Lung Volumes and Capacities

Normally the volume of air entering the lungs during a single inspiration is approximately equal to the volume leaving on the subsequent expiration and is called the **tidal volume**. The tidal volume during normal quiet breathing is termed the resting tidal volume and is approximately 500 ml. As illustrated in Figure 15–16, the maximal amount of air that can be increased above this value during deepest inspiration is termed the **inspiratory reserve volume** (and is about 3000 ml that is, six fold greater than resting tidal volume).

After expiration of a resting tidal volume, the lungs still contain a very large volume of air. As described earlier, this is the resting position of the lungs and chest wall (that is, the position that exists when there is no contraction of the respiratory muscles); it is termed **the functional residual capacity** (and averages about 2500 ml). Thus, the 500 ml of air inspired with each resting breath adds to and mixes with the much larger volume of air already in the lungs, and then 500 ml of the total is expired. Through maximal active contraction of the expiratory muscles, it is possible to be expired much more of the air remaining after the resting tidal volume has been expired; this additional expired volume is termed the **expiratory reserve volume** (about 1500 ml). Even after a maximal active expiration, approximately 1000 ml of air still remains in the lungs and is termed the **residual volume**.

A useful clinical measurement is the **vital capacity**, the maximal volume of air that a person can expire after a maximal inspiration. Under these conditions, the person expiring the **resting tidal volume** and **inspiratory reserve volume** just inspired, plus the **expiratory reserve volume** (Figure 15–16). In other words, the vital capacity is the sum of these three volumes.

A variant on this method is the **forced expiratory volume** (FEV1), in which the person takes a maximal inspiration and then exhales maximally as fast as possible. The important value is the fraction of the total “forced” vital capacity expired in 1 s. Normal individual can expire approximately 80 percent of the vital capacity in this time.

These **pulmonary function tests** are useful diagnostic tools. For example, people with **obstructive lung diseases** (increased airway resistance) typically have a FEV1 which is less than 80 percent of the vital capacity because it is difficult for them to expired air rapidly through the narrowed airways. In contrast to obstructive lung diseases, **restrictive lung diseases** are characterized by normal airway resistance but impaired respiratory movements because of abnormalities in the lung tissue, the pleura, the chest wall, or the neuromuscular machinery. Restrictive lung diseases are characterized by a reduced vital capacity but a normal ratio of FEV1 to vital capacity.
Alveolar Ventilation

The total ventilation per minute, termed the minute ventilation, is equal to the tidal volume multiplied by the respiratory rate:

\[ \text{Minute ventilation} = \text{Tidal volume} \times \text{Respiratory rate} \quad (15-5) \]

For example, at rest, a normal person moves approximately 500 ml of air in and out of the lungs with each breath and takes 10 breaths each minute. The minute ventilation is therefore 500 ml/breath \( \times \) 10 breaths/minute = 5000 ml of air per minute. However, because of dead space, not all this air is available for exchange with the blood.

Dead Space

The conducting airways have a volume of about 150 ml. Exchanges of gases with the blood occur only in the alveoli and not in this 150 ml of the airways. Picture, then, what occurs during expiration of a tidal volume, which in this example we'll set at 450 ml instead of the 500 ml mentioned earlier. The 450 ml of air is forced out of the alveoli and through the airways. Approximately 300 ml of this alveolar air is exhaled at the nose or mouth, but approximately 150 ml still remains in the airways at the end of expiration.

During the next inspiration, 450 ml of air flows into the alveoli, but the first 150 ml entering the alveoli is not atmospheric air but the 150 ml left behind in the airways from the last breath. Thus, only 300 ml of new atmospheric air enters the alveoli during the inspiration. The end result is that 150 ml of the 450 ml of atmospheric air entering the respiratory system during each inspiration never reaches the alveoli but is merely moved in and out of the airways. Because these
airways do not permit gas exchange with the blood, the space within them is termed the **anatomic dead space**.

Thus the volume of fresh air entering the alveoli during each inspiration equals the tidal volume minus the volume of air in the anatomic dead space. For the previous example:

Tidal volume = 450 ml  
Anatomic dead space = 150 ml  
Fresh air entering alveoli in one inspiration = 450 ml - 150 ml = 300 ml  
The total volume of fresh air entering the alveoli per minute is called the **alveolar ventilation**:

\[
\text{Alveolar ventilation} = (\text{Tidal volume} - \text{Dead space}) \times \text{Respiratory rate}
\]

When evaluating the efficacy of ventilation, one should always focus on the alveolar ventilation, not the minute ventilation. This generalization is demonstrated readily by the data in Table 15–4. In this experiment, subject A breathes rapidly and shallowly, B normally, and C slowly and deeply. Each subject has exactly the same minute ventilation; that is, each is moving the same amount of air in and out of the lungs per minute. Yet, when we subtract the anatomic dead space ventilation from the minute ventilation, we find marked differences in alveolar ventilation. Subject A has no alveolar ventilation and would become unconscious in several minutes, whereas C has considerably greater alveolar ventilation than B, who is breathing normally.

Another important generalization to be drawn from this example is that increased **depth** of breathing is far more effective in elevating alveolar ventilation than is an equivalent increase in breathing **rate**. Conversely, a decrease in depth can lead to a critical reduction in alveolar ventilation. This is because a fixed volume of each tidal volume goes to the dead space. If the tidal volume decreases, the fraction of the tidal volume going to the dead space increases until, as in subject A, it may represent the entire tidal volume. On the other hand, any increase in tidal volume goes entirely toward increasing alveolar ventilation. These concepts have important physiological implications. Most situations that produce increased ventilation, such as exercise, reflex call forth a relatively greater increase in breathing depth than rate.

The anatomic dead space is not the only type of dead space. Some fresh inspired air is not used for gas exchange with the blood even though it reaches the alveoli because some alveoli, for various reasons, have little or no blood supply. This volume of air is known as **alveolar dead space**. It is quite small in
normal persons but may be very large in several kinds of lung disease. As we shall see, it is minimized by local mechanisms that match air and blood flows. The sum of the anatomic and alveolar dead spaces is known as the **physiologic dead space**.

<table>
<thead>
<tr>
<th>Table 15-4 Effect of Breathing Patterns on Alveolar Ventilation</th>
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<td>Subject</td>
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**Exchange of Gases in Alveoli and Tissues**

We have now completed our discussion of the lung mechanics that produce alveolar ventilation, but this is only the first step in the respiratory process. Oxygen must move across the alveolar membranes into the pulmonary capillaries, be transported by the blood to the tissues, leave the tissue capillaries and enter the extra cellular fluid, and finally cross plasma membranes to gain entry into cells. Carbon dioxide must follow a similar path in reverse.

In the steady state, the volume of oxygen that leaves the tissue capillaries and is consumed by the body cells per unit time is exactly equal to the volume of oxygen added to the blood in the lungs during the same time period. Similarly, in the steady state, the rate at which carbon dioxide is produced by the body cells and enters the systemic blood is identical to the rate at which carbon dioxide leaves the blood in the lungs and is expired. Note that these statements apply to the steady state; transiently, oxygen utilization in the tissues can differ from oxygen uptake in the lungs and carbon dioxide production can differ from elimination in the lungs, but within a short time these imbalances automatically produce changes in diffusion gradients in the lungs and tissues that reestablish steady-state balances.

The amounts of oxygen consumed by cells and carbon dioxide produced, however, are not necessarily identical to each other. The balance depends primarily upon which nutrients are being used for energy. The ratio of CO₂ produced/O₂ consumed is known as the **respiratory quotient (RQ)**. On a mixed diet, the RQ is approximately 0.8; that is, 8 molecules of CO₂ are produced for every 10 molecules of O₂ consumed. (The RQ is 1 for carbohydrate, 0.7 for fat, and 0.8 for protein). Typical exchange values during 1 min for a person at rest, assuming a cellular oxygen consumption of 250 ml/min, a carbon dioxide production of 200 ml/min, alveolar ventilation (supply of fresh air to the alveoli) of 4000 ml/min, and a cardiac output of 5000 ml/min.

Since only 21 percent of the atmospheric air is oxygen, the total oxygen entering the alveoli per min is 21 percent of 4000 ml, or 840 ml/min. Of this inspired
oxygen, 250 ml crosses the alveoli into the pulmonary capillaries, and the rest is subsequently exhaled. Note that blood entering the lungs already contains a large quantity of oxygen, to which the new 250 ml is added. The blood then flows from the lungs to the left heart and is pumped by the left ventricle through the tissue capillaries, where 250 ml of oxygen leaves the blood to be taken up and utilized by cells. Thus, the quantities of oxygen added to the blood in the lungs and removed in the tissues are identical.

The story reads in reverse for carbon dioxide: There is already a good deal of carbon dioxide in systemic arterial blood; to this is added an additional 200 ml, the amount produced by the cells, as blood flows through tissue capillaries. This 200 ml leaves the blood as blood flows through the lungs, and is expired. Blood pumped by the heart carries oxygen and carbon dioxide between the lungs and tissues by bulk flow, but diffusion is responsible for the net movement of these molecules between the alveoli and blood, and between the blood and the cells of the body. Understanding the mechanisms involved in these diffusion exchanges depends upon some basic chemical and physical properties of gases, which we will now discuss.

Partial Pressures of Gases

Gas molecules undergo continuous random motion. These rapidly moving molecules exert a pressure, the magnitude of which is increased by anything that increases the rate of movement. The pressure a gas exerts is proportional to (1) the temperature (because heat increases the speed at which molecules move) and (2) the concentration of the gas—that is, the number of molecules per unit volume.

As stated by Dalton's law, in a mixture of gases, the pressure exerted by each gas is independent of the pressure exerted by the others. This is because gas molecules are normally so far apart that they do not interfere with each other. Since each gas in a mixture behaves as though no other gases are present, the total pressure of the mixture is simply the sum of the individual pressures. These individual pressures, termed partial pressures, are denoted by a $P$ in front of the symbol for the gas. For example, the partial pressure of oxygen is represented by $P_{O_2}$. The partial pressure of a gas is directly proportional to its concentration. Net diffusion of a gas will occur from a region where its partial pressure is high to a region where it is low.

Atmospheric air consists primarily of nitrogen (approximately 79 percent) and oxygen (approximately 21 percent), with very small quantities of water vapor, carbon dioxide, and inert gases. The sum of the partial pressures of all these gases is termed atmospheric pressure, or barometric pressure. It varies in different parts of the world as a result of differences in altitude (it also varies with local weather conditions), but at sea level it is 760 mmHg. Since the partial pressure of any gas in a mixture is the fractional concentration of that gas times
the total pressure of all the gases, the \( PO_2 \) of atmospheric air is 0.21\( \times \) 760 mmHg = 160 mmHg at sea level.

![Diagram of respiratory system](image)

**FIGURE 15–18**
Summary of typical oxygen and carbon dioxide exchanges between atmosphere, lungs, blood, and tissues during 1 min in a resting individual. Note that the values given in this figure for oxygen and carbon dioxide in blood are not the values per liter of blood but rather the amounts transported per minute in the cardiac output (5 L in this example). The volume of oxygen in 1 L of arterial blood is 200 ml O\(_2\)/L of blood—that is, 1000 ml O\(_2\)/5 L of blood.

**Alveolar-Blood Gas Exchange**

The blood that enters the pulmonary capillaries is, of course, systemic venous blood pumped to the lungs via the pulmonary arteries. Having come from the tissues, it has a relatively high \( PCO_2 \) (46 mmHg in a normal person at rest) and a relatively low \( PO_2 \) (40 mmHg). The differences in the partial pressures of oxygen and carbon dioxide on the two sides of the alveolar-capillary membrane result in the net diffusion of oxygen from alveoli to blood and of carbon dioxide from blood to alveoli. As this diffusion occurs, the capillary blood \( PO_2 \) raises and its \( PCO_2 \) falls. The net diffusion of these gases ceases when the capillary partial pressures become equal to those in the alveoli. In a normal person, the rates at which oxygen and carbon dioxide diffuse are so rapid and the blood flow through the capillaries so slow that complete equilibrium is reached well before the end of the capillaries. Only during the most strenuous exercise, when blood flows through the lung capillaries very rapidly, is there insufficient time for complete equilibration.

Thus, the blood that leaves the pulmonary capillaries to return to the heart and be pumped into the systemic arteries has essentially the same \( PO_2 \) and \( PCO_2 \) as alveolar air. Accordingly, the factors described in the previous section—atmospheric \( PO_2 \), cellular oxygen consumption and carbon dioxide production, and alveolar ventilation—determine the alveolar gas pressures, which then
determine the systemic arterial gas pressures. Given that diffusion between alveoli and pulmonary capillaries normally achieves complete equilibration, the more capillaries participating in this process, the more total oxygen and carbon dioxide can be exchanged. Many of the pulmonary capillaries at the apex (top) of each lung are normally closed at rest.

During exercise, these capillaries open and receive blood, thereby enhancing gas exchange. The mechanism by which this occurs is a simple physical one; the pulmonary circulation at rest is at such a low blood pressure that the pressure in these apical capillaries is inadequate to keep them open, but the increased cardiac output of exercise raises pulmonary vascular pressures, which opens these capillaries. The diffusion of gases between alveoli and capillaries may be impaired in a number of ways, resulting in inadequate oxygen diffusion into the blood, particularly during exercise when the time for equilibration is reduced. For one thing, the surface area of the alveoli in contact with pulmonary capillaries may be decreased. In lung infections or pulmonary edema, for example, some of the alveoli may become filled with fluid. Diffusion may also be impaired if the alveolar walls become severely thickened with connective tissue, as, for example, in the disease (of unknown cause) called diffuse interstitial fibrosis. Pure diffusion problems of these types are restricted to oxygen and do not affect elimination of carbon dioxide, which is much more diffusible than oxygen.

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