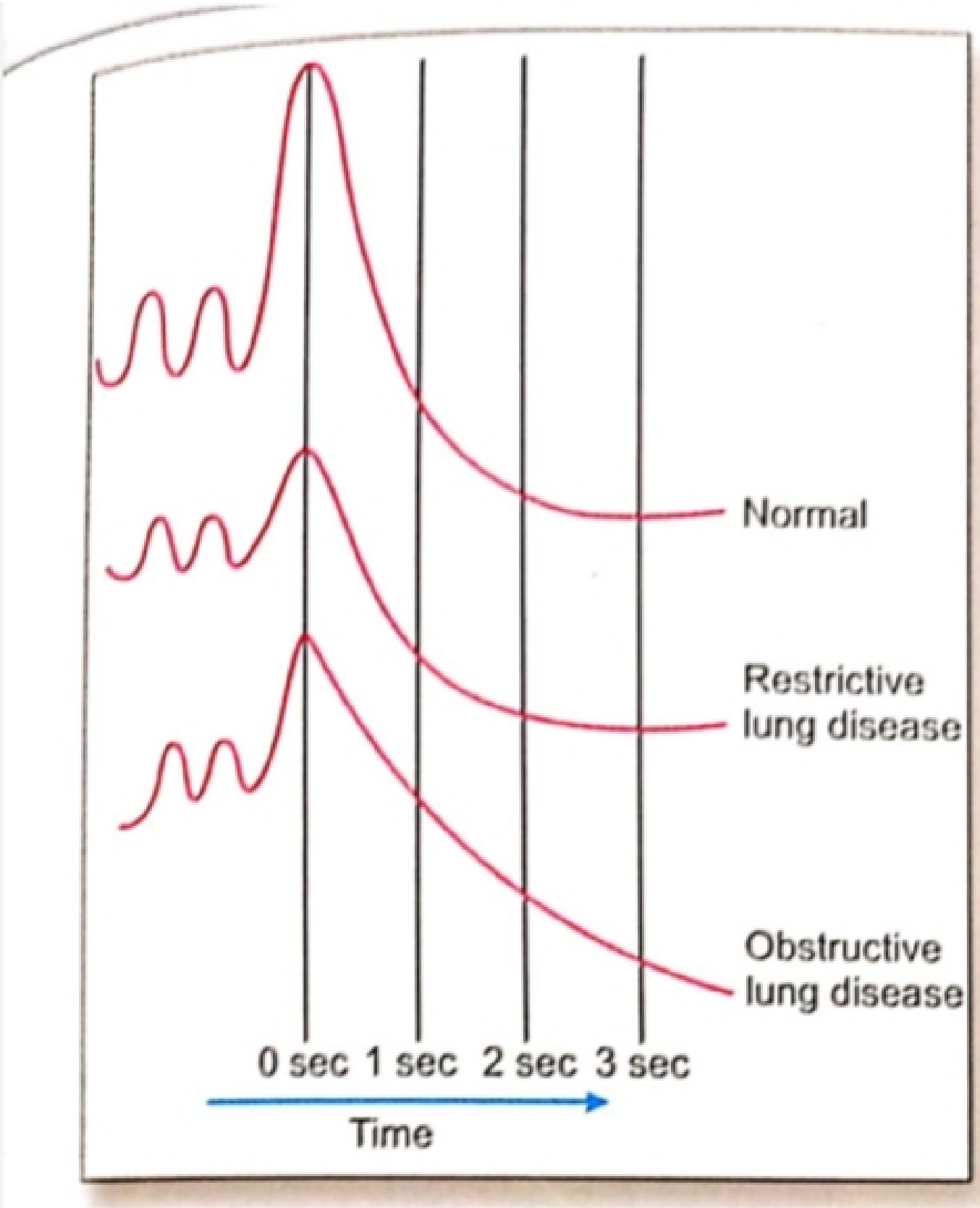


Fig. 19.3: Timed vital capacity in normal lung, restrictive lung disease and obstructive lung disease

COMPLIANCE

1. Compliance is stretchability of the lungs or chest wall. Lung compliance is defined as the change in lung volume per unit change in transpulmonary pressure. Its normal value is 0.2 L/cm of H_2O.
2. Thus, it should be noted that when the compliance is high, a lower transpulmonary pressure is required to expand the lung.

On the other hand, when the compliance is low (the lung is stiff), a greater than normal transpulmonary pressure (requiring more energy) must be generated to expand the lung during inspiration. Work of breathing will naturally be increased.

COMPLIANCE OF THE LUNGS & CHEST WALL

Compliance is developed due to the tendency for tissue to resume its original position after an applied force has been removed. After an expiration during quiet breathing (eg, at the FRC), the lungs have a tendency to collapse and the chest wall has a tendency to expand. The interaction between the recoil of the lungs and recoil of the chest can be measured through a spirometer that has a valve just beyond the mouthpiece. The mouthpiece contains a pressure-measuring device. After the person inhales a given amount, the valve is shut, closing off the airway. The respiratory muscles are then relaxed while the pressure in the airway is recorded. The procedure is repeated after inhaling or actively exhaling various volumes. The curve of airway pressure obtained in this way, plotted against volume, is the **pressure-volume curve** of the total respiratory system (P_{TR} in Figure 34–10). The pressure is zero at a lung volume that corresponds to the volume of gas in the lungs at **FRC (relaxation volume).** As can be noted from Figure 34–10, this relaxation pressure is the sum of slightly negative pressure component from the chest wall (P_w) and a slightly positive pressure from the lungs (P_L). P_{TR} is positive at greater volumes and negative at smaller volumes. **Compliance** of the lung and chest wall is measured as the slope of the P_{TR} curve, or, as a change in lung volume per unit change in airway pressure ($\Delta V/\Delta P$). It is normally measured in the pressure range where the relaxation pressure curve is steepest, and normal values are ~0.2 L/cm H_2O in a healthy adult man. However, compliance depends on lung volume and thus can vary. In an extreme example, an individual with only one lung has approximately half the ΔV for a given ΔP. Compliance is also slightly greater when measured during deflation than when measured during inflation. Consequently, it is more informative to examine

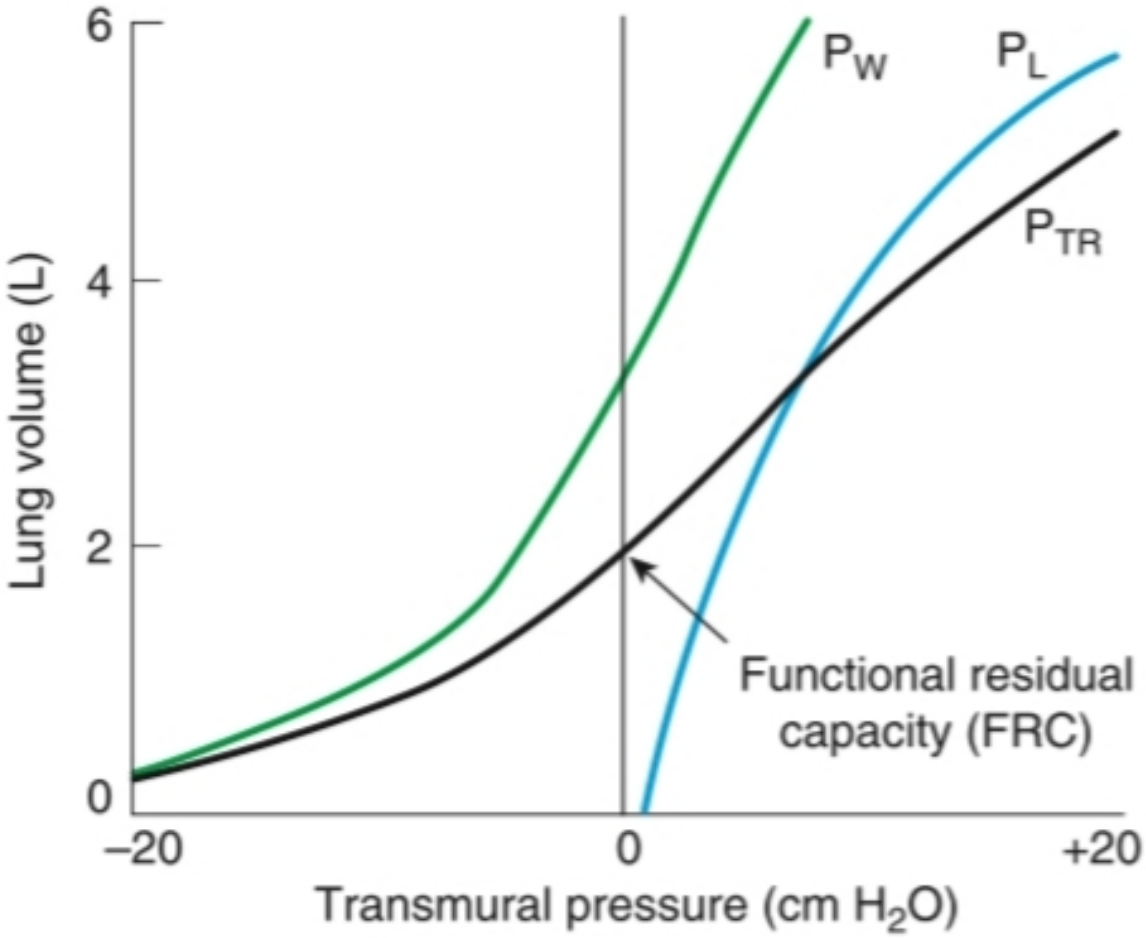


FIGURE 34–10 Pressure-volume curves in the lung. The pressure-volume curves of the total respiratory system (P_{TR}, black line), the lungs (P_L, blue line), and the chest (P_W, green line) are plotted together with standard volumes for functional residual capacity and tidal volume. The transmural pressure is intrapulmonary pressure minus intrapleural pressure in the case of the lungs, intrapleural pressure minus outside (barometric) pressure in the case of the chest wall, and intrapulmonary pressure minus barometric pressure in the case of the total respiratory system. From these curves, the total and actual elastic work associated with breathing can be derived. (Modified with permission from Mines AH: *Respiratory Physiology*, 3rd ed. New York, NY: Raven Press; 1993.)

Surfactant

Surfactant is important at birth. The fetus makes respiratory movements in utero, but the lungs remain collapsed until birth. After birth, the infant makes several strong inspiratory movements and the lungs expand. Surfactant keeps them from collapsing again. Surfactant deficiency is an important cause of **infant respiratory distress syndrome** (IRDS, also known as **hyaline membrane disease**), the serious pulmonary disease that develops in infants born before their surfactant system is functional. Surface tension in the lungs of these infants is high, and the alveoli are collapsed in many areas **(atelectasis).** An additional factor in IRDS is retention of fluid in the lungs. During fetal life, Cl^- is secreted with fluid by the pulmonary epithelial cells. At birth, there is a shift to Na^+ absorption by these cells via the epithelial Na^+ channels (ENaCs), and fluid is absorbed with the Na^+. Prolonged immaturity of the ENaCs contributes to the pulmonary abnormalities in IRDS.

Overproduction/dysregulation of surfactant proteins can also lead to respiratory distress and is the cause of pulmonary alveolar proteinosis (PAP).

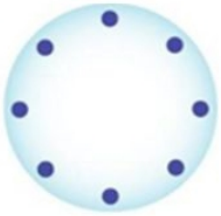
Composition of Surfactant

- **Composition** — Pulmonary surfactant is a complex mixture of lipids and proteins that lowers alveolar surface tension.
- **Lipid** — Approximately 70 percent of the lipid in surfactant is phosphatidylcholine species. Of this, approximately 60 percent is disaturated palmitoylphosphatidyl choline (DPPC)
- **Protein** — Surfactant also contains small proteins. These consist of the hydrophobic surfactant proteins SP-B and SP-C and the hydrophilic proteins SP-A and SP-D

Role of surfactant

During Inspiration

- When alveoli expand, surfactant molecules move apart.



During Expiration

- When lungs shorten, surfactant molecules move together and become concentrated → surface tension is reduced

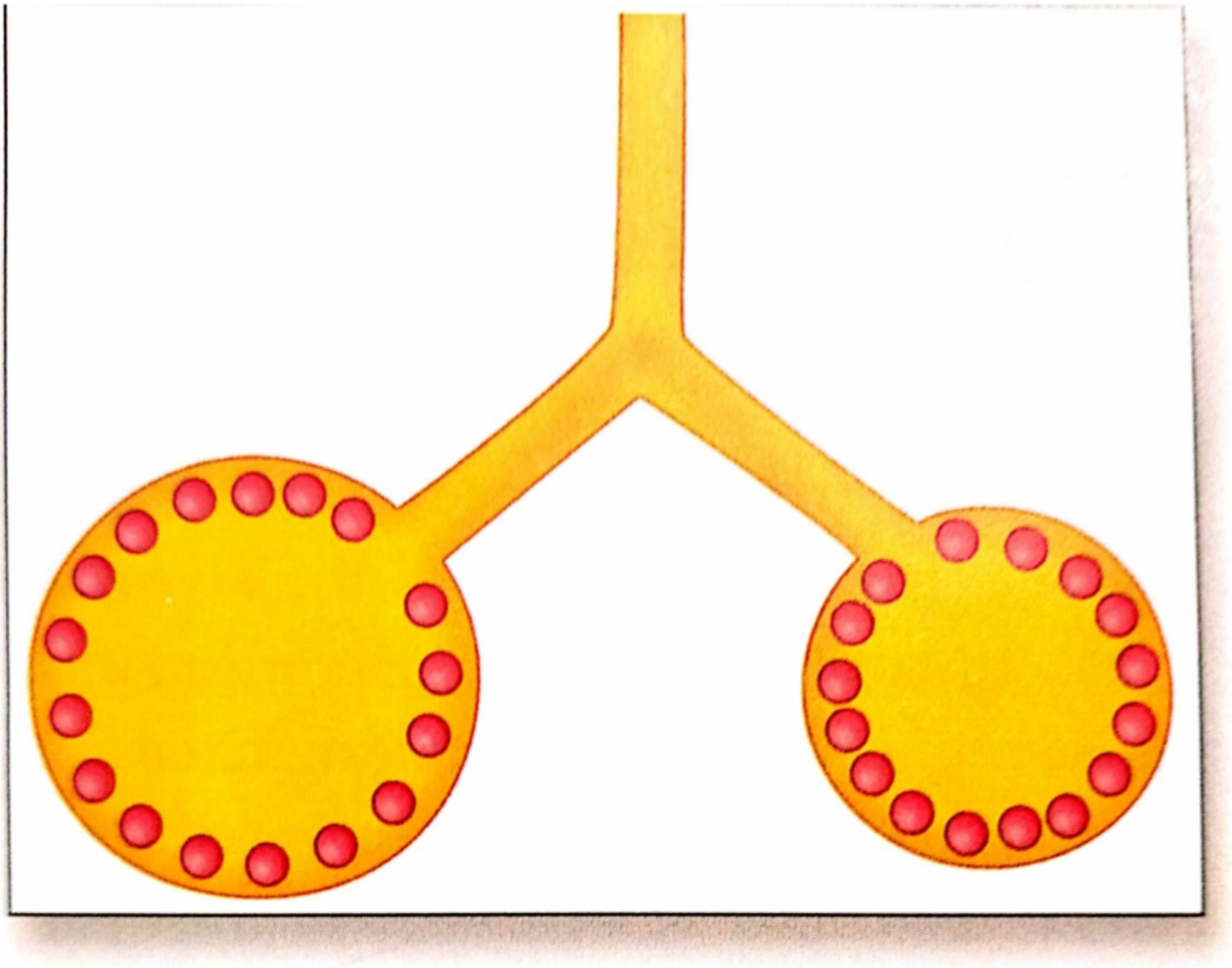


Fig. 20.5: Molecules of surfactant are spread out over a larger surface area in the large alveolus, resulting in low concentration of surfactant. In contrast, in the smaller alveolus, surfactant concentration is high

Functions of surfactant

- Decreases the surface tension.
- To promote lung expansion during inspiration.
- To prevent alveolar collapse and loss of lung volume at the end of expiration.
- Facilitates recruitment of collapsed alveoli.

normally occurs at an unconscious level, comes to consciouness. Dyspnea is felt when dyspneic index is 70% or less.

DEAD SPACE

1. The respiratory passages can be divided into:
   a. *A conducting zone* which conducts air from outside atmosphere into lungs.
   b. *An exchange zone* where gaseous exchange takes place.
   c. There is a small *transition zone* in between the two.
2. a. As, no gaseous exchange takes place in the conducting zone, it is known as dead space. (Conducting zone extends from external nares to terminal bronchioles). This dead space is called *anatomical dead space* and it differs from physiological dead space. Normally, volume of anatomical dead space is about 150 mL. Its value in mL is approximately equal to body weight in pounds.
   b. *Physiological dead space* may be defined as the total amount of wasted ventilation in the lung, i.e. the amount of air that does not take part in gaseous exchange in the whole lung. It is equal to anatomical dead space plus any additional dead space in the exchange zone. (The latter is called alveolar dead space). In a healthy individual, anatomical dead space = Physiological dead space, as there is no dead space in exchange zone in health.
   c. Any additional dead space in exchange zone, which is included in physiological dead space, may be caused by emboli (displaced thrombi from a distant site) which occlude capillaries supplying lung alveoli. As there is no blood flow to the alveoli due to occlusion of alveolar blood vessels, the ventilation is wasted and dead space is produced in the exchange zone. Physiological dead space is also increased when there is excess ventilation with respect to perfusion, causing wasted ventilation.
3. Measurement of anatomical dead space: (Fig. 19.4)
   a. Anatomical dead space can be measured by single breath O_2 inhalation followed by rapid N_2 meter analysis of nitrogen content of the expired gas.
   b. The subject takes a single inhalation of pure O_2 and a rapid N_2 meter analyses the nitrogen content of the steadily expired gas. The expiration can be divided into several phases.
   c. In phase I, the first portion of expired gas comes out from the anatomical dead space and contains pure O_2. The nitrogen concentration in this expired gas is almost nil and the N_2 concentration curve plotted against volume of expired gas runs along the baseline.
   d. In phase II, the gas coming out is a mixture of dead space gas with alveolar gas. So, N_2 concentration begins to rise.
   e. In phase III, when all dead space gas has been expired, pure alveolar air comes out and the N_2 concentration line becomes horizontal, signifying a constant N_2 concentration.
   f. Anatomical dead space is measured by adding the volume of gas expired in phase I to approximately half the volume of gas expired in phase II.

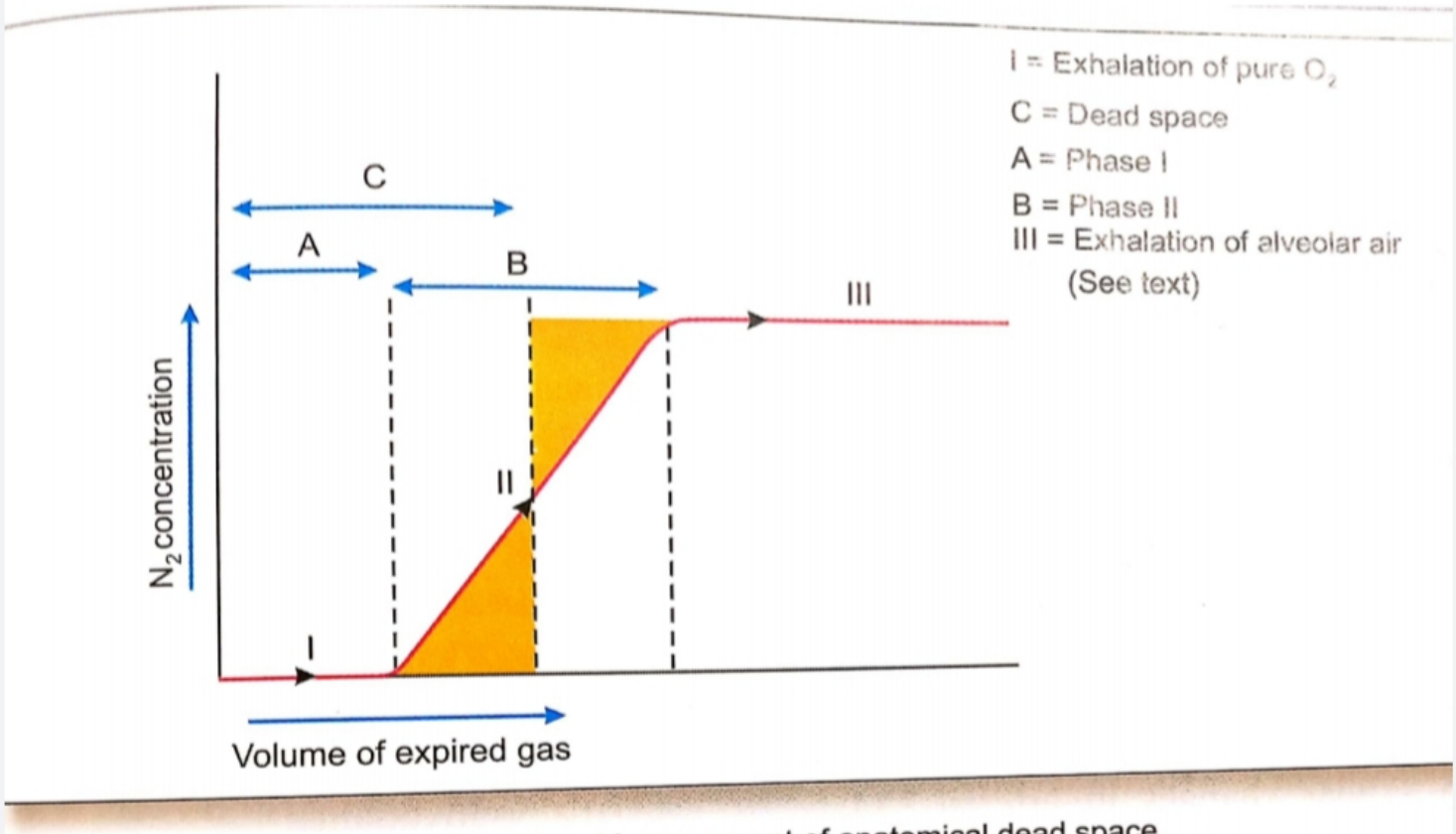


Fig. 19.4: Measurement of anatomical dead space

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