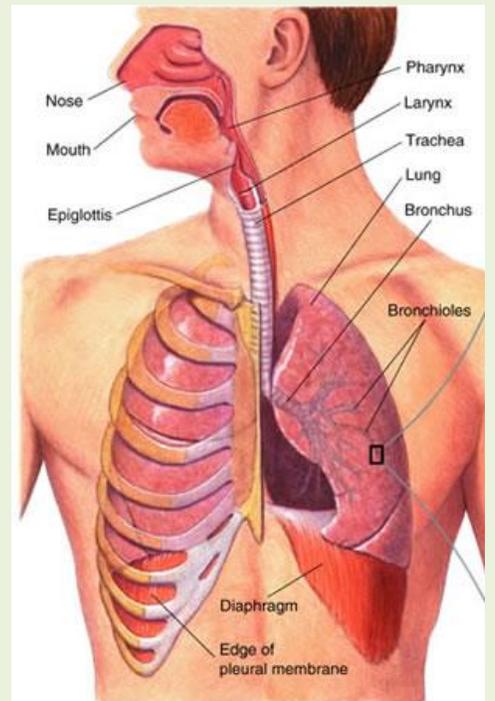
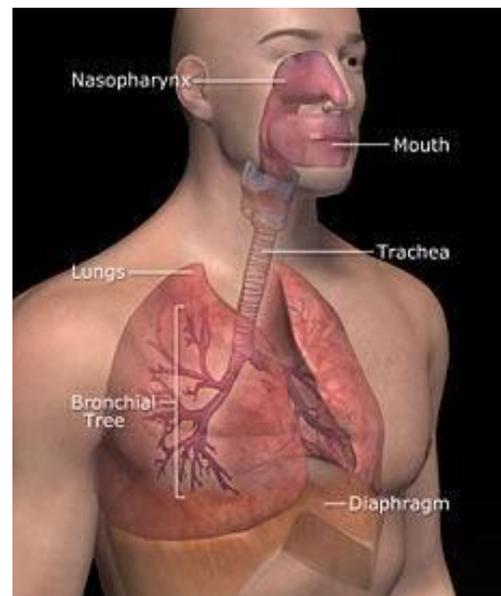


# Mechanics Of Ventilation

You will relate to this story. When I was growing up, I always thought breathing was a conscious event that was instigated in the nose or mouth. After-all, when you think about breathing, the thought process centers on the nose and mouth, so why doesn't breathing start there? Sounds logical, right? Well, of course, relying too much on logic is relying too much on assumptions rather than facts. The control of breathing is a bit of mystery for why we think of the face when we breathe and not our diaphragm. However, hold that thought. Take a few really deep inspirations followed by deep expirations and try to pay attention to what you focus on. As soon as you voluntarily increase the depth of breathing, you do think about your chest – especially expansion of the ribs during inhalation, and compression of the ribcage during exhalation. Perhaps we are neutrally “wired” so that we do not have to divert conscious effort to breathing at rest. So, the crux of this story is clearly that we instigate breathing, or ventilation of the lungs, not from the face, but from the diaphragm during normal resting ventilation. Ventilation is a process of bulk air flow down a pressure gradient generated by expansion of specific region(s) of the lungs, lowering pressure and suctioning air flow through our conducting zone to the respiratory zones of the lungs.



When we breathe air in through our nose or mouth, or both, the process actually starts within our **thoracic cavity**. To understand this, we have to once again go back to some features of the anatomy of the lung and thoracic cavity. Figure 1 shows the **trachea** and diverging bronchioles of the lung conducting zone, connecting to the lungs. Each lung is also connected to a surrounding double membrane (**pleural membranes**) that surrounds the outer layers of the lungs and connects them to inner walls of the thoracic cavity, and in particular to the **diaphragm** muscle at the base of the lungs. Between the two pleural membranes is the **pleural cavity** which contains a thin region of fluid.



**Figure 1. The anatomical structures of the pulmonary system involved in external respiration.**

During resting breathing, the diaphragm contracts and relaxes. During diaphragmatic contraction, the connections between the diaphragm, pleural membranes and lungs cause a small expansion of the pleural cavity and a concomitant decrease in the intra-

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pleural pressure. The connection of the pleural membranes to the lungs transfers this force to the lung, causing expansion and due to this, the lowering of the air pressure within all the air structures (respiratory and conducting) of the lung. If the trachea is open (not blocked by the epiglottis), air then rushes in to equalize this pressure differential, and this is the process of **inhalation** or **inspiration**. Note that the changes in pleural and airway pressures are not large, and approximate up to 7 cmH<sub>2</sub>O, or 5 mmHg at rest. During more forceful ventilation, such pressure differentials increase more rapidly and with higher magnitude, causing more rapid changes in air flow.

How does an increased depth of breathing occur? You are the best model to work with to answer this question. Place your hands over your abdomen, and breathe normally. What happens? During such breathing, you will feel your stomach region distend and then constrict. This is caused by the lowering/contraction (inhalation) and raising/relaxation/recoil (exhalation) of the diaphragm. Now, keep your hands over the abdomen, and then breathe in and out deeply. What else happens to further inflate the lungs? You should notice that you expand and raise your rib cage, and even to a small extent your shoulders. These events also occur in a consistent order;

1. your diaphragm, then
2. your rib cage, then
3. your upper chest and shoulders.

You should also be aware that the additional work of the muscles that move your rib cage out and up function to more evenly and thoroughly inflate the lungs. Remember, quiet breathing at rest primarily inflates the lower regions of the lungs. Then, as we breathe more deeply we further inflate upper regions of the lung, ending in the need to raise the upper ribs to completely inflate the superior regions of the lungs. The muscles that raise and expand the rib cage are the external intercostal muscles. Added superior lung inflation is aided by the accessory muscles of ventilation, consisting of the scalene, superior serratus posterior and sternocleidomastoid muscles.

During **exhalation** or **expiration**, the elastic recoil of the rib cage caused by the relaxation of the muscles of inspiration aid the decrease in lung volume, making exhalation less dependent on muscular contraction, physical work and oxygen consumption. In other words, the **work of breathing** is greater for inhalation than exhalation. This does not mean the exhalation is not without effort. When there is the need for rapid exhalation, such as during exercise, lung and rib cage elastic recoil is aided by contraction of the internal intercostal, abdominal, transverse thoracic and inferior serratus posterior muscles.

## ***Pressure-Volume Relationships***

Figure 2 reveals the **pressure-volume curve** of the lung for inspiration and expiration. Notice that the curve for inspiration is steepest during the mid-range of pleural pressures, meaning that to inflate the lungs during mid-range volumes requires the least effort, with greatest effort (lowest slope) occurring at low and high lung volumes.

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Expiration involves a consistent mono-exponential function, with an increasingly steeper slope (less work per volume exhaled) as lung volume decreases.

To understand the **hysteresis** (different response compared to expiration) of the pressure-volume relationship of inspiration, you need to understand another physical law. The **law of LaPlace** states that for a sphere that can be inflated, and has a given surface tension, the air pressure within the sphere increases as the radius of the sphere decreases, as expressed in Equation 1.

An alternate way to view the pressure component of the equation is to view it as the pressure required to inflate, or maintain inflation of the sphere. This latter interpretation aids application of the law of LaPlace to lung alveoli. For example, this law tells us that when a sphere with a given **surface tension** decreases in radius, as during exhalation, there is an increasing intra-spherical pressure favoring continued expulsion (exhalation) of air. The law also tells us that a sphere requires the greatest pressure to inflate when the radius is smallest.

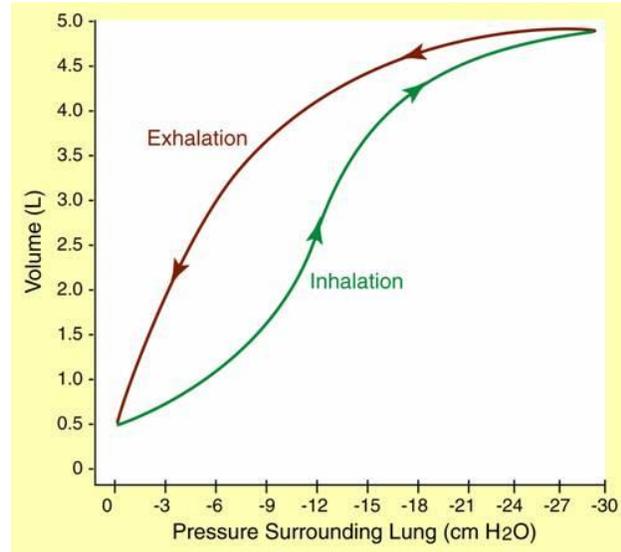


Figure 2. The pressure-volume curve for the lung.

$$\text{Pressure (P)} = \frac{2 T_s}{r}; \text{ where } T_s = \text{surface tension, and } r = \text{sphere radius} \quad \text{Equation 1}$$

When inflating a balloon, have you noticed that the most difficult time to inflate the balloon is in the beginning, when the radius is smallest. Once inflation begins, there is less effort required to inflate the balloon. When you release an inflated balloon, it flies around the room, with a gradually increasing velocity. Why? Because as the radius of the balloon decreases, the pressure within the balloon increases, and the velocity of air release from the balloon increases, providing increasing air propulsion to move the balloon at a faster and faster velocity through the air. The end air expulsion occurs at the fastest velocity, causing a rapid balloon projectile finale followed by an empty balloon that now falls to the ground.

## Roles of Surfactant and the Residual Volume

Clearly, our lungs do not function like a balloon. Thank goodness for this, as we would strain each breathe to inflate our lungs, and during exhalation there would be increasing intra-alveolar forces leading to rapid air expulsion and eventual alveolar collapse. Furthermore, the law of LaPlace tells us that if neighboring alveoli had different dimensions, the largest alveoli would have the lowest air pressure, and air would move from smaller alveoli into the larger one, eventually leading to rupture of the alveoli and

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the inability to evenly inflate the entire lung, or the region of the lung involved in ventilation for the given metabolic condition.

In contrast, as shown in Figure 2, it is relatively easy to inflate the lungs at low lung volumes. You can also see in Figure 2 that at end expiration (prior to the next inspiration) there is already air in the lung, caused by the sum of the residual volume and expiratory reserve volume (note that for the data of the Figure, these curves are for an animal lung and the residual volume presented is smaller than a typical human!). Also, alveoli obviously do not collapse at end expiration. Why?

Well, first of all, let's talk about **surfactant**. Surfactant is a complex substance consisting of phospholipids and apoproteins, and is produced by type II alveolar epithelial cells. Surfactant is diffusely and evenly distributed over the membranes of the alveoli and functions to decrease surface tension. The surfactant concentration surrounding the alveolar membranes is also proportional to the radius of the alveoli, where the surfactant concentrates as the radius decreases, and vice-versa. Thus, because of surfactant, surface tension decreases more as radius decreases, opposing the law of LaPlace and an increasing intra-alveolar pressure, thereby allowing stable alveolar pressures and even alveolar inflation throughout the lung region involved in ventilation. Of course, Figure 2 also shows that surfactant is not perfect in this phenomenon, as the best range of lung inflation associated with least work to generate negative intra-pleural pressures is around mid-lung volumes. Presumably this is the range of alveolar inflation where the surfactant concentration and alveolar radius is best.

Another fair and important question is what causes the **residual volume**? Perform some complete exhalations, as far as you can go in completely exhaling all possible air from your lungs. What do you notice about the effort and force you have to generate to do this? During forced full exhalation, the pleural pressures are increased to such an extent that intra-thoracic pressure is forced to increase and close the small airways within the lung. Figure 3 illustrates the flow volume loop for a cycle of inspiration and expiration during the vital capacity maneuver. Notice the consistent slope of flow during decreasing volume during the exhalation phase of the cycle (top portion of graph). Due to small airway closure, no matter how much we try to force exhalation, we cannot overcome this flow limitation which again is caused by closing small airways. Finally, at end

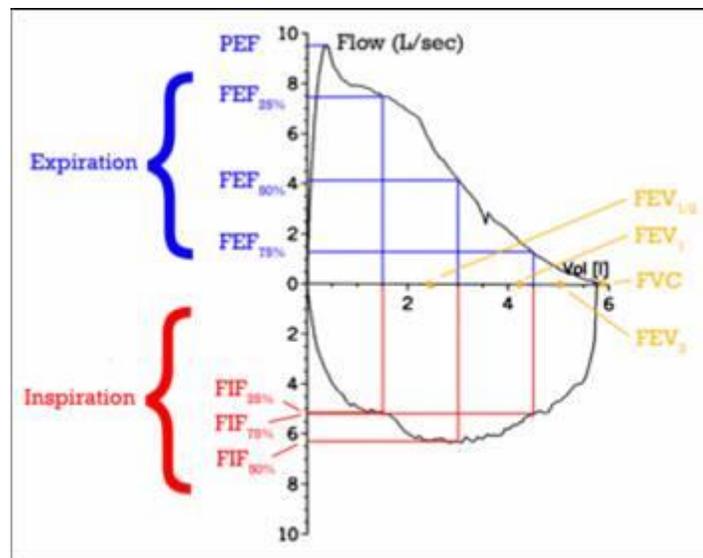


Figure 3. The maximal forced flow volume loops for inspiration and expiration.

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expiration, our airways are closed and there is a volume of air trapped in the lungs – the residual volume.

Measurement of residual volume is a sensitive inexpensive method for diagnosing conditions of the small airways. As the structure of the walls of these airways diminishes, such as during emphysema from cigarette smoking, or prolonged exposure to lung irritants such as pollution or coal dust, the airways are more readily closed when exposed to external pressure, trapping more air in the lung and increasing residual volume. An abnormally large residual volume for your age may be a sign of lung problems, either caused by deteriorating lung structure, or perhaps even by a dysfunction of the mitral valve that causes increased pulmonary circulatory blood pressures and lung dysfunction.

Having a residual volume is a good thing, as it sustains external respiration even when not inhaling air, such as between breaths or during exhalation. It also explains why we can “hold our breath” for perhaps as long as 1 to 2 minutes without collapsing from hypoxia. Another benefit of the residual volume is shown in Figure 2. Starting lung inflation with the lung already partially inflated improves the pressure-volume curve for inspiration, and thus decreases the work of breathing. You should also be able to see the benefit of this based on the law of LaPlace.

## Glossary Words

**thoracic cavity** is the internal region of the body surrounded laterally and superiorly by the rib cages, and inferiorly by the diaphragm muscle.

**trachea** is the long tube directing air flow during ventilation to and from the left and right bronchi.

**plural membranes** surround the lungs and connect them to the internal walls of the thoracic cavity.

**diaphragm** is the muscle located above the abdomen and inferior to the thoracic cavity. The contraction of the diaphragm is responsible for lowering the inferior wall of the thoracic cavity, expansion of the lungs, and the movement of air into the lungs (inhalation or inspiration). Ventilation at rest is totally caused by the contraction of the diaphragm.

**pleural cavity** is the thin fluid filled space between the two pleural membranes that surround the lungs.

**inhalation** is the movement of air by bulk flow into the lungs.

**inspiration** is another term for the movement of air by bulk flow into the lungs.

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**exhalation** is the movement of air by bulk flow out of the lungs.

**expiration** is another term for the movement of air by bulk flow out of the lungs.

**work of breathing** refers to the metabolic cost (typically quantified by  $VO_2$ ) of muscle contraction to cause inspiration and expiration.

**pressure-volume curve** is the resulting relationship between inspiratory and expiratory pressures and air volume (typically lung volume) during inspiration and expiration, respectively.

**hysteresis** is a term used to describe an altered responses. In pulmonology, a hysteresis refers to the different pressure-volume profiles between inspiration and expiration.

**law of LaPlace** is the mathematical law used to define the relationships between a differential pressure, the surface tension of a membrane surrounding a circular structure, and the and circular structure radius.

**surface tension** is a property of liquid-to-air interfaces, where there is a force of attraction between successive water molecules, causing an effective inward force that must be overcome to sustain a dimension, or exceeded to cause expansion.

**surfactant** is a phosphor-lipoprotein molecule synthesized by specialized cells neighboring alveoli, which causes a reduction in surface tension and therefore decreases resistance to alveolar expansion (inhalation).

**residual volume** is the volume of air remaining in the lungs after complete forced exhalation.