Lung Volumes and Capacities
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OBJECTIVES:

After studying this lecture, the student should understand:

1. Lung volumes and capacities and how they are measured with spirometry.
2. The difference between anatomic and physiologic dead space.
3. How to calculate minute ventilation and alveolar ventilation.
4. Forced expiratory volumes and FEV$_1$ and the changes in these that occur in obstructive and restrictive disease.

I. STRUCTURE OF THE RESPIRATORY SYSTEM

The lungs exchange O$_2$ and CO$_2$ between air and pulmonary capillary blood. The interface between air and blood, the alveolar-pulmonary capillary barrier, is very thin and has a large surface area for gas exchange. The large surface area is accomplished by wrapping pulmonary capillaries around an enormous number of alveoli (about 300 million alveoli per human lung!).

The respiratory system includes the lungs and the airways. The conducting zone (or conducting airways) brings air into and out of the lungs; the respiratory zone is lined with alveoli and is the site of gas exchange.
The conducting zone includes the nose, nasopharynx, larynx, trachea, bronchi, bronchioles, and terminal bronchioles. These structures bring air into and out of the respiratory zone, and warm, filter, and humidify the air. The trachea divides into two bronchi, one for each lung; these bronchi divide into two smaller bronchi, which divide again and again, with a total of 23 such divisions. The conducting airways are lined with mucus-secreting cells and ciliated cells that remove inhaled particles. The walls of the conducting airways contain smooth muscle with sympathetic and parasympathetic innervation.

A. Sympathetic adrenergic neurons activate $\beta_2$ receptors that dilate the airways; importantly, these $\beta_2$ receptors are also activated by circulating catecholamines (epinephrine) and by drugs that are $\beta_2$ agonists (e.g., isoproterenol). Thus, $\beta_2$ agonists are useful in treating asthma because they dilate the airways and decrease resistance to airflow.

B. Parasympathetic cholinergic neurons activate muscarinic receptors that constrict the airways and increase resistance to airflow.

The respiratory zone includes structures lined by alveoli and, therefore, participates in gas exchange. These include respiratory bronchioles, alveolar ducts, and alveolar sacs. The respiratory bronchioles have cilia and smooth
muscle, like the bronchioles of the conducting zone. The alveolar ducts are lined with alveoli, but contain no cilia and little smooth muscle. Alveolar ducts terminate in alveolar sacs that are lined with alveoli. The alveolar walls contain elastic fibers and epithelial cells called alveolar cells (or pneumocytes). Type II alveolar cells synthesize surfactant, which is required to reduce surface tension of alveoli and prevent their collapse.

II. LUNG VOLUMES AND CAPACITIES - THE SPIROMETRY DIAGRAM

Lung volumes and capacities are measured by spirometry. The subject breathes into and out of a spirometer, displacing a bell. The volume displaced is recorded on graph paper. The subject first breathes quietly (normally), then takes a maximal inspiration followed by a maximal expiration.

![Spirometry Diagram](image)

**Figure 2.**

A. Lung volumes (four)

1. **Tidal volume**, or $V_T$, is the volume inspired and expired in normal, quiet breathing. Tidal volume includes the volume of air that fills alveoli plus the volume that fills the airways. Normal tidal volume is about 500 ml.
2. **Inspiratory reserve volume** is the volume that can be inspired above tidal volume during maximal inspiration. It is approximately 3000 ml.
3. **Expiratory reserve volume** is the volume that be expired following expiration of a tidal volume during maximal expiration. It is approximately 1200 ml.

4. **Residual volume** is the volume remaining in the lungs following maximal expiration. It is approximately 1200 ml. Residual volume is not measured by spirometry, but with a helium dilution method or a body plethysmograph.

B. **Lung capacities (four).** Each lung capacity consists of two or more lung volumes.

1. **Inspiratory capacity** is the tidal volume + inspiratory reserve volume. It is the total volume inspired during maximal inspiration.

2. **Functional residual capacity**, or FRC, is the volume remaining in the lungs following a normal tidal expiration. FRC is the expiratory reserve volume + residual volume. FRC is considered the **equilibrium volume** of the lungs (volume in the lungs between normal breaths).

3. **Vital capacity**, or VC, is the inspiratory capacity + the expiratory reserve volume. Vital capacity is the total volume that can expired following maximal inspiration. Vital capacity increases with male gender, body size, and physical conditioning. In adults, it decreases with age.

4. **Total lung capacity** is the sum of all the lung volumes, i.e., it is vital capacity + residual volume.

III. **DEAD SPACE**

Dead space is the volume of the airways and the lungs that does not participate in gas exchange. The **anatomic dead space** is the volume of the conducting airways. Since conducting airways have no alveoli, they cannot possibly participate in gas exchange. The volume of the anatomic dead space is about **150 ml**. That is, in a normal tidal volume of 500 ml, 150 ml (1/3) fills the anatomic dead space and 350 ml (2/3) fills the alveoli.

The figure shows a tidal volume, 1/3 of which will fill the anatomic dead space. At the end of expiration, the conducting airways are filled with air that *had been* in the alveoli (i.e., air that already exchanged gases with pulmonary capillary blood). As you will learn, we call this alveolar gas. With inspiration of the next breath, this air is first to enter the alveoli; however, it will not undergo further gas exchange because it has already “been there, done that.” The next air to enter the alveoli is fresh air from the inspired tidal volume that will undergo gas exchange. Finally, some of the inspired tidal volume does not make it to the alveoli, but stays in the conducting airways (anatomic dead space); this air does not undergo gas exchange (i.e., is dead) and is the first air expired.
The **physiologic dead space** is comprised of the anatomic dead space plus a “functional dead space” in alveoli. Functional dead space refers to alveoli that are ventilated but not perfused with blood; because they are not perfused, they cannot participate in gas exchange, i.e., functionally “dead.” In normal persons, there is little functional dead space, and the physiologic dead space is nearly equal to the anatomic dead space. However, in lung diseases in which a so-called ventilation/perfusion defect develops, functional dead space increases and causes the physiologic dead space to increase.

Thus, the physiologic dead space includes *all* lung spaces that are ventilated but are not participating in gas exchange. In the physiologic dead space, no O\(_2\) or CO\(_2\) is exchanged. As a result, in the dead space alveolar P\(_{O_2}\) and P\(_{CO_2}\) approach their values in inspired air. The calculation of physiologic dead space by Bohr’s equation is shown in the lectures on V/Q defects.

### IV. VENTILATION RATES

Ventilation rate is the volume of air moved into and out of the lungs per unit time, expressed in ml/minute or L/minute.

A. **Minute ventilation** is the total volume of air moving into and out of the lungs per unit time and is calculated as:

\[
\text{Minute ventilation} = V_T \times \text{breaths/minute}
\]
Where VT is tidal volume.

B. **Alveolar ventilation** is the minute ventilation corrected for physiologic dead space and is calculated as:

\[
\text{Alveolar ventilation} = (\text{VT} - \text{VD}) \times \text{breaths/minute}
\]

Where VT is tidal volume and VD is physiologic dead space.

V. **FORCED EXPIRATORY VOLUMES (FEV)**

Vital capacity has already been defined as the total volume that can be expired following a maximal inspiration. Really, we should say *forcibly* expired, since that is the only way to expire all the air one possibly can. Thus, vital capacity is the same as *forced vital capacity*, or FVC. In spirometry, the subject inspires maximally and then, with forced maximal effort, expires all the air he can, as fast as possible. The total volume expired is the forced vital capacity, or vital capacity.

Vital capacity changes with age, conditioning and, importantly, disease. We are also interested in the time-course of expiration of the forced vital capacity because the time-course (i.e., how fast it is expired) is altered in many lung diseases. The volume of air that can be forcibly expired in the first second of expiration is called \( \text{FEV}_1 \), the volume that can be forcibly expired in the first two seconds of expiration is called \( \text{FEV}_2 \), and the volume that can be forcibly expired in the first three seconds of expiration is called \( \text{FEV}_3 \). (Because normal persons expire the entire vital capacity in three seconds, there is no need for an “\( \text{FEV}_4 \).”) \( \text{FEV}_1 \) is very sensitive to changes in airway resistance (a point that will be reiterated).

In several important lung diseases (asthma, chronic obstructive lung disease, and fibrosis), there are changes in both forced vital capacity (FVC) and \( \text{FEV}_1 \). To interpret the \( \text{FEV}_1 \), therefore, it is important to calculate the ratio of \( \text{FEV}_1 \) to FVC (\( \text{FEV}_1/\text{FVC} \)), i.e., the fraction of the vital capacity that can be expired in the first second of forced expiration.
A. **Normal.** In normal persons, 80% of the vital capacity is expired in the first second of forced expiration. In other words, FEV₁/FVC is 0.8, or 80%.

B. **Obstructive disease** (e.g., asthma; chronic obstructive pulmonary disease [COPD]). In obstructive lungs diseases, there is obstruction of airways, which increases the resistance to airflow. Expiration and FEV₁ are very sensitive to changes in airway resistance. In obstructive lung disease, both FVC and FEV₁ are decreased, but FEV₁ is decreased *more* than FVC such that the ratio of FEV₁/FVC is decreased.

C. **Restrictive disease** (e.g. fibrosis). In restrictive lung diseases, there is increased stiffness and elastic recoil of lung tissues and, as a result, increased elastic recoil force for expiration (discussed in next lectures). Don’t be fooled by the name “restrictive”......it does not mean increased resistance; it refers to increased stiffness (“restriction”) of the lungs. In restrictive lung disease, like in obstructive lung disease, FVC is decreased. However, because the elastic recoil of lung structures is increased, FEV₁ is decreased *less* than FVC. As a result, FEV₁/FVC is increased (or normal).

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VI. PRACTICE QUESTIONS

1. The volume remaining in the lungs after expiring a tidal volume is:
   A. Residual volume
   B. Expiratory reserve volume
   C. Expiratory reserve volume + residual volume
   D. Vital capacity - residual volume
   E. Total lung capacity - residual volume

2. Which lung volume or capacity can be inspired above FRC?
   A. Inspiratory reserve volume
   B. Inspiratory capacity
   C. Tidal volume
   D. Inspiratory residual capacity
   E. None of the above

3. FEV₁ is:
   A. The fraction of the vital capacity that can be expired in one second.
   B. The fraction of the total lung capacity that can be expired in one second.
   C. The volume that can be expired in the first second following maximal inspiration.
   D. The volume that can be expired in the first second following inspiration of a tidal volume.

4. In the physiologic dead space:
   A. Gas exchange does not occur.
   B. Gas exchange occurs only in the conducting airways.
   C. Gas exchange occurs, but is decreased.
   D. O₂ exchange occurs, but CO₂ exchange does not.
   E. CO₂ exchange occurs, but O₂ exchange does not.

EXPLANATIONS

1. *Answer = C. FRC is expiratory reserve volume + residual volume*
2. *Answer = B*
3. *Answer = C*
4. *Answer = A*