Pulmonary Ventilation: Inspiration and Expiration

Inspiration

visualize the thoracic cavity as a gas-filled box with a single entrance at the top, the tube like trachea. The volume of this box is changeable and can be increased by enlarging all of its dimensions, thereby decreasing the gas pressure inside it. This drop in pressure causes air to rush into the box from the atmosphere, because gases always flow down their pressure gradients. The same things happen during normal quiet inspiration, when the inspiratory muscles—the diaphragm and external intercostal muscles—are activated. Here’s how quiet inspiration works:

1. Action of the diaphragm. When the dome-shaped diaphragm contracts, moves inferiorly and flattens out (Figure 22.13, top). As a result, the superior-inferior dimension (height) of the thoracic cavity increases.

2. Action of the intercostal muscles. Contraction of the external intercostal muscles lifts the rib cage and pulls the sternum superiorly (Figure 22.13, top). Because the ribs curve downward as well as forward around the chest wall, the broadest lateral and anteroposterior dimensions of the rib cage are normally directed obliquely downward. But when the ribs are raised and drawn together, they swing outward, expanding the diameter of the thorax both laterally and in the anteroposterior plane. This is much like the action that occurs when a curved bucket handle is raised.

Although these actions expand the thoracic dimensions by only a few millimeters along each plane, this is enough to increase thoracic volume by almost 500 ml—the usual volume of air that enters the lungs during a normal quiet inspiration. Of the two types of inspiratory muscles, the diaphragm is far more important in producing the volume changes that lead to normal quiet inspiration. As the thoracic dimensions increase during inspiration, the lungs are stretched and the intrapulmonary volume increases. As a result, $P_{\text{pul}}$ drops about 1 mm Hg relative to $P_{\text{atm}}$. Anytime the intrapulmonary pressure is less than the atmospheric pressure ($P_{\text{pul}} < P_{\text{atm}}$), air rushes into the lungs along the pressure gradient. Inspiration ends when $P_{\text{pul}} = P_{\text{atm}}$. During the same period, $P_{\text{ip}}$ declines to about (~ 6 mm Hg) relative to $P_{\text{atm}}$ (Figure 22.14). During the deep or forced inspirations that occur during vigorous exercise and in some chronic obstructive pulmonary diseases, the thoracic volume is further increased by activity of accessory muscles. Several muscles, including the scalenes and sternocleidomastoid muscles of the neck and the pectoralis minor of the chest, raise the ribs even more than occurs during quiet inspiration. Additionally, the back extends as the thoracic curvature is straightened by the erector spinae muscles.
FIGURE 22.14 Changes in intrapulmonary and intrapleural pressures during inspiration and expiration. Notice that normal atmospheric pressure (760 mm Hg) is given a value of 0 on the scale.
FIGURE 22.13 Changes in thoracic volume during inspiration (top) and expiration (bottom). The sequence of events in the left column includes volume changes during inspiration and expiration. The lateral views in the middle column show changes in the superior-inferior dimension (as the diaphragm alternately contracts and relaxes) and in the anterior-posterior dimension (as the external intercostal muscles alternately contract and relax). The superior views of transverse thoracic sections in the right column show lateral dimension changes resulting from alternate contraction and relaxation of the external intercostal muscles.
Expiration

Quiet expiration in healthy individuals is a passive process that depends more on lung elasticity than on muscle contraction. As the inspiratory muscles relax and resume their resting length, the rib cage descends and the lungs recoil. Thus, both the thoracic and intrapulmonary volumes decrease. This volume decrease compresses the alveoli, and $P_{pul}$ rises to about 1 mm Hg above atmospheric pressure (see Figure 22.14). When $P_{pul} > P_{atm}$, the pressure gradient forces gases to flow out of the lungs.

Forced expiration is an active process produced by contraction of abdominal wall muscles, primarily the oblique and transversus muscles. These contractions (1) increase the intra-abdominal pressure, which forces the abdominal organs superiorly against the diaphragm, and (2) depress the rib cage. The internal intercostal muscles also help to depress the rib cage and decrease thoracic volume. Control of accessory muscles of expiration is important when precise regulation of air flow from the lungs is desired. For instance, the ability of a trained vocalist to hold a musical note depends on the coordinated activity of several muscles normally used in forced expiration.

Lung Compliance

Healthy lungs are unbelievably stretchy, and this distensibility is referred to as lung compliance. Specifically, lung compliance ($CL$) is a measure of the change in lung volume ($\Delta V_L$) that occurs with a given change in the transpulmonary pressure [\( \Delta (P_{pul} – P_{pip}) \)]. This is stated as:

$$CL = \frac{\Delta V_L}{\Delta (P_{pul} – P_{pip})} \quad (15-4)$$

The more a lung expands for a given rise in transpulmonary pressure, the greater its compliance. Said another way, the higher the lung compliance, the easier it is to expand the lungs at any given transpulmonary pressure. Lung compliance is determined largely by two factors: (1) distensibility of the lung tissue, and (2) alveolar surface tension. Because lung distensibility is generally high and alveolar surface tension is kept low by surfactant, the lungs of healthy people tend to have high compliance, which favors efficient ventilation. Lung compliance is diminished by a decrease in the natural resilience of the lungs. Chronic inflammation, or infections such as tuberculosis, can cause nonelastic scar tissue to replace normal lung tissue (fibrosis).

Another factor that can decrease lung compliance is a decrease in production of surfactant. The lower the lung compliance, the more energy is needed just to breathe. Since the lungs are contained within the thoracic cavity, we also need to consider the compliance (distensibility) of the thoracic wall. Factors that decrease the compliance of the thoracic wall hinder the expansion of the lungs. The total compliance of the respiratory system is comprised of lung compliance and thoracic wall compliance.
Persons with low lung compliance due to disease therefore tend to breathe shallowly and must breathe at a higher frequency to inspire an adequate volume of air.

**Determinants of Lung Compliance**

There are two major determinants of lung compliance. One is the stretch ability of the lung tissues, particularly their elastic connective tissues. Thus a thickening of the lung tissues decreases lung compliance. However, an equally important determinant of lung compliance is not the elasticity of the lung tissues, but the surface tension at the air-water interfaces within the alveoli.

The surface of the alveolar cells is moist, and so the alveoli can be pictured as air filled sacs lined with water. At an air-water interface, the attractive forces between the water molecules, known as surface tension, make the water lining like a stretched balloon that constantly tries to shrink and resists further stretching. Thus, expansion of the lung requires energy not only to stretch the connective tissue of the lung but also to overcome the surface tension of the water layer lining the alveoli.

Indeed, the surface tension of pure water is as great as the alveoli lined with pure water; lung expansion would require exhausting muscular effort and the lungs would tend to collapse. It is extremely important, therefore, that the **type II** alveolar cells secrete a detergent-like substance known as pulmonary surfactant, which markedly reduces the cohesive forces between water molecules on the alveolar surface. Therefore, *surfactant lowers the surface tension, which increases lung compliance and makes it easier to expand the lungs* (Table 15–3).
Surfactant

Is a complex of both lipids and proteins, but its major component is a phospholipids that forms a monomolecular layer between the air and water at the alveolar surface. The amount of surfactant tends to decrease when breaths are small and constant. A deep breath, which people attempts frequently in their breathing pattern, acts to stretch type II cells, which stimulates the secretion of surfactant. This is why patients who had thoracic or abdominal surgery their breathing is shallow because of the pain must be urged to take occasional deep breaths.

A striking example of what occurs when surfactant is deficient a disease known as respiratory distress syndrome of the newborn. This is the second leading cause of death in premature infants, in whom the surfactant-synthesizing cells may be too immature to function adequately. Because of low lung compliance, the infant is able to inspire only by the most strenuous efforts, which may ultimately cause complete exhaustion, inability to breathe, lung collapse,
and death. Therapy in such cases is assisted breathing with a mechanical ventilator and the administration of natural or synthetic surfactant via the infant's trachea.

### TABLE 15–3 Some Important Facts about Pulmonary Surfactant

1. Pulmonary surfactant is a mixture of phospholipids and protein.
2. It is secreted by type II alveolar cells.
3. It lowers surface tension of the water layer at the alveolar surface, which increases lung compliance (that is, makes the lungs easier to expand).
4. A deep breath increases its secretion (by stretching the type II cells). Its concentration decreases when breaths are small.
**FIGURE 15–13**
Sequence of events during expiration. Figure 15–12 illustrates these events quantitatively.
Airway Resistance

As previously stated, the volume of air that flows into or out of the alveoli per unit time is directly proportional to the pressure difference between the atmosphere and alveoli and inversely proportional to the resistance to flow offered by the airways (Equation 15-2). The factors that determine airway resistance are analogous to those determining vascular resistance in the circulatory system: tube length, tube radius, and interactions between moving molecules (gas molecules, in this case). As in the circulatory system, the most important factor by far is the tube radius: Airway resistance is inversely proportional to the fourth power of the airway radii.

Airway resistance to air flow is normally so small that very small pressure differences suffice to produce large volumes of air flow. As we have seen (Figure 15–12), the average atmosphere-to-alveoli pressure difference during a normal breath at rest is less than 1 mmHg; yet, approximately 500 ml of air is moved by this tiny difference.

Airway radii and therefore resistance are affected by physical, neural, and chemical factors. One important physical factor is the transpulmonary pressure, which exerts a distending force on the airways, just as on the alveoli. This is a major factor keeping the smaller airways those without cartilage to support them from collapsing. Because, as we have seen, transpulmonary pressure increases during inspiration, airway radius becomes larger and airway resistance smaller as the lungs expand during inspiration. The opposite occurs during expiration.
A second physical factor holding the airways open is the elastic connective-tissue fibers that link the outside of the airways to the surrounding alveolar tissue. These fibers are pulled upon as the lungs expand during inspiration, and in turn they help pull the airways open even more than between breaths. This is termed lateral traction. Thus, putting this information and that of the previous paragraph together, both the transpulmonary pressure and lateral traction act in the same direction, reducing airway resistance during inspiration.

Such physical factors also explain why the airways become narrower and airway resistance increases during a forced expiration. Indeed, because of increased airway resistance, there is a limit as to how much one can increase the airflow rate during a forced expiration no matter how intense the effort. In addition to these physical factors, a variety of sympathetic and Parasympathetic factors can influence airway smooth muscle and thereby airway resistance. For example, the hormone epinephrine relaxes airway smooth muscle (via an effect on beta-adrenergic receptors). One might wonder why physiologists are concerned with all the many physical and chemical factors that can influence airway resistance when we earlier stated that airway resistance is normally so low that it is no impediment to air flow. The reason is that, under abnormal circumstances, changes in these factors may cause serious increases in airway resistance. Asthma and chronic obstructive pulmonary disease provide important examples.

**Asthma**

Disease characterized by intermittent attacks in which airway smooth muscle contracts strongly, markedly increasing airway resistance. The basic defect in asthma is chronic inflammation of the airways, the causes of which vary from person to person and include, among others, allergy and virus infections. The important point is that the underlying inflammation causes the airway smooth muscle to be hyper responsive and to contract strongly when, depending upon the individual, the person exercises (especially in cold, dry air) or is exposed to cigarette smoke, environmental pollutants, viruses, allergens, normally released bronchoconstrictor chemicals, and a variety of other potential triggers. The therapy for asthma is twofold: (1) to reduce the chronic inflammation and hence the airway hyper responsiveness with so-called anti-inflammatory drugs, particularly glucocorticoids taken by inhalation; and (2) to overcome acute excessive airway smooth-muscle contraction with bronchodilator drugs that is, drugs that relax the airways. The latter drugs work on the airways either by enhancing the actions of bronchodilator neuroendocrine or by blocking the actions of bronchoconstrictor. For example, one class of bronchodilator drug mimics the normal action of epinephrine on beta-adrenergic receptors.

**Chronic Obstructive Pulmonary Disease**

The term chronic obstructive pulmonary disease refers to (1) emphysema, (2) chronic bronchitis, or (3) a combination of the two. These diseases, which cause severe difficulties not only in ventilation but in oxygenation of the blood, are
among the major causes of disability and death in the United States. In contrast to asthma, increased smooth-muscle contraction is not the cause of the airway obstruction in these diseases. Emphysema is discussed later in this chapter; suffice it to say here that the cause of obstruction in this disease is destruction and collapse of the smaller airways.

**Chronic bronchitis**

Characterized by excessive mucus production in the bronchi and chronic inflammatory changes in the small airways. The cause of obstruction is an accumulation of mucus in the airways and thickening of the inflamed airways. The same agents that cause emphysema smoking, for example also cause chronic bronchitis, which is why the two diseases frequently coexist.

**References**

2. Human Physiology the Basis of Medicine 2nd, Edition. By Gillian Pocock and Christopher D.R.