Relation of the Lungs to the Thoracic (Chest) Wall

The lungs are situated in the thorax, which is a closed compartment and completely separated from the abdomen by the diaphragm a large, dome-shaped sheet of skeletal muscle. The wall of the thorax is formed by the spinal column, the ribs, the breastbone (sternum), and several groups of muscles that run between the ribs (collectively termed the intercostal muscles). The thoracic wall also contains large amounts of elastic connective tissue.

Each lung is surrounded by a completely closed sac, known as pleural sac, consisting of a thin sheet of cells called pleura. The two pleural sacs, one on each side of the midline, are completely separate from each other. The relationship between a lung and its pleural sac can be visualized by imagining what happens when you push a fist into a balloon. The arm represents the major bronchus leading to the lung, the fist is the lung, and the balloon is the pleural sac.

The fist becomes coated by one surface of the balloon. In addition, the balloon is pushed back upon itself so that it’s opposite surfaces lie close together. Unlike the hand and balloon, however, the pleural surface coating the lung (visceral pleura) is firmly attached to the lung by connective tissue. Similarly, the outer layer (parietal pleura) lines the interior thoracic wall and diaphragm. The two layers of pleura in each sac are so close to each other; they are separated by an extremely thin layer of intrapleural fluid. The intrapleural fluid volume is only a few milliliters totally surrounds the lungs and lubricates the pleural surfaces so that they can slide over each other during breathing. The changes in the hydrostatic pressure of the intrapleural fluid cause the lungs and thoracic wall to move in and out together during normal breathing. The intrapleural pressure (Pip) (also termed intrathoracic pressure).
Intrapulmonary and intrapleural pressure relationships. Differences in pressure relative to atmospheric pressure (760 mm Hg) are given in parentheses. Values shown are at the end of a normal expiration. For illustration, the size of the pleural cavity has been greatly exaggerated.

Ventilation and Lung Mechanics

It is helpful to preview some steps involved in respiration before beginning the detailed descriptions of each step (Figure 15–6) beginning with ventilation. Ventilation is defined as the exchange of air between the atmosphere and alveoli. Air moves by bulk flow, from a region of high pressure to one of low pressure. The bulk flow can be described by the equation:

\[ F = \frac{\Delta P}{R} \]

(15-1).

Flow, \( F \) is proportional to the pressure difference \( \Delta P \) between two points and inversely proportional to the resistance \( R \). For air flow into or out of the lungs, the relevant pressures are (alveolar pressure \( P_{alv} \)) and (atmospheric pressure, \( P_{atm} \)), or the gas pressure at the nose and mouth, normally the pressure of the air surrounding the body:

\[ F = \frac{(P_{atm} - P_{alv})}{R} \]

(15-2).

Avery important point must be made at this point: All pressures in the respiratory system, as in the cardiovascular system, are given relative to atmospheric.
pressure. This is 760 mmHg at sea level. For example, the alveolar pressure between breaths is said to be 0 mmHg, which means that it is the same as atmospheric pressure.

During ventilation, air moves into and out of the lungs because the alveolar pressure is alternately made less than and greater than atmospheric pressure (Figure 15–7). These alveolar pressure changes are caused, as we shall see, by changes in the dimensions of the lungs.

(Figure 15-6)
1- Ventilation: Exchange of air between atmosphere and alveoli by bulk flow
2- Exchange of O₂ and CO₂ between alveolar air and blood in lung capillaries by diffusion
3- Transport of O₂ and CO₂ through pulmonary and systemic circulation by bulk flow
4- Exchange of O₂ and CO₂ between blood in tissue capillaries and cells in tissues by diffusion
5- Cellular utilization of O₂ and production of CO₂

Pulmonary ventilation is a mechanical process that depends on volume changes in the thoracic cavity. A rule to keep in mind throughout the following discussion is that volume changes lead to pressure changes, and pressure changes lead to the flow of gases to equalize the pressure. The relationship between the pressure and volume of a gas is given by Boyle’s law: At constant temperature, the pressure of a gas varies inversely with its volume

That is, \[ P_1V_1 = P_2V_2 \] (Figure 15–8).
Where $P$ is the pressure of the gas, $V$ is its volume, and subscripts 1 and 2 represent the initial and resulting conditions respectively. Gases always fill their container. Therefore, in a large container, the molecules in a given amount of gas will be far apart and the pressure will be low. But if the volume of the container is reduced, the gas molecules will be forced closer together and the pressure will rise. A good example is an inflated automobile tire. The tire is hard and strong enough to bear the weight of the car because air is compressed to about one-third of its atmospheric volume in the tire, providing the high pressure. Now let us see how this relates to inspiration and expiration.

It is essential to recognize the correct causal sequences in ventilation: During inspiration and expiration the volume of the “container”—the lungs—is made to change, and these changes then cause, by Boyle’s law, the alveolar pressure changes that drive air flow into or out of the lungs. Our descriptions of ventilation must focus, therefore, on how the changes in lung dimensions are brought about. The lungs are passive elastic structures—like balloons—and their volume, therefore, depends upon:

1. The difference in pressure between the inside the (Lung alveoli $P_{alv}$) and the outside of the lungs (intra pleural $P_{ip}$) termed the transpulmonary pressure.

2. How stretch ability of the lungs is? Thus,

   Transpulmonary pressure = $P_{alv} - P_{ip}$ .......................... (15-3)

Compare this equation to Equation 15-2 (the equation that describes air flow into or out of the lungs), as it will be essential to distinguish these equations from each other (Figure 15–9).

The chest wall muscles used in respiration are not attached to the lung surface. When they contract or relax, they are directly changing the dimensions of the chest, which in turn change the transpulmonary pressure ($P_{alv}-P_{ip}$). The change in transpulmonary pressure then causes a change in lung size, which causes changes in alveolar pressure and, thereby, the difference in pressure between the atmosphere and the alveoli ($P_{atm} - P_{alv}$). This difference in pressure that causes air flow into or out of the lungs. Now apply these concepts to the phases of the respiratory cycle: the period between breaths, inspiration, and expiration.
The Stable Balance between Breaths

Figure 15–10 illustrates the situation that normally exists at the end of an unforced expiration—that is, between breaths when the respiratory muscles are relaxed and no air is flowing. The alveolar pressure ($P_{alv}$) is 0 mmHg; means the same as atmospheric pressure. The intrapleural pressure ($P_{ip}$) is approximately (4 mmHg) less than atmospheric pressure that is (- 4 mmHg) using the standard convention of giving all pressures relative to atmospheric pressure. Therefore, the transpulmonary pressure ($P_{alv} - P_{ip}$) equals [0 mmHg - (- 4 mmHg)] = 4 mmHg. As emphasized in the previous section, this transpulmonary pressure is the force acting to expand the lungs; it is opposed by the elastic recoil of the partially expanded and, therefore, partially stretched lungs. Elastic recoil is defined as the tendency of an elastic structure to oppose stretching or distortion. In other words, inherent elastic recoil tending to collapse the lungs is exactly balanced by the transpulmonary pressure tending to expand them, and the volume of the lungs is stable at this point. As we shall see, a considerable volume of air is present in the lungs between breaths.
At the same time, there is also a pressure difference of 4 mmHg pushing inward on the chest wall that is tending to compress the chest, for the following reason. The pressure difference across the chest wall is the difference between atmospheric pressure and intrapleural pressure \((P_{atm} - P_{ip})\). \(P_{atm}\) is 0 mmHg, and \(P_{ip}\) is (- 4 mmHg); accordingly, \(P_{atm} - P_{ip}\) is \([0 \text{ mmHg} - (-4 \text{ mmHg})]\) = 4 mmHg directed inward. This pressure difference across the chest wall just balances the tendency of the partially compressed elastic chest wall to move outward, and so the chest wall, like the lungs, is stable in the absence of any respiratory muscular contraction.

Clearly, the sub atmospheric intrapleural pressure is the essential factor keeping the lungs partially expanded and the chest wall partially compressed between breaths. The important question now is: What has caused the intrapleural pressure to be sub atmospheric? As the lungs (tending to move inward from their stretched position because of their elastic recoil) and the thoracic wall (tending to move outward from its compressed position because of its elastic recoil) “try” to move ever so slightly away from each other, there occurs an infinitesimal enlargement of the fluid-filled intrapleural space between them. But fluid cannot expand the way air can, and so even this tiny enlargement of the intrapleural space—so small that the pleural surfaces still remain in contact with each other—drops the intrapleural pressure below atmospheric pressure. In this way, the elastic recoil of both the lungs and chest wall creates the sub atmospheric intrapleural pressure that keeps them from moving apart more than a very tiny amount.

The importance of the transpulmonary pressure is for achieving a stable balance can be seen. During surgery or trauma, the chest wall is pierced without damaging the lung. Atmospheric air rushes through the wound into the intrapleural space (pneumothorax), and the intrapleural pressure increase from - 4 mmHg to 0 mmHg. Thus, total elimination of the transpulmonary pressure, lungs collapses no more open. At the same time, the chest wall moves outward since its elastic recoil is also no longer opposed.
FIGURE 15–10
Alveolar ($P_{alv}$), intrapleural ($P_{ip}$), and transpulmonary ($P_{alv} - P_{ip}$) pressures at the end of an unforced expiration—that is, between breaths. The transpulmonary pressure exactly opposes the elastic recoil of the lung, and the lung volume remains stable. (The volume of intrapleural fluid is greatly exaggerated for purposes of illustration.)

References

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