CHAPTER 3

Lung Mechanics: Putting the Blueprints of Gas Exchange into Action

As we've stated multiple times now, diffusion is the driver behind all movement of gases in the body. *Ultimately, what keeps oxygen moving into the cells and carbon dioxide out of the cells is diffusion!* And, all of this diffusion takes place collectively through the hundreds of millions of alveolar–capillary units. Remember that in the end, O_2 delivery and CO_2 removal is only as fast as its diffusion gradient allows! Diffusion, as you recall from the diffusion equation (which, by the way, is tested on STEP 1 USMLE Board Exams), is improved by (1) increasing concentrations of particles on one side of the membrane, that is, increasing the ΔP; (2) increasing membrane surface area; and (3) decreasing distance between compartments. Intrinsic to the blueprints themselves, to the anatomy, are (2) and (3). The distance and surface area are intrinsic to the system. Delta P, however, is what happens once the movement—the mechanics of the lungs—gets turned on. All of these variables play a role in the continuous exchange of O_2 and CO_2, but the ΔP is the driving force behind gas exchange. If there is no pressure difference, it does not matter how small the distance or how big the surface area available. This is why the renewal of alveolar air with fresh atmospheric air is critical! Keeping the alveolar partial pressure of O_2 (P_AO_2) higher, and the alveolar pressure of CO_2 (P_ACO_2) lower than the partial pressures inside the body keeps the ΔP stable. By keeping ΔP stable, you keep oxygen flowing in and CO_2 flowing out.

Key

Alveolar air must be continually renewed with atmospheric air in order to allow for diffusion of both O_2 and CO_2.

Given that the ΔP is so important, let's take a closer look at the actual numbers involved in the movement of O_2 and CO_2. Remember from Chapter 1 that atmospheric pressure at sea level is 760 mmHg.

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This number is a result of the added partial pressures of all the gases that make up the atmosphere. Thus, atmospheric air is made up of approximately 78% nitrogen (N_2), 21% oxygen (O_2), and trace amounts of other gases including carbon dioxide (CO_2).

Take a look at Table 3.1 and you'll be able to see how these percentages translate into actual pressures. On an average day, in the atmospheric air, the pressure of O_2 is approximately 160 mmHg. As you breathe the air in, however, there's a sharp decrease in the pressure of O_2 to 150 mmHg. This is because normal body temperature of 37°C generates water vapor. This water vapor has a partial pressure of 47 mmHg and will dilute the rest of the gases in that fraction of air, therefore PO_2 will decrease to 150 mmHg and PN_2 will decrease to 563 mmHg. As air finally reaches the alveoli, there's an even bigger drop in alveolar PO_2 (P_AO_2) to 104 mmHg because oxygen is being consumed. The PCO_2 in the alveoli (P_ACO_2) is approximately 40 mmHg, and as we will see later, this is closely regulated. An alveolar PO_2 of 104 mmHg and P_ACO_2 of 40 allows for an adequate exchange of O_2 and CO_2 with blood, and ensures oxygen continues to move in and carbon dioxide out. Remember that these gases continue to be used in every cell of the body as the cells are constantly consuming oxygen and producing carbon dioxide. Thus if the alveoli are not constantly receiving fresh atmospheric air, this gradient, this ΔP, will be wiped out! Eventually, most of the O_2 would be sucked out of the alveoli, consumed by the cells and replaced by CO_2. Therefore, renewal with fresh air is essential to sustaining life.

In this chapter and the next, we will see what happens once the blueprints are put into action, the mechanics of ventilation. This chapter will be focused on understanding the methods employed to generate

Table 3.1 Approximate Standard Partial Pressures of Gases at Sea Level on an Average Day

Partial Pressures of Gases in mmHg				
	Atmospheric Air	**Moist Tracheal Air**	**Alveolar Air**	**Expired Air**
PO_2	160	150	104	120
PCO_2	0	0	40	27
PH_2O	0	47	47	47
PN_2	600	563	569	566
Ptotal	760	760	760	760

air movement within the lungs and the physics behind it. The next chapter will focus on what this looks like when integrated more fully with the human anatomy—the respiratory cycle. So let's get started!

IN AND OUT: HOW GAS MOVES

The goal of the lungs is to "mix" the gases coming from the true atmosphere and the gases coming from the cells in the body. This aids in maintaining the diffusion gradients for O_2 and CO_2. But, believe it or not, the lungs are relatively passive bystanders in all of this. How so? Well, the lungs themselves cannot actively contract or expand. Some of the conduction airways have smooth muscle in them, but as a whole the lungs don't. If the lungs cannot actively contract or expand themselves to move air in and out, it means that it is mainly external forces—that is, movement of the diaphragm, the chest wall, surface tension, and so on—that are acting upon the lungs to either expand them or contract them. This is an extremely important concept that is bypassed regularly by many students as obvious; however, the implications are far reaching in both health and disease. In order to understand this a little better, let's first study the relevant anatomy from a functional and mechanical point of view.

Key

The expansion and contraction of the lungs is passive and it depends on external factors such as the diaphragm, chest wall motion, surface tension, and lung tissue elastic recoil.

Functional and Mechanical Anatomy

A simple analogy for lung function would be that the lungs are one big balloon (actually many hundreds of millions of interconnected little balloons, but the same logic can be applied). So, how does inflating a balloon work? Well, let's take a look. Figure 3.1 shows us the basics of the pressure differentials required to inflate a balloon. The principle to inflating the balloon is:

> *Establish a pressure gradient so that there is a pressure force driving air from the mouth of the balloon (high pressure) to the inside of the balloon (low pressure). This will begin to increase the pressure within the balloon until the gradient is lost and the pressure on both sides is equal.*

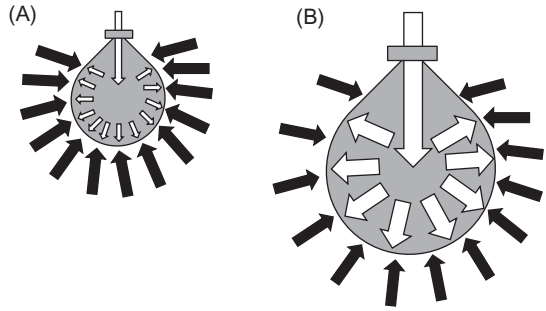

Figure 3.1 When the inflation pressure (white arrows) is less than the sum of the elastic resistance of the balloon and the pressure exerted by the atmosphere (black arrows) the balloon will not inflate (A). However, when the inflation pressure overcomes both elastic resistance and atmospheric pressure, the balloon inflates (B).

In addition to establishing a pressure gradient from the mouth of the balloon to the inside of the balloon, two things need to be overcome first in order to drive air into the balloon:

- The elastic resistance of the balloon itself
- The pressure exerted on the balloon by the atmosphere

In Figure 3.1A, the black arrows represent the pressure working against the balloon being inflated (in this case elasticity and outside atmospheric pressure). The white arrows represent the pressure inside that is trying to inflate the balloon. Remember, pressure is just force over a given area, so what we're looking at is a balance of forces. It is the imbalance between these forces that will determine whether or not the balloon inflates. This means that when the inflation pressure is greater than both the elastic resistance and the atmospheric pressure, the balloon will inflate (see Figure 3.1B). If the inflation pressure is not large enough to overcome these opposing forces, then the balloon won't inflate. Simple enough, right?

The Importance of the $\triangle$P: It's Not Just at the Alveoli, but Getting to the Alveoli as Well

What will ultimately move air in and out of the lungs is a pressure difference between the lungs and the atmosphere. So, let's analyze this phenomenon with a two-compartment model like that shown in Figure 3.2A. In it, we can see two compartments communicating via a channel between them. These compartments are at sea level, so the pressure of air inside them is 760 mmHg. Now, the channel that communicates both compartments is open, thereby the pressure of gas in both compartments equalizes

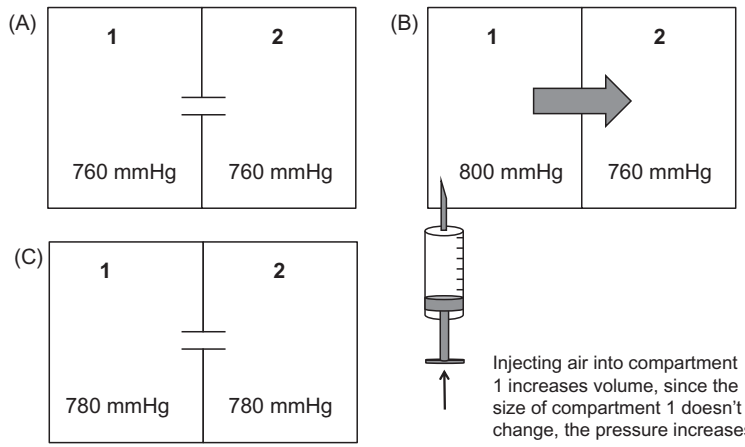

Figure 3.2 The movement of gas from one compartment to another requires the presence of a pressure gradient. At a steady state, where the pressure is the same in both compartments (A), no air will move. By increasing the pressure in compartment 1 (B) a gradient of +40 mmHg is generated from compartment 1 to 2, which favors diffusion of gas until the pressure is again equalized between compartments (C).

and is therefore the same; that is, there is *no* pressure gradient. Put as a formula, in order to calculate the ΔP between compartments:

$$\Delta P = P1 - P2$$

in this case:

$$P1 = 760 \text{ mmHg}$$
$$P2 = 760 \text{ mmHg}$$

therefore:

$$\Delta P = 760 \text{ mmHg} - 760 \text{ mmHg}$$

So:

$$\Delta P = 0 \text{ mmHg}$$

Key

Δ (*delta*) = difference, so ΔP means the difference in pressure between two compartments and ΔVol refers to the difference in volume between two compartments.

A ΔP of 0 mmHg means that there is no pressure difference between compartments, so there is no net gradient for movement of

gas from one compartment to the other. But what would happen if we injected air into compartment 1 without changing the size of the container? The pressure in compartment 1 would rise from 760 mmHg to 800 mmHg (Figure 3.2B) and the pressures between compartments would no longer be equal. If we use our ΔP formula again:

$$\Delta P = P1 - P2$$

in this case:

$$P1 = 800 \text{ mmHg}$$
$$P2 = 760 \text{ mmHg}$$

therefore:

$$\Delta P = 800 \text{ mmHg} - 760 \text{ mmHg}$$

So:

$$\Delta P = +40 \text{ mmHg}$$

The ΔP is now positive! Which means that air will flow from compartment 1 into compartment 2 until the pressures are equalized. In this case, let's say that the pressures equalize at 780 mmHg (Figure 3.2C). If we now plug these numbers into our formula, you'll see that the ΔP is now 0 again. This is because even though total pressure of our system increased (780 mmHg vs 760 mmHg), there is no net pressure difference between compartments.

Now, back to the balloon. This time, we placed the balloon inside of a bottle (Figure 3.3A). Note that the mouth of the balloon is open and attached to the opening of the bottle. So, similar to what we saw in Figure 3.2, we have two compartments in our balloon-in-a-bottle model:

- The compartment outside of the balloon (the atmosphere), Compartment 1
- The compartment inside the balloon, Compartment 2

It's important to note that the inside of the bottle is *not* communicating with either the outside atmosphere or the balloon (greyed out area in Figure 3.3). It is sealed off. In Figure 3.2 we had two freely communicating compartments, so the pressures between them always equalized. In Figure 3.3, the atmosphere and the balloon are freely

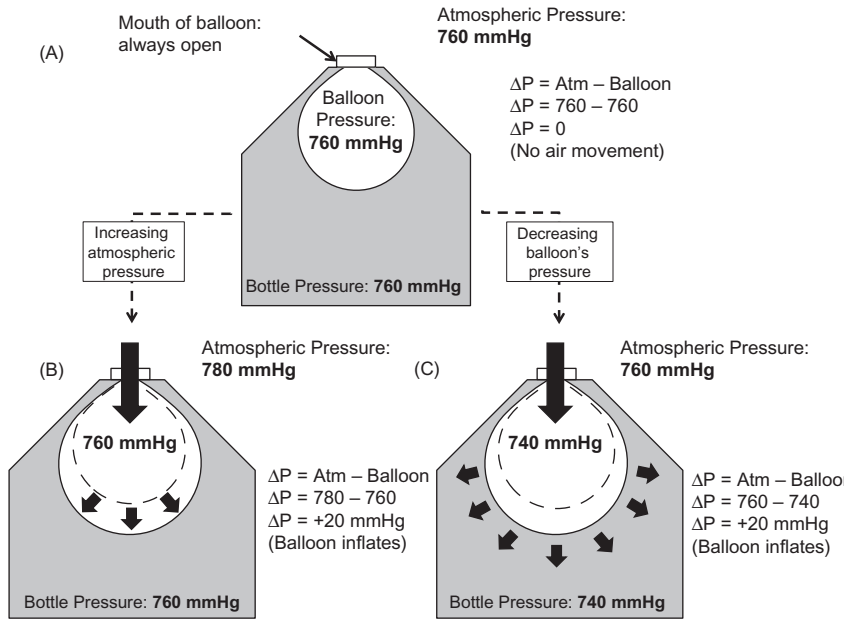

Figure 3.3 A simplified model of lung function. At equilibrium (A), there is no net movement of air. The pressure inside the balloon (white) will always attempt to reach equilibrium with the atmospheric pressure. Increases in atmospheric pressure (B) or decreases in bottle pressure (C) will both generate a gradient that favors balloon inflation.

communicating because the mouth of the balloon is open. So, if the atmospheric pressure in Figure 3.3A is 760 mmHg, what do you think the pressure will be inside the balloon? Exactly the same: 760 mmHg! (Remember, the pressures in compartments that are freely communicated will tend to equalize.) This means that the ΔP between the atmosphere (760 mmHg) and the balloon (760 mmHg) is 0. Therefore, there will be no net movement of air in or out of the balloon, because there is no gradient between the atmosphere and the inside of the balloon.

Our ultimate goal is to inflate the balloon. In order to do that the pressure inside the balloon needs to be less than the pressure outside. Therefore if we plug this into our formula for ΔP, then:

$$\Delta P = \text{Atmospheric Pressure} - \text{Balloon Pressure}$$

If we want the ΔP to be positive (and have the balloon inflate) we can do one of two things:

- We can increase the atmospheric pressure.
- We can decrease the pressure inside the balloon.

If we increased the atmospheric pressure the ΔP would be positive from the outside to the inside. In Figure 3.3B we increase the atmospheric pressure to 780 mmHg. This generates a ΔP of +20 mmHg, favoring air going into the balloon. However increasing the atmospheric pressure is relatively hard to do. Assuming you're not hiking up a mountain with the balloon or diving into the depths of the ocean, the atmospheric pressure is constant. Unlike in Figure 3.2, where the pressures average out between the two compartments, the atmospheric pressure will always remain at 760 mmHg at sea level. This is because the earth atmosphere is so large, you can almost think of it as an infinitely large compartment. So, realistically, if we wanted to do this, we would have to seal off the mouth of the balloon and attach some sort of pump to it to make this work. In other words, we'd have to create a new atmospheric pressure.

Clinical Correlate

Positive Pressure Ventilation

Movement of air in and out of the lungs is dependent on changes in pressure that favor a decreased pressure inside of the lungs relative to the atmosphere to bring air in, and an increased pressure inside the lungs relative to the atmosphere to let air out. However when a patient can't breathe on his or her own, we use something called Positive Pressure Ventilation (PPV). PPV does just what its name describes. Generally this is done through an endotracheal tube (tube that is placed in the trachea) that is connected to a ventilator, which moves air in and out of the lungs. To get air into the lungs it generates a positive pressure in the ventilator (so the pressure inside the lungs is less), which generates a gradient from the outside in. To get air out of the lungs, the reverse happens. The pressure in the ventilator is decreased allowing air to flow out of the lungs.

But we want to try and get this balloon to inflate without some sort of external pump. So what other option do we have? Well, we can try to decrease the pressure inside the balloon relative to the atmosphere. Decreasing the pressure inside the balloon would still create a ΔP. But how would you do this? This is where the bottle that we've so casually ignored up to this point comes in.

Given that the balloon is inside the bottle, and the bottle is effectively sealed from the outside, the pressure inside the balloon will be a direct reflection of the pressure inside the bottle. Consequently if we

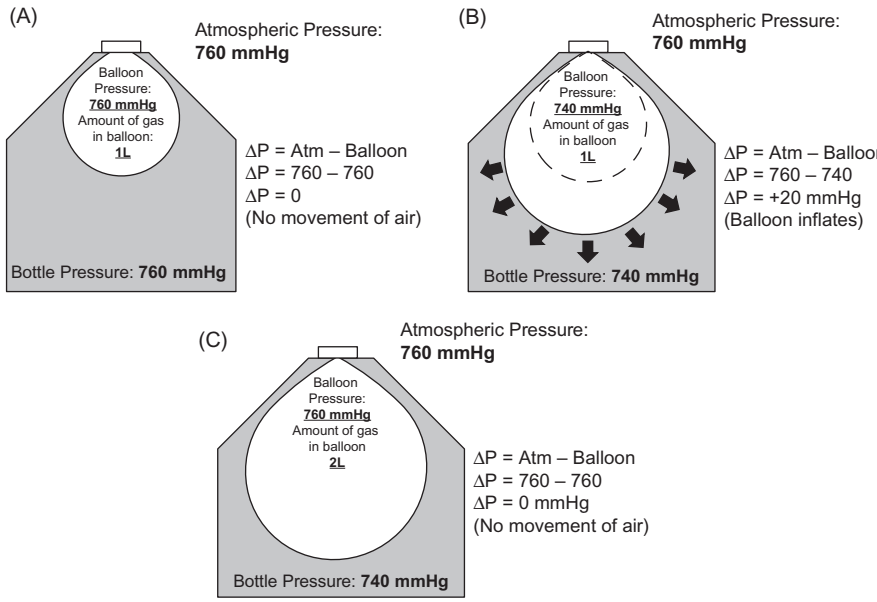

Figure 3.4 The decrease in bottle pressure (A to B) will initially lead to a transient decrease in balloon pressure (B). The decreased balloon pressure will generate a pressure gradient from the outside in. Air will flow down the pressure gradient and fill the balloon with air until the atmospheric pressure and balloon pressures are equal again (C).

decrease the pressure inside the bottle, this creates a gradient relative to the outside, which will in turn decrease the pressure inside the balloon (Figure 3.3C). Think about it like this: In Figure 3.3B the positive atmospheric pressure pushes the balloon open and fills it with air (the black arrows are inside the balloon pushing out). In Figure 3.3C, the pressure inside the bottle is less than atmospheric pressure. That makes it *by definition* a vacuum! It therefore acts as a vacuum in "pulling" the balloon open from the outside (the arrows are outside the balloon pulling its walls open). By pulling on the balloon's walls, two things happen in sequential order (Figure 3.4):

1. The size of the balloon increases (i.e., the balloon gets bigger). This will decrease the air pressure inside the balloon because the same amount of air is distributed in a larger space, therefore in order to equalize the pressure with the outside you need air to flow in (Figure 3.4A, B).
2. Once the balloon starts to get bigger and the pressure inside begins to decrease, air starts flowing into the balloon in order to equalize the pressures. The air flow into the balloon will increase the volume

of gas inside the balloon from 1 L (Figure 3.4B) to 2 L (Figure 3.4C). As the balloon fills with air, the pressure gradient between the outside and the inside of the balloon decreases. When the balloon and the atmosphere once again reach equilibrium at 760 mmHg, the pressure gradient between the atmosphere and the inside of the balloon will disappear and the airflow will stop.

It is important to note that the pressure inside the bottle in Figure 3.4C is still 740 mmHg. In spite of this negative pressure inside the bottle relative to the atmospheric pressure, there is no pressure gradient between the atmosphere and the balloon. What accounts for this? The volume of gas inside the balloon! In Figure 3.4A the balloon requires 1 L of air in order to maintain a pressure of 760 mmHg, however in Figure 3.4C, the balloon requires 2 L of air in order to maintain a pressure of 760 mmHg.

Taking this new set point of 2 L of air in the balloon and a pressure of 760 mmHg as a starting point, what do you think will happen if we further decrease the pressure inside the bottle? Let's take a look. In Figure 3.5A all we've done is taken our balloon with 2L and pasted it there, nothing has changed. The pressure inside the balloon and the atmosphere is the same (760 mmHg). Therefore there is no pressure gradient and air will not be moving. If we decrease the pressure in the bottle to 735 mmHg (Figure 3.5B), what do you think is going to happen? The same thing that happened before when we decreased the pressure in the bottle—the pressure in the balloon will decrease accordingly, generating a pressure gradient from the outside in. This will make the balloon inflate in order to equalize the pressures between the atmosphere and the balloon once again. In this case the volume of air inside the balloon rose by 500 mL to 2.5 L. Once the volume of air in the balloon increases to 2.5 L the pressures equalize and there no longer is movement of air.

What will happen if we then increase the bottle pressure from 735 mmHg back to 740 mmHg? The exact opposite—the pressure inside the balloon will increase to higher than the atmospheric pressure and air would leak out of the balloon until equilibrium was once again reached at 760 mmHg, which would mean moving back to Figure 3.5A. If we were to graphically plot out the changes in volume of air inside the balloon we would get something like Figure 3.5C, where you can see time (sec) on the X-axis and volume of air on the Y-axis. As air goes into the balloon there would be an upstroke (white arrow) in the volume, and as

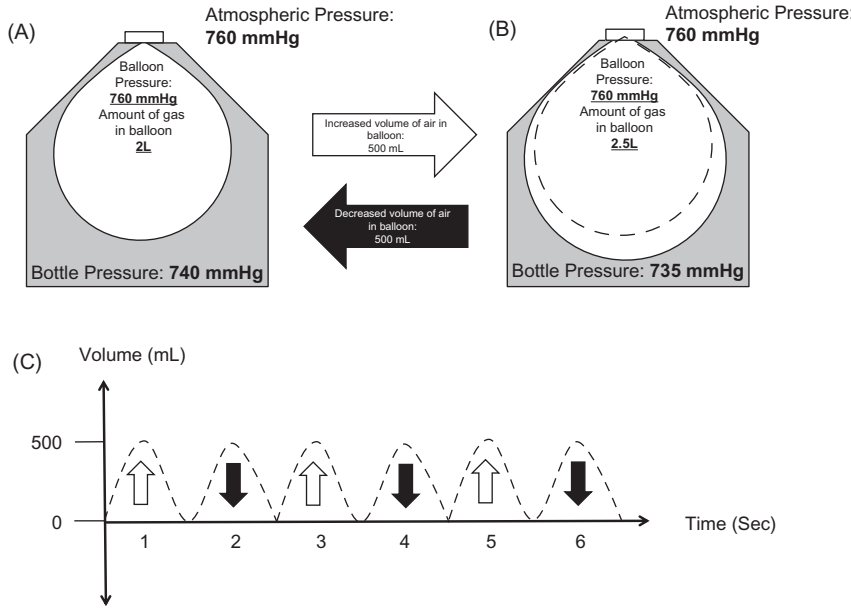

Figure 3.5 Changes in bottle pressure from A to B and then from B to A will lead to a cyclical movement of air in and out of the balloon (C).

air then leaves the balloon, a downstroke (black arrow). So by doing this several times in a row, what we're effectively doing is moving air in and out of the balloon by changing the pressure in the bottle. I think you know where we're going with this: The lungs work in a way that is very similar to our balloon in a bottle, where the bottle represents the chest cavity and the balloon represents the lungs.

PLEURAL PRESSURES: NEGATIVE VERSUS POSITIVE PRESSURE

The lungs and the chest work in a way that is very similar to the balloon-in-a-bottle model that we've been discussing thus far. If you look at Figure 3.6, you'll see some basic anatomy of the respiratory system. In our model, the mouth of the balloon/bottle is the glottis and trachea. The balloon represents the lungs. The bottle is the thoracic cavity. If you imagined the entire inside surface of the bottle and the outside surface of the bottle covered in one contiguous film or membrane, it would be a good approximation of the pleura. The lungs and the pleura hang inside the thoracic cavity and communicate with the outside via the trachea. The glottis acts as the door to the trachea, and can open

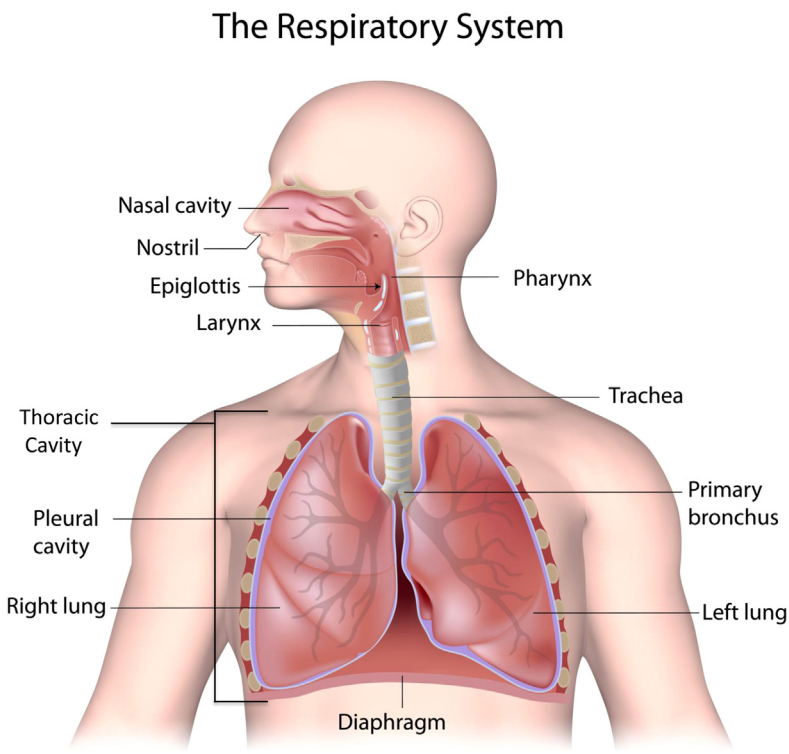

Figure 3.6 Anatomical representation of the ventilation system.

and close. Similarly, the remaining upper airway structures also have some ability to open and close (your mouth, e.g.). If you take a look at Figure 3.7, you'll see a simplified diagram of the relevant anatomy. In it you can see that the pressure inside the chest is negative!! This negative pressure is the one responsible for "propping open" the lungs with air from the outside. How is the negative pressure achieved within the chest? Again referring to Figure 3.7, you'll see that the lungs (white) are hanging inside the pleural space (greyed out area). The pleural space is defined as the space between two very thin layers of cells that cover the outside of the lungs (visceral pleura) and that line the inside of the chest wall (parietal pleura). In order to generate negative pressure in the pleural space, the lymphatic system is constantly removing fluid from the pleural space. Through this constant removal of fluid, the pressure inside the pleural space is negative compared to the outside. In general, the pleural pressure is said to be a pressure of -3 to -5 cmH_2O. Does this mean that the pressure inside the pleural space is a true vacuum?

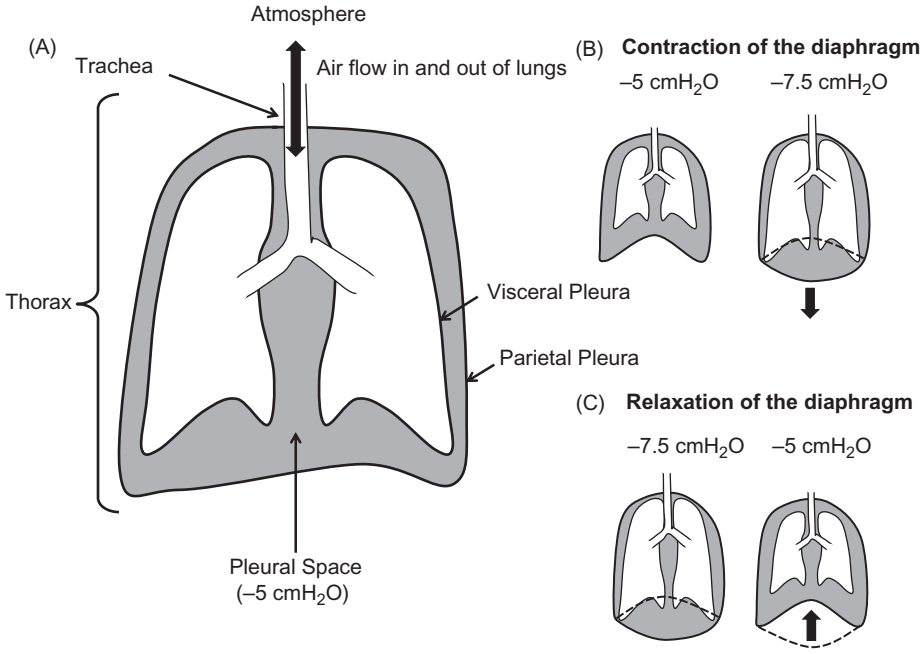

Figure 3.7 Simplified anatomical representation of the lungs and the thoracic cavity (A). When the diaphragm contracts (B) it gets shorter and thereby increases the size of the pleural space. The increased size of the pleural space decreases the pressure from −5 cmH_2O to −7.5 cmH_2O. Conversely, when the diaphragm relaxes it gets longer and decreases the size of the pleural space, thereby increasing the pleural pressure from −7.5 cmH_2O to −5 cmH_2O (C).

Not at all, what this means is that relative to atmospheric pressure the pleural space has a pressure that is slightly less. This "less than atmospheric" pressure is enough to bring air into the lungs!

Clinical Correlate

Pneumothorax

In order to keep the lungs inflated the pleural space needs to have negative pressure. There are certain clinical situations in which there can be a hole in either the parietal pleura (e.g., a rib fracture or stab wound) or in the visceral pleura (e.g., tear in the lung from a burst bullae). These holes allow for air to leak into the pleural space, which collapses the lung. The clinical name for this is a "pneumothorax" or "air in the thorax." If the air leak is caused by a wound that acts as a sort of one-way valve that lets air in but doesn't let it out, massive amounts of air can build up inside of the chest cavity. This type of pneumothorax is called a "tension pneumothorax," and it is a medical emergency because it can compress the heart and great vessels in the chest and cause circulatory collapse.

This is precisely why it kills you if left untreated. The treatment for a tension pneumothorax is a rapid decompression by puncturing a hole in the second intercostal space along the mid-clavicular line of the affected side to relieve the pressure. This should be followed by the placement of a chest tube to correct the negative pressure inside the chest.

The negative pleural pressure we've been discussing thus far (i.e., $-5\ cmH_2O$) serves as our baseline for lung function. This means that our starting point (think Figure 3.7A) is $-5\ cmH_2O$ in the pleural space. And just as we saw with the bottle, the pleural space pressure can be modified by changing the size of the chest cavity.

Key

The pleural pressure is the difference in pressure relative to the atmospheric pressure. If the pressure inside the pleura is less than the atmospheric pressure it is referred to as a negative pressure.

In order to change the size of the chest cavity, we must use the muscles of respiration. These are specialized muscles, which change the size of the thorax as they relax and contract. The most important muscle of ventilation is the diaphragm, which lies just below the lungs and divides the thorax from the abdomen. As the diaphragm contracts, it gets shorter. As it gets shorter it moves down into the abdomen and makes the chest cavity larger! If we increase the size of the chest cavity as in Figure 3.7B, the pressure in the pleural space will decrease from $-5\ cmH_2O$ to $-7.5\ cmH_2O$. Therefore, contracting the diaphragm will decrease the pressure inside the lungs, which makes air flow in (see how the lungs get bigger in Figure 3.7B). Conversely in order to increase the pressure within the chest and force air out, the diaphragm needs to relax (get longer), and as it does this it rises back up, effectively shrinking chest cavity. This will increase the pressure in the chest cavity from $-7.5\ cmH_2O$ to $-5\ cmH_2O$ and air will escape the lungs into the atmosphere (Figure 3.7C). These are the basic tenets of how air moves in and out of the lungs using negative pressure. However, before we completely address the specific pressures of the lung and the chest we need to address two very important concepts: compliance and elastance, since they will be key determinants of the amount of air that moves in and out of the lungs.

LUNGS OUTSIDE THE BODY: TISSUE DYNAMICS

As we've briefly reviewed, the lungs expand and contract in response to changes in the size of the thoracic cavity. But how the lungs respond to changes in the pleural pressure depends on the tissue characteristics of the lungs themselves. So, let's take a quick look at some of the properties of the lung tissue that define how the lungs expand and contract.

Compliance and Elastance

When understanding lung mechanics, there are two concepts that are absolutely key: compliance and elastance. Compliance is defined as change in volume divided by change in pressure, or in other words: How much does the volume change in response to a change in pressure? In equation form:

$$\text{Compliance} = \frac{\Delta\text{Volume}}{\Delta\text{Pressure}}$$

Keeping with our previous example, if a balloon is highly compliant (Figure 3.8A), a small change in pressure will generate a very large change in volume. If the balloon is not compliant (Figure 3.8B), the same change in pressure as before will generate only a small change in volume. If we were to plot this as a graph, the compliance is the slope of the relationship between volume and pressure. (Remember from algebra that you get the slope by plotting $\frac{\text{rise}}{\text{run}}$; in this case $\frac{\text{rise}}{\text{run}} = \frac{\Delta\text{Volume}}{\Delta\text{Pressure}}$). This means that the slope represents the compliance. So, if we were to calculate the compliance of **A** and **B**, we would get the following.

Compliance of balloon in Figure 3.8A

$$= \frac{6\text{L} - 1\text{L}}{0\ \text{cmH}_2\text{O} - (-5\ \text{cmH}_2\text{O})} = \frac{5\ \text{L}}{5\ \text{cmH}_2\text{O}} = 1\ \text{L/cmH}_2\text{O}$$

Compliance of balloon in Figure 3.8B

$$= \frac{2\text{L} - 1\text{L}}{0\ \text{cmH}_2\text{O} - (-5\ \text{cmH}_2\text{O})} = \frac{1\text{L}}{5\ \text{cmH}_2\text{O}} = 0.2\ \text{L/cmH}_2\text{O}$$

Take a look at Figure 3.8C and you'll be able to see just this. The graph depicts the compliance of both balloons plotted against each other. It very clearly shows that with the same ΔPressure (5 cmH_2O) the ΔVolume is higher for A (more compliant) and lower for B (less compliant).

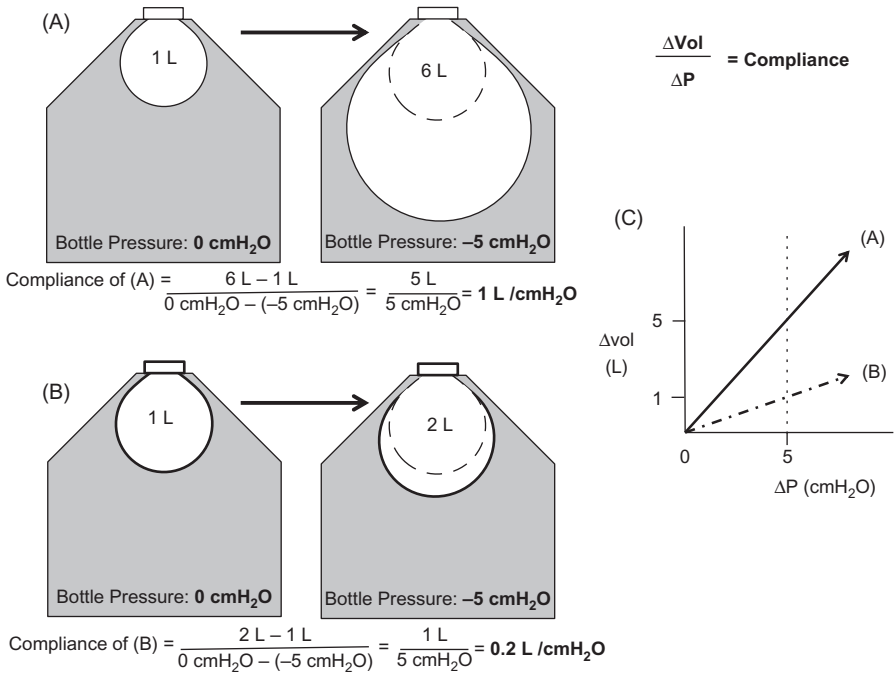

Figure 3.8 Compliance is determined as the Δvol/Δpressure. The balloon in (A) is more compliant than that of (B) because with the same Δpressure, the volume in (A) increases 5 times as much as the volume in (B). A graphical representation of the compliance of both balloons (C), clearly shows how A is more compliant than B.

Key

Compliance is defined as ΔVolume/ΔPressure, and it defines how much the volume will change in response to changes in pressure.

In Figure 3.8, the reason behind the difference in compliance between the two balloons is very easy to identify! (Just in case you haven't, look at the thickness of the balloon wall.) The balloon in Figure 3.8A has a wall that is very thin relative to that of the balloon in Figure 3.8B. Therefore we can say that something that is more compliant is more easily stretched out, while something that is less compliant is harder to stretch out. Along the same lines, something that is easily stretched out is less likely to recover its original shape, whereas something that opposes stretch is more likely to recover its original shape. The property of opposing stretch is called elastance and it's the reciprocal of compliance.

$$\text{Elastance} = \frac{\Delta\text{Pressure}}{\Delta\text{Volume}}$$

This means that the more compliant something is, the less elastic it will be, and the more elastic something is, the less compliant it will be. So, going back to Figure 3.8, the balloon in **A** is very compliant and not very elastic, whereas the balloon in **B** is not very compliant and very elastic. Why is this important? Understanding compliance and elastance is key in understanding lung mechanics because the compliance and elastance of the lungs determine how much they will inflate or deflate with changes in pleural pressure. The relationship between compliance and pleural pressure is the focus of our next subsection.

Transpulmonary Pressure

Thus far, whenever we've changed the pressure inside the bottle (e.g., Figures 3.4, 3.8) the change of pressure inside the balloon has been identical (i.e., if we decreased the pressure inside the bottle by 5 cmH_2O the pressure in the balloon changed by the same 5 cmH_2O). This would mean that absolutely no pressure is lost on the rubber of the balloon itself. This is not entirely accurate. In order to inflate the balloon, some of the pressure will be lost on the rubber that makes up the balloon; that is, the rubber that composes the balloon is also going to have a resistance to stretching that needs to be overcome in order to inflate the balloon. In Figure 3.8 we saw two balloons that had different compliances. We mentioned only that it had to do with the wall thickness. If you take another look at these balloons, though, you'll see the increased wall thickness of balloon B makes it less compliant. Since balloon B is less compliant, it means that for the same change in pressure (in this case, -5 cmH_2O) the change in volume is small compared to a more compliant balloon A. If we wanted to increase the volume in balloon B to a full 5 L we would theoretically require a pressure of -25 cmH_2O. That's five times as much pressure for balloon B as for balloon A—what the heck is all the extra negative pressure doing? The simple answer is that it's holding the wall of the balloon open, so the thicker the wall, the more pressure that's going to be required.

The lungs work in a very similar fashion. As we previously discussed, the lungs are made up of multiple types and layers of tissue with the sole purpose of getting air to the alveolar sac where it can be used in the diffusion of O_2 and CO_2. All these tissue fibers, and predominantly collagen and elastin, are going to behave similarly to the

wall of the balloon we've been discussing. Figure 3.9A shows us the lungs sitting inside the pleural space, but inside the lungs you will see a gross representation of the alveolar sacs. Since it's in the alveolar sacs where exchange takes place, it's in the alveolar sacs where the pressure needs to decrease to allow air to flow in and increase to push the air out. So keep in mind that what we're really interested in is the change in pressure at the level of the alveolar sacs.

Take a look at Figure 3.9B; it's the same schematic as Figure 3.9A, but this time, we've included the ΔP in cmH_2O relative to the atmosphere. So, this means that if the pressure is 0 cmH_2O (it doesn't mean that the pressure in the alveoli is a vacuum), it means that the ΔP between the alveoli and the atmosphere is 0 cmH_2O. In other words, it is the same pressure in the alveoli as in the atmosphere, so there is no net pressure gradient for the movement of air. If 0 cmH_2O is our baseline, when we mention that the pressure in the alveoli becomes negative that means that it is lower than the atmosphere and air will flow into the lungs. When the pressure in the alveoli becomes positive it means that the pressure in the alveoli is higher than the pressure in the atmosphere and therefore air will flow out of the lungs.

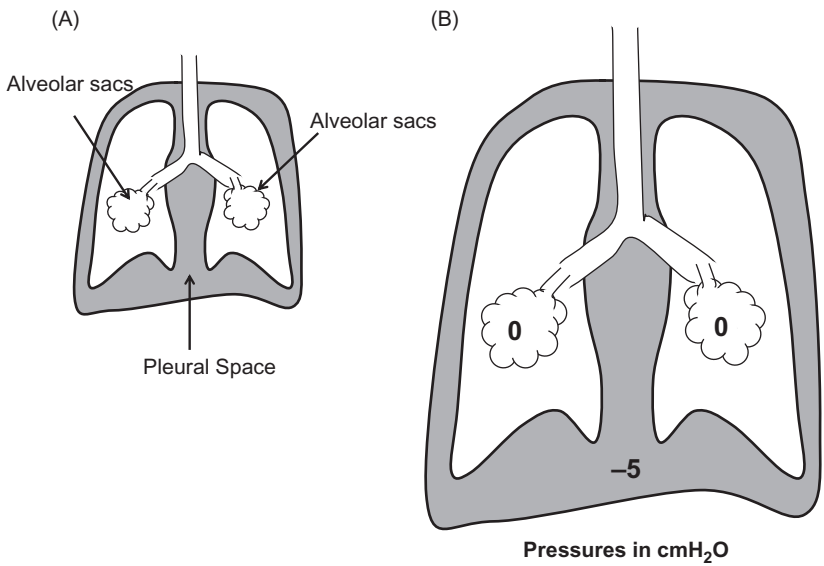

Figure 3.9 The alveolar pressure will always try to equilibrate with the atmospheric pressure. Once equilibrated there will be NO pressure gradient between the alveoli and the atmosphere; i.e., the Δ pressure will be 0 cmH_2O.

Let's take a look at Figure 3.10. This is a busy figure, so we'll walk you through it step by step. Across the top are schematics for different alveolar pressures and the corresponding pleural pressures. The sequence of events that we're about to analyze happens almost simultaneously. However, in order to make the progression from one step to the other a little clearer, we'll break down each step to its simplest elements. The lungs on the left (Figure 3.10A) are the starting point, with a baseline pleural pressure of $-5\ cmH_2O$ and an alveolar pressure difference of $0\ cmH_2O$. Remember a ΔP of $0\ cmH_2O$ in the alveoli means that it's the same as atmospheric pressure. In order to bring air into the lungs, the pressure in the alveoli needs to decrease. This is achieved by increasing the size of the pleural space (like we studied in Figure 3.7). In Figure 3.10B you can see how by increasing the volume of the pleural space, the pleural pressure has gone from $-5\ cmH_2O$ to $-7.5\ cmH_2O$. This sudden decrease in pleural pressure will decrease the alveolar pressure difference from $0\ cmH_2O$ to $-1\ cmH_2O$, which means that the alveolar pressure is now $1\ cmH_2O$ *lower* than the atmospheric pressure. Given this pressure difference, air will begin to flow

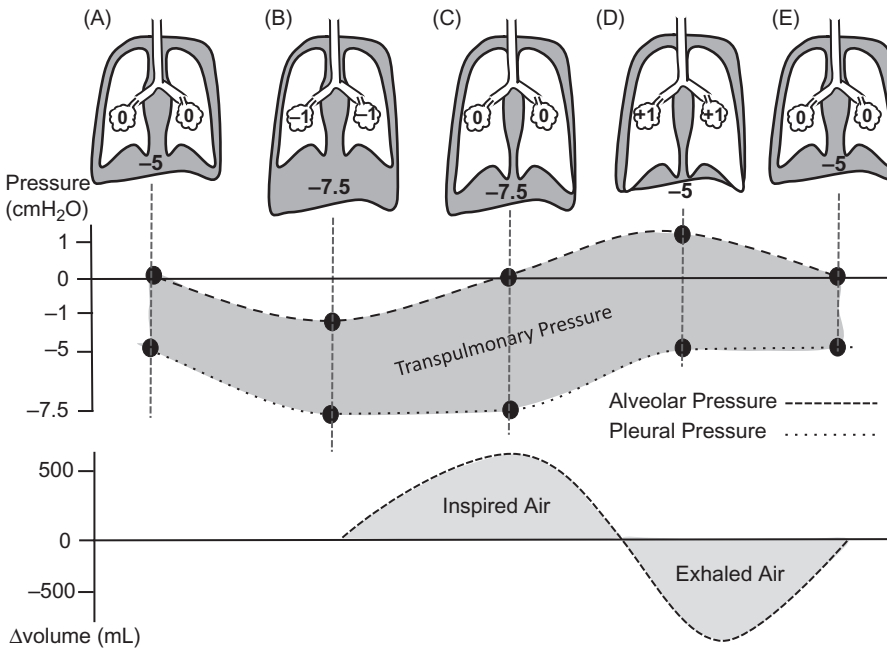

Figure 3.10 The respiratory cycle. Sequential changes in the pleural pressure lead to changes in the alveolar pressure. When the alveolar pressure is negative with respect to the atmospheric pressure, air flows into the lungs. When the alveolar pressure is positive with respect to the atmospheric pressure, air flows out of the lungs.

into the lungs and they will inflate (Figure 3.10C). As the lungs get bigger (Figure 3.10C), the pressure difference between the atmosphere and the alveoli disappears. Even though the pleural pressure is still $-7.5\ cmH_2O$, the alveolar pressure is now $0\ cmH_2O$ due to the increase in lung volume.

So that's how you get air in. But how do you push the air out? By reversing the alveolar-atmosphere pressure gradient. This is achieved by increasing the pleural pressure from $-7.5\ cmH_2O$ to $-5\ cmH_2O$ (Figure 3.10D). As the pressure inside the pleural space increases, the excess volume inside the lungs will be compressed, which will in turn increase the pressure in the alveoli to $+1\ cmH_2O$ relative to atmospheric pressure. This pressure difference now favors the exit of the air from the lungs into the atmosphere (Figure 3.10E) and a subsequent return to baseline volume and pressure.

All the concepts that we just discussed are presented in graphical format in the bottom-most part of Figure 3.10. Let us first draw your attention to the bottom graph. In it you can see the changes in volume (Y-axis) that are associated with changes in pressure (X-axis) within the lungs. And, as we just saw, when the alveolar pressure is lower than that of the atmosphere (Figure 3.10B), air will begin to flow in until the pressure is equalized (Figure 3.10C). In this particular case, the $-1\ cmH_2O$ pressure difference moves 500 mL of air into the lungs (shaded area labeled inspired air). Conversely, a $+1\ cmH_2O$ increase in alveolar pressure pushes 500 mL of air out of the lungs (shaded area labeled exhaled air). Notice that, in the balloons with which we had been working, the change in pressure in the balloon was identical to the change in pressure in the bottle. In the lungs however, the change in pleural pressure is *larger* than the change in alveolar pressure; that is, the pleural pressure changes $2.5\ cmH_2O$ for every $1\ cmH_2O$ change in alveolar pressure. This means that you need a larger pressure difference to expand or contract the lungs. This difference in pressure is called the *transpulmonary pressure*, and literally means the pressure across the lung. It is the measured pressure difference between the pleural pressure and the alveolar pressure.

$$\text{Transpulmonary Pressure} = \text{Alveolar Pressure} - \text{Pleural Pressure}$$

Let's analyze this a bit further. The alveolar pressure in both Figures 3.10A and 3.10C is the same (0 mmHg) but the transpulmonary

pressure is different. Figure 3.10A has a transpulmonary pressure of −5 cm H_2O, and in Figure 3.10C it's −7.5 cm of H_2O. What's going on? Well, look at the volume of both lungs. The volume of Figure 3.10A is less than the volume of Figure 3.10C. Therefore, we can say that the pressure in the alveolus is equal to the pressure in the atmosphere; that is, the pressure gradient is 0 cmH_2O. The transpulmonary pressure is the amount of pressure we need to keep the lung volumes constant. If the transpulmonary pressure remains constant there will be no change in lung volume. It is the changes in transpulmonary pressure that modify alveolar pressures, but what exactly is transpulmonary pressure a function of?

Lung tissue naturally opposes stretching (this is a function of both tissue resistance and surface tension), and transpulmonary pressure is a measure of how much resistance the lung poses to stretching. This concept is directly related to compliance! The more compliant a lung is, the less pressure you need to change the volume. Therefore a lung that is highly compliant will require a SMALL transpulmonary pressure to inflate; it has a small resistance to stretching, which means you only need a small pressure gradient to stretch it. Meanwhile, a lung that is not compliant, which requires a lot of pressure to change the volume, will require a very LARGE transpulmonary pressure to inflate. How is this even remotely relevant to clinical medicine, you ask? Well, if the lung is very stiff and requires a large transpulmonary pressure, it's going to be difficult to change the pressure in the alveoli and therefore extremely difficult to inflate the lungs! So diseases that alter lung compliance will affect transpulmonary pressure, which will have a direct effect on lung function.

How exactly can disease alter lung compliance? There are two particular disease states that can modify lung compliance: emphysema and fibrosis. Emphysema destroys the collagen and elastin fibers in the lung, thereby making the lungs highly compliant (floppy), whereas fibrosis deposits fibrous tissue that stiffens the lung and makes them less compliant (stiff). Figure 3.11 is a graph depicting the differences between normal lung compliance (**A**), the increased compliance of an emphysematous lung (**B**), and the decreased compliance of a fibrotic lung (**C**). The differences in compliance are clearly evident because the same change in pressure (dotted line) has very different effects on the change in volume in each of the lungs. The more compliant emphysematous

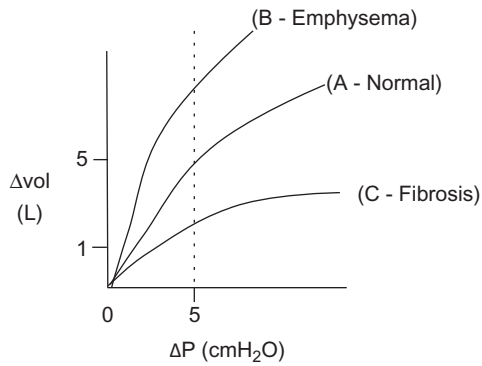

Figure 3.11 Lung compliance curves for (A) normal lungs; (B) Emphysematous lungs, which have an increased compliance (more Δvol); and (C) Fibrotic lungs, which have a decreased compliance (less Δvol) to the same pressure.

lung will have a much larger change in volume than the normal lung or the fibrotic lung, which will only have a modest increase in volume. This is to say, the more compliant a lung is, the easier it is to inflate, whereas the less compliant it is, the harder it is to inflate.

Clinical Correlate

Fibrosis and Emphysema

Lung compliance is a key factor that determines how lungs will behave under different changes in pressure. Emphysema, which destroys the collagen and elastin fibers in the lung, makes the lungs highly compliant, whereas fibrosis, which stiffens the lungs, makes them less compliant. As you can imagine, the clinical scenarios between both conditions are different. One lung has trouble getting air in (fibrosis), while another has trouble getting air out (emphysema)!

Aside from intrinsic tissue resistance, there are two other very important factors that play a role in lung mechanics: alveolar radius and surface tension. However, in order to visualize how radius and surface tension play a role in lung mechanics, we need to graph a compliance curve for the entire ventilation system (i.e., lung and chest wall put together). But to do that we need to understand how the compliance of the entire system is calculated. In Figure 3.11, we saw what the compliance of the lungs outside of the chest cavity looks like. Let's begin by plotting normal lung compliance as we did previously (Figure 3.12). As we saw earlier, the lungs tend to collapse, so we need

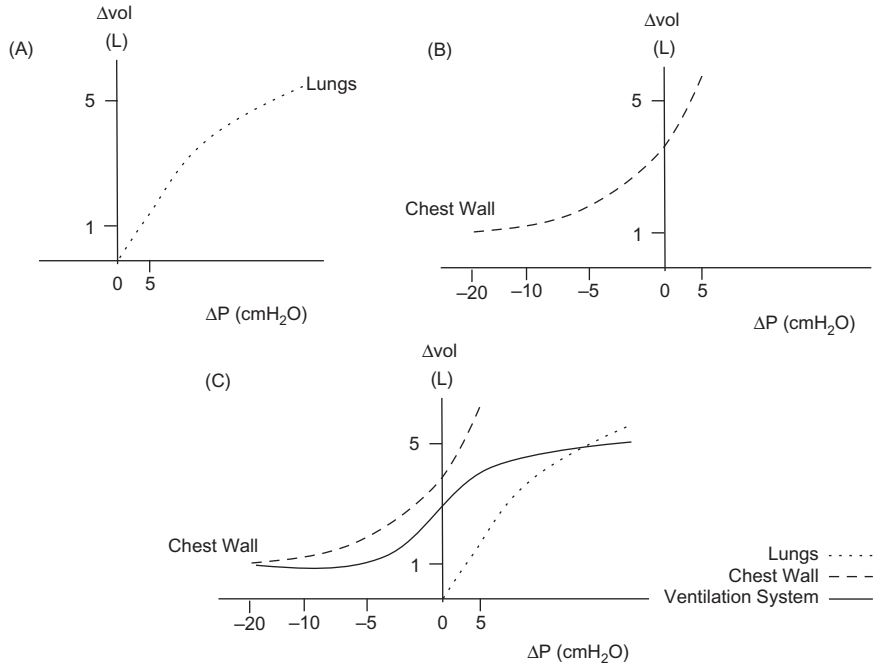

Figure 3.12 The compliance curve of the entire ventilation system (solid black line) (C) is a function of the combined compliance of the compliance of the lungs, which tend to collapse (A), and the compliance of the chest wall which tends to stay open (B).

a positive pressure to keep them open. We can see in Figure 3.12A that as pressure increases, the volume of the lungs increases. The chest wall functions in the opposite manner. The thoracic cavity tends to stay open at baseline, so we need to decrease the pressure inside the chest to get it to collapse. In Figure 3.12B, we see exactly that. As we decrease the pressure, the volume of the chest decreases. The compliance of our entire ventilation system, the lungs situated inside the thoracic cavity, is a combination of these two compliance curves, inward and outward. And that is exactly what we see in Figure 3.12C. The compliance to the ventilation system as whole (solid line) sits in the middle of both compliance curves. The take-home message from this graph is that altering the compliance of either chest wall or the lungs can alter whole ventilation system compliance.

Alveolar Diameter and Role of Surfactant

So far we've discussed "lung resistance" as an abstract concept that is generated by the tissue characteristics of the lungs themselves. It's time

to go into a little more detail. Of the lung resistance we have been discussing, approximately one third is due to the collagen and elastin fibers that are woven into the lung tissue. The remaining two thirds, however, are the result of surface tension. In very simple terms, surface tension is the pull generated by the water molecules that are in contact with air. But in order to understand how surface tension works, let's take a step back and study the law of Laplace.

The law of Laplace states that for a spherical vessel:

$$P = \frac{2T}{r}$$

This means that the pressure to keep the alveoli open (P) is a function of the wall tension (T) and the radius (r). In the alveoli, the greatest contributor of wall tension is the surface tension of the water, which lines the alveolar wall. So, in practical terms, the amount of pressure required to keep the alveoli open (P) increases as the radius (r) decreases or the surface tension (T) increases. When discussing the lungs, P can be roughly equated to transpulmonary pressure. Therefore the transpulmonary pressure required to open the alveoli is a function of the surface tension and the radius of the alveoli. If the radius is small and/or the surface tension is high, we're going to need a higher transpulmonary pressure not only to keep the alveoli open, but also to move air in and out.

Key

A decreased radius or increased surface tension leads to decreased compliance and greater resistance to inflation.

Taking whole ventilation system compliance into account, let's understand the role that surface tension plays on compliance. Take a look at Figure 3.13. On the X-axis we will plot the pleural pressure and on the Y-axis we will plot the ΔVol. Let us draw your attention to the first curve (A). In the example in curve (A), the lungs are filled with saline solution. Even if the lungs are filled with fluid, pressure can still be applied to them in order to expand them. The dotted line (A) represents the compliance curve as the pressure is decreased and increased in saline-filled lungs. The solid line (B), on the other hand, represents the lungs that are filled with air. As amazing as it may

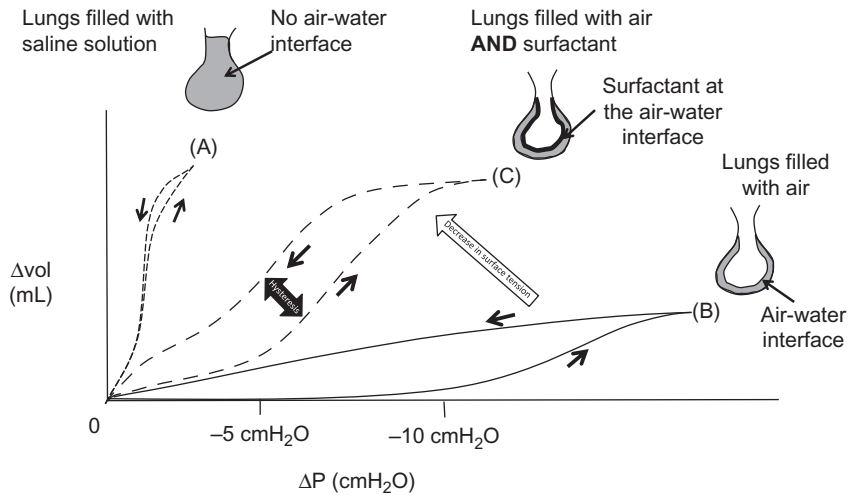

Figure 3.13 Whole ventilation system compliance curves for lungs filled with saline solution (A) no air water interface, lungs filled with air but with no surfactant; (B) high surface tension from air–water interface, and lungs filled with air AND surfactant; (C) decreased surface tension provided by the surfactant, which makes the lungs more compliant.

seem, these compliance curves represent the same lung tissue under different conditions. It only takes a very small amount of pressure to inflate the lungs in A, while the lungs in curve B, even though they're subjected to huge pressures, never really inflate to their full volume. So what's going on?

In Curve A, all the lungs are filled with saline solution, so there is no air–water interface at the level of the alveoli. In this way, only tissue resistance (i.e., collagen and elastin fibers) opposes inflation. With Curve B something else is going on. In physiologic conditions, there is always a small amount of water that lines the walls of all alveoli. (Remember from Chapter 2 that the partial pressure of H_2O in the airways is 47 mmHg.) When the lungs are filled with air, the air–water interface that forms at the level of the alveoli generates surface tension. As we said, surface tension is the pull generated by the water molecules that are in contact with air. As the water molecules "pull" toward one another the generated tension, which tends to collapse the alveoli, makes the radius smaller. And, according to the law of Laplace, the smaller the radius, the higher the pressure required to inflate the alveoli.

As you can see, the resistance that the air–water interface possesses is huge. (Just take a look at curve B!) So, how does the body

counteract the enormous resistance generated by surface tension? In simple terms, it makes a special chemical agent called surfactant.

Surfactant, or "surface active agent" is the name give to a group of lipid molecules that have a hydrophobic group (tails) and a hydrophilic group (heads). This property allows them to position themselves right at the air–water interface (imagine pouring oil into a glass of water). As surfactant positions itself at the air–water interface, it disrupts surface tension, and by disrupting surface tension it decreases the pressure inside the alveoli, thereby curtailing alveolar collapse. What would happen to the compliance curve B from Figure 3.13 if we added surfactant? Take a look at compliance curve C. Compliance curve C represents lungs filled with air, but with surfactant lining the alveolar wall. The lungs in compliance curve C are a lot easier to inflate than the lungs in B, all due to the presence of surfactant. Surfactant will not only decrease surface tension across the alveoli, but it will do so in a way that the pressures are evenly distributed across the lung. This is an important feature if trying to maintain an even inflation throughout the lungs. (It's no good if one part of the lung is very well inflated while another is collapsed.)

Clinical Correlate

Respiratory Distress Syndrome

One of the biggest concerns when treating preterm babies i.e. (babies born before 37 weeks of pregnancy) is lung maturity. Surfactant starts being produced in sufficient amounts after 32 weeks. If the baby is born before that, the lack of adequate surfactant production will make breathing extremely difficult. If surfactant is not present, the surface tension will be very high and the pressures required to move air in and out are too high for a baby's tiny chest to be able to move air efficiently. This is called Respiratory Distress Syndrome (RDS). The lung compliance curves for lungs with RDS look similar to the curve C in Figure 3.13. The treatment for RDS is the administration of exogenous surfactant, which will help support the baby until he or she starts producing enough surfactant to allow for an adequate respiratory effort.

Of note, the pressure–volume loops shown in Figure 3.13 look somewhat different from the compliance graph we saw in Figure 3.12C. There seems to be a different compliance to the lungs as they're inflating versus. when they're deflating, no? This difference is

termed *hysteresis*, which is the difference in compliance of the lungs in inspiration versus expiration. A simple, albeit incomplete explanation of this phenomenon is that as the lungs inflate, the alveolar radii increase. As each alveolar radius increases, the alveoli become more compliant (think Laplace). When the lungs are deflating, the opposite occurs. Since the radius is decreasing, the surface tension increases and it shifts the curve to the left. Therefore, even during normal quiet breathing there are variations in lung compliance.

As we have seen, ventilation system compliance can be altered by many factors including tissue resistance, surface tension, and chest wall dynamics. All these variables can alter the amount of air that moves in and out of the lungs in various pathological conditions, and this can ultimately have an impact on the underlying treatment of specific diseases.

CLINICAL VIGNETTES

Scenario 1

A previously healthy 30-year-old man comes into the Emergency Department after being involved in a motor vehicle accident (MVC). He was impacted on the left side by an oncoming vehicle. On arrival his vital signs are HR 99; RR 15; BP 110/65; O_2Sat – 98% on a 40% FiO_2 non-rebreather mask. He's complaining of chest pain, and there are decreased breath sounds on the left side. Since the patient is stable, a chest X-ray is ordered, which shows a large left-sided pneumothorax (air in the pleural space).

1. In this patient is the compliance of the left lung (pneumothorax) and the right lung the same?
   A. Left > Right
   B. Right > Left
   C. The same

Answer: B. Compliance as defined previously is the ΔVolume/ΔPressure; again in simpler terms, how much would the volume of each lung change if the same pressure was applied? In this case, the tissue resistance, which is provided by collagen and elastin, would be the same, but the total resistance is the summation of both tissue resistance and the resistance provided by wall tension. Since there is air in the left pleural space, the negative pressure required to keep the lung open

has been lost. The loss of negative pressure then collapses the lung, which decreases the radius of all the alveoli. By decreasing the radius of the alveoli, the pressure required to inflate the left lung is now a lot greater than the pressure required to inflate the right lung.

2. What would be the best treatment for this patient?
   A. Endotracheal intubation with a very high positive pressure to inflate both lungs accordingly and improve the volume of the left lung.
   B. Endotracheal administration of surfactant to decrease the surface tension in the left lung so the patient can distend the left lung.
   C. Decompression of the left thoracic cavity with a chest tube in order to remove the air and recover the negative intrapleural pressure.
   D. Leave the patient alone, he seems to be able to breathe with one lung.

Answer: C. The goal of treatment is to try and correct the initial problem. In this case after the MVC the patient probably ruptured either the parietal or the visceral pleura, which allowed air to enter the pleural space. Even though the patient is stable, option D is not ideal given the mechanism of injury and the size of the pneumothorax. (Small pneumothoraces can be observed in a select group of patients, and the body will, slowly but surely, eliminate the excess air if there is no persistent leak.) Increasing the pressure of inflation through endotracheal intubation would not be ideal because both lungs would be subject to the increased pressure and this could potentially lead to an overdistention of one or both lungs, causing alveolar injury and further damage to the lung tissue. Administration of surfactant would also not be appropriate because assuming this patient was healthy, would be producing more than enough surfactant, adding more would have no effect on lung function.

Scenario 2

An obese 40-year-old man comes to your office for a check up. Since his last visit, he has put on 10 Kg, and now weighs 120 Kg and has a BMI of 44 kg/m^2. He complains of difficulty breathing when lying down, but after a thorough examination there is no indication of heart failure.

1. How would obesity impact the compliance of the ventilation system?
   A. Increase compliance
   B. Decrease compliance
   C. No impact on compliance

Answer: B. Obesity decreases chest wall compliance significantly. The addition of fat to the chest wall makes it more difficult to move. Additionally, the deposition of fat in the intercostal muscles, the abdominal cavity and the diaphragm decreases ventilation muscle function. This results in having an increased respiratory effort. (Remember decreased compliance means less volume for the same amount of pressure difference.)