

**Functions and organization  
of the respiratory system**

## Learning Objectives

- **By the end of this lecture you will be able to:-**
  - 1-Describe the structures and respiratory zones functions of the conductive and of airways.
  - 2-Understand the difference between internal and external respiration.
  - 3-Understand the functions of the respiratory system, including non-respiratory functions, like clearance mechanism by mucus and cilia, production of surfactant and its physiological significance.

### Functions of the respiratory system include

- Gas exchange (respiratory function).
- Phonation: is the production of sounds by the movement of air through the vocal cords.
- Pulmonary defense
  - Immunoglobulin A (IgA),
  - Alpha-1 antitrypsin
  - *The pulmonary macrophages in the alveoli: engulf smaller particles which pass through the mucocilliary barrier filter.*

#### **Immunoglobulin A (IgA)**

a type of antibody that protects against infections of the mucous membranes lining the mouth, airways, and digestive tract. It is the most common of the primary antibody deficiencies, IgA is the predominant Ig isotype in mucosal tissue and is believed to be involved in defense against viral and bacterial infections at these sites

#### **Alpha-1 antitrypsin**

It is generally known as serum inhibitor, It protects tissues from enzymes of inflammatory cells

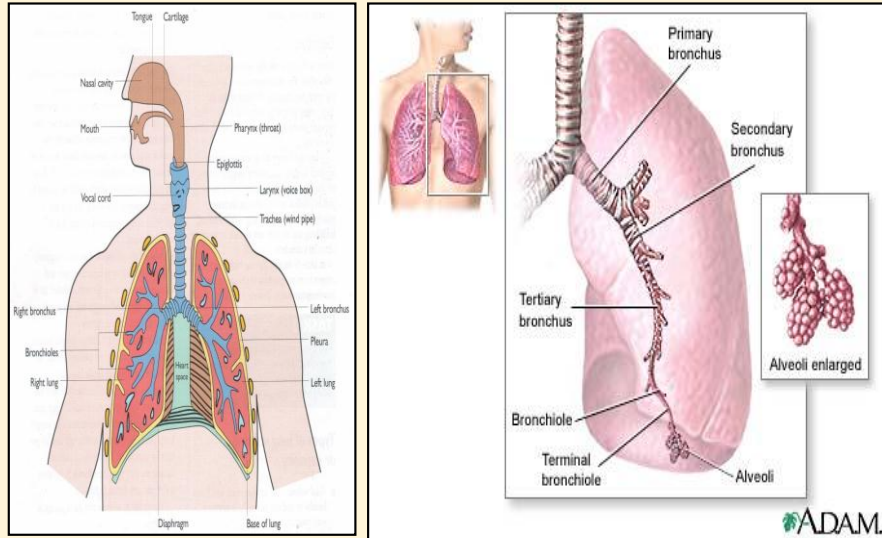
### Cont..non respiratory functions of lung

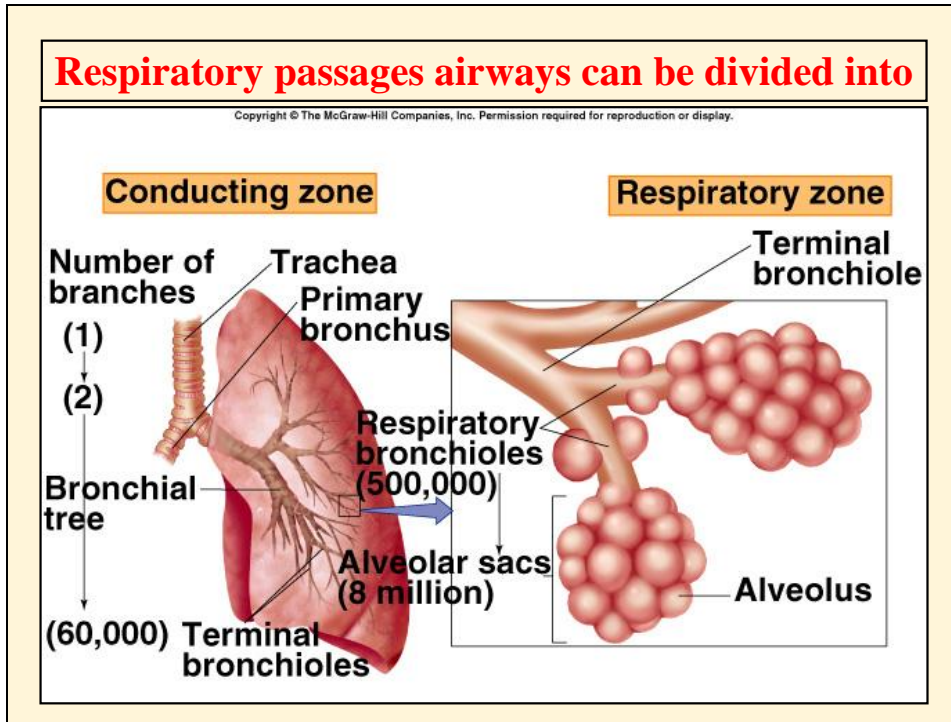
- Angiotensin I is converted to angiotensin II with the help of angiotensin converting enzymes formed by the lungs.
- Regulating the acid- base status of the body by washing out extra carbon dioxide from the blood.
- Secretion of important substances like surfactant .

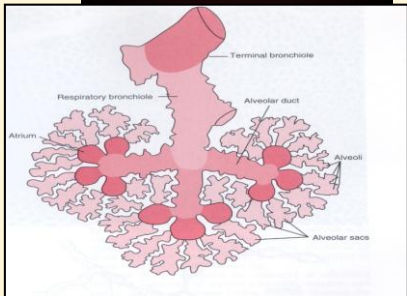
**Angiotensin** is a peptide hormone that causes vasoconstriction and a subsequent increase in blood pressure

**Angiotensin-converting enzyme** or "ACE" indirectly increases blood pressure by causing blood vessels to constrict. It does that by converting angiotensin I to angiotensin II, which constricts the vessels. For this reason, drugs known as ACE inhibitors are used to lower blood pressure.

## Respiratory passages ( airways)

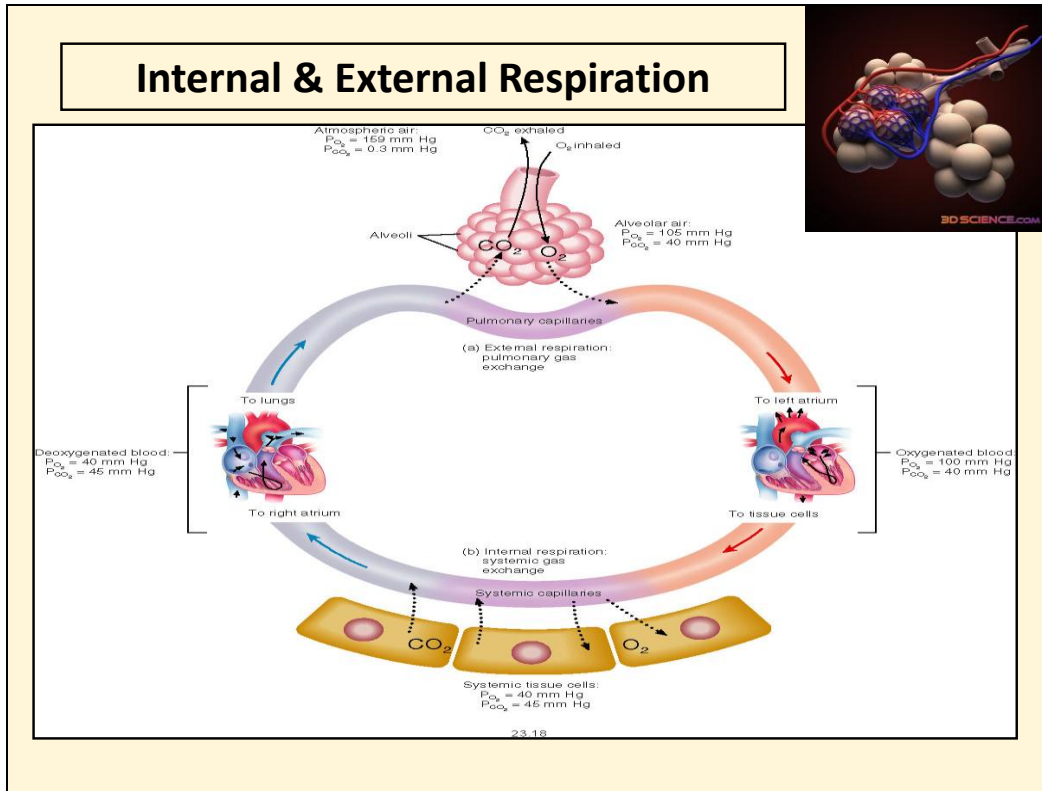




<b>I- Conductive Zone</b>	<b>II- Respiratory Zone (Respiratory unit)</b>
<ul style="list-style-type: none"><li>• Starts from nose to the end of terminal bronchioles.</li><li>• Help <i>warming, humidification, filtration</i> of inspired air.</li><li>▪ Contains the <i>olfactory receptors</i> for smell sensation.</li><li>▪ <i>Conducts the sound</i> during speech.</li><li>▪ Protective function by cough and sneezing reflexes.</li></ul>	<ul style="list-style-type: none"><li>• Includes: Respiratory bronchioles, alveolar ducts, alveolar sacs, alveoli</li><li>• Function in gas exchange.</li></ul> 

the alveoli, alveolar sacs, alveolar ducts, and respiratory bronchioles. The rate at which new air reaches these areas is called *alveolar ventilation*. Or respiratory zone





### **Internal & External respiration**

**3** major functional events occurs during it:

- **1-Pulmonary ventilation**: inward and outward movement of air between lung and atmosphere.
- **2- Diffusion** of oxygen and CO<sub>2</sub> between the alveoli and the pulmonary capillary blood
- **3- Transport** of O<sub>2</sub> & Co<sub>2</sub> in the blood and body fluids to and from the cells

Respiration could be either

*Resting* : normal breathing during resting conditions

*Forced (maximal)*: during exercise, in patients with asthma, allergy,...

### Lining cells of the alveoli

- 1- Type I alveolar cells  
( type I pneumocytes)
- 2- Type II alveolar cells  
( type II pneumocytes)  
(Secrete surfactant)
- 3- Alveolar macrophages

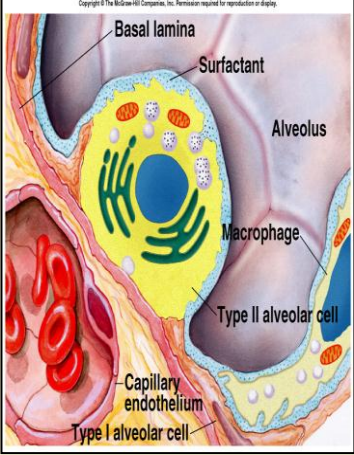
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(a) Labels: Macrophage, Air space within alveolus, Type II pneumocyte (surfactant-secreting cell), Type I pneumocyte, Capillary endothelium (wall), Red blood cell. Grouped as Alveolar epithelium (wall).

(b) Labels: Alveolus, Alveolar fluid (with surfactant), Alveolar epithelium, Basement membrane of alveolar epithelium, Interstitial space, Basement membrane of capillary endothelium, Capillary endothelium, Capillary, Diffusion of O<sub>2</sub>, Diffusion of CO<sub>2</sub>, Red blood cell. Grouped as Respiratory membrane.

### Surface Tension

- H<sub>2</sub>O molecules at the surface are attracted to other H<sub>2</sub>O molecules by attractive forces that resist distension (inflation) called surface tension.
- Surface tension tends to oppose alveoli expansion
- Pulmonary surfactant reduces surface tension



The diagram illustrates the structure of an alveolus and its interaction with a nearby capillary. The alveolus is a sac-like structure lined by a thin layer of Type I alveolar cells. A layer of surfactant is shown coating the inner surface of the alveolus, reducing surface tension. A Type II alveolar cell is shown secreting surfactant into the alveolus. A macrophage is also present in the alveolus. The capillary endothelium is shown adjacent to the alveolus, separated by a basal lamina.

### Principle of Surface Tension.

When water forms a surface with air, the water molecules on the surface of the water have an especially strong attraction for one another. As a result, the water surface is always attempting to contract. This is what holds raindrops together—a tight contractile membrane of water molecules around the entire surface of the raindrop. Now let us reverse these principles and see what happens on the inner surfaces of the alveoli. Here, the water surface is also attempting to contract. This results in an attempt to force the air out of the alveoli through the bronchi and, in doing so, causes the alveoli to try to collapse. The net effect is to cause an elastic contractile force of the entire lungs, which is called the *surface tension elastic force*.

### Surfactant and Its Effect on Surface Tension.

Surfactant is a *surface active agent in water*, which means that it greatly reduces the surface tension of water. It is secreted by special surfactant-secreting epithelial cells called type II alveolar epithelial cells, which constitute about 10 percent of the surface area of the alveoli. These cells are granular, containing lipid inclusions that are secreted in the surfactant into the alveoli.

## Surfactant

- Surfactant is a complex substance containing phospholipids and a number of apoproteins.
- Secreted by the Type II alveolar cells { the earliest detection begins between 6-7<sup>th</sup> month much as the earliest detection but this could be delayed in others} **at wk 35 of** intrauterine life from fetal alveoli.
- Surfactant reduces surface tension throughout the lung, prevents alveolar collapse, decreases airway resistance and the work of breathing.

Surfactant is a complex mixture of several phospholipids, proteins, and ions. The most important components are the phospholipid *dipalmitoylphosphatidylcholine*, *surfactant apoproteins*, and *calcium ions*. The dipalmitoylphosphatidylcholine and several less important phospholipids are responsible for reducing the surface tension. They do this by not dissolving uniformly in the fluid lining the alveolar surface. Instead, part of the molecule dissolves while the remainder spreads over the surface of the water in the alveoli. This surface has from one-twelfth to one-half the surface tension of a pure water surface.

In quantitative terms, the surface tension of different water fluids is approximately the following: pure water, 72 dynes/cm; normal fluids lining the alveoli but without surfactant, 50 dynes/cm; normal fluids lining the alveoli and *with* normal amounts of surfactant included, between 5 and 30 dynes/cm.

## Cont...surfactant

- Deficiency in premature babies cause respiratory distress syndrome of the new born (RDS). ( hyaline membrane disease)
- Smoking in adult, hypoxia or hypoxemia (low oxygen in the arterial blood) or both, decrease the secretion of surfactant and cause adult respiratory distress syndrome.

**distress syndrome of the new born** is a problem often seen in premature babies. The condition makes it hard for the baby to breathe. he disease is mainly caused by a lack of a oily substance in the lungs called surfactant. This substance helps the lungs fill with air and keeps the air sacs from deflating. Surfactant is present when the lungs are fully developed.

example of deficiency of surfactant.

**In neonatal respiratory distress syndrome**

surfactant is lacking. In the developing fetus Infants born before week 24 will *never* have surfactant Without surfactant, small alveoli have increased surface tension and increased pressures, and will collapse (**atelectasis**). Collapsed alveoli are not ventilated and, therefore, cannot participate in gas exchange



### **Innervations of lungs and bronchi**

- Is by autonomic nerves
- Sympathetic causes dilatation of the bronchi
- Parasympathetic causes constriction of the bronchi.
- Locally secreted factors :histamine, slow reacting substances of anaphylaxis (SRSA) by mast cells, due to allergy ( as in patients with asthma) often cause bronchiolar constriction and increase airway resistance.

The **autonomic nervous system (ANS)**, also known as the **visceral nervous system** and **involuntary nervous system**, is a division of the peripheral nervous system that influences the function of internal organs The autonomic nervous system is a control system that acts largely unconsciously and regulates the heart rate, digestion, respiratory rate, pupillary response, urination

SRSA stands for Slow Reacting Substance of Anaphylaxis  
prolonged contraction of  
muscle than does histamine; it is active in the presence of antihistamines



Mechanics of pulmonary  
ventilation

### Learning Objectives

- By the end of this lecture you will be able to:
  - 1- List the muscles of respiration and describe their roles during inspiration and expiration.
  - 2- Understand the importance of the following pressures in respiration: atmospheric, alveolar, intrapleural, and transpulmonary.
  - 3- Explain why intrapleural pressure is always subatmospheric under normal conditions, and the significance of the thin layer of the intrapleural fluid surrounding the lung.
  - 4- Define lung compliance and list the determinants of compliance.

## Muscles That Cause Lung Expansion and Contraction

- lungs can be expanded and contracted:
- (1) downward and upward movement of the diaphragm to lengthen or shorten the chest cavity
- (2) by elevation and depression of the ribs to increase and decrease the anteroposterior diameter of the chest cavity

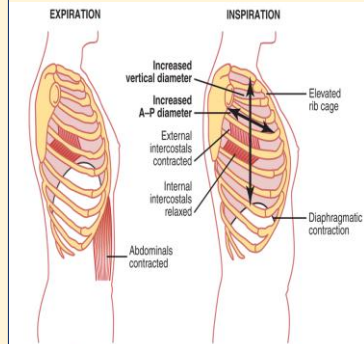
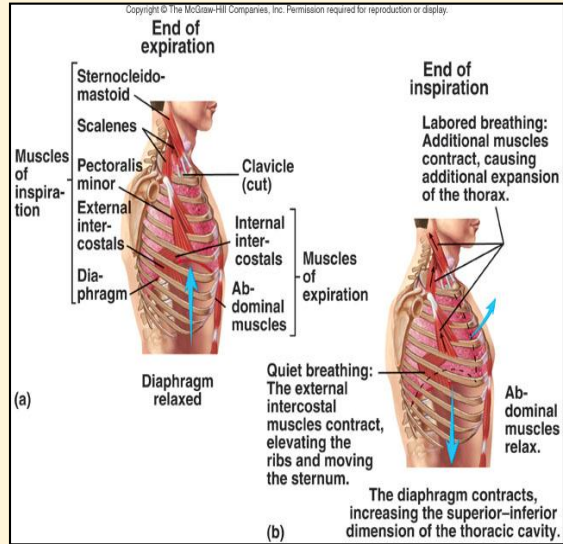


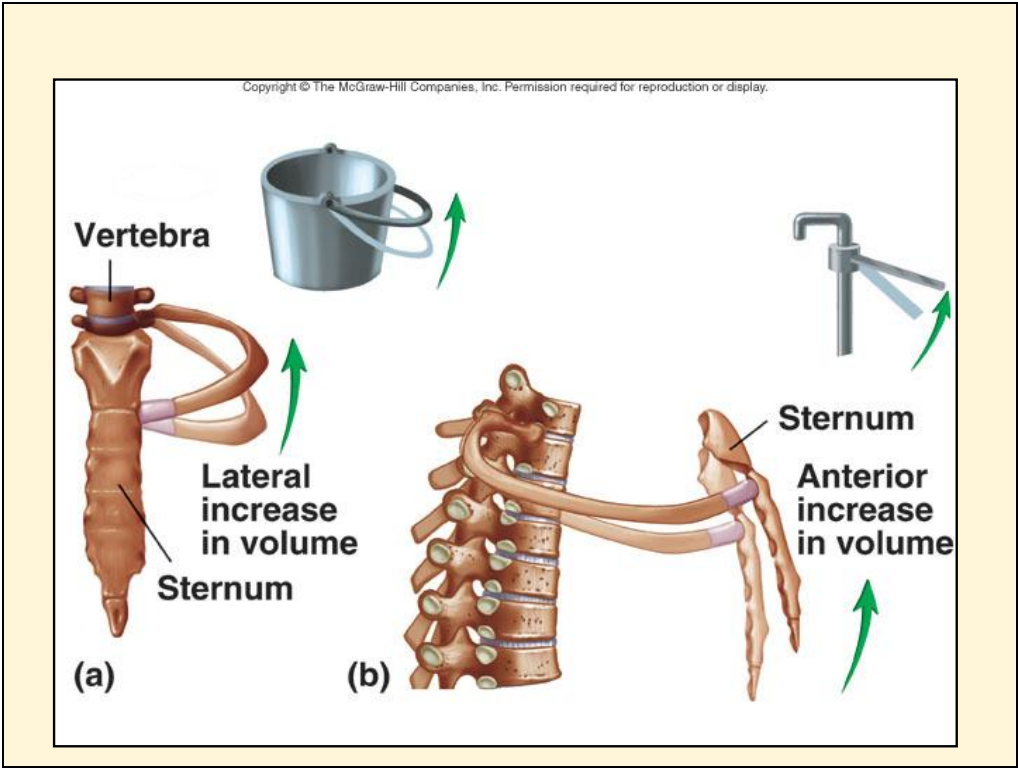
Figure 37-1 Contraction and expansion of the thoracic cage during expiration and inspiration...

## Respiratory muscles

**Inspiratory muscles**  
(resting- forced)

**Expiratory muscles**  
(forced expiration-  
muscles that depress  
the rib cage)



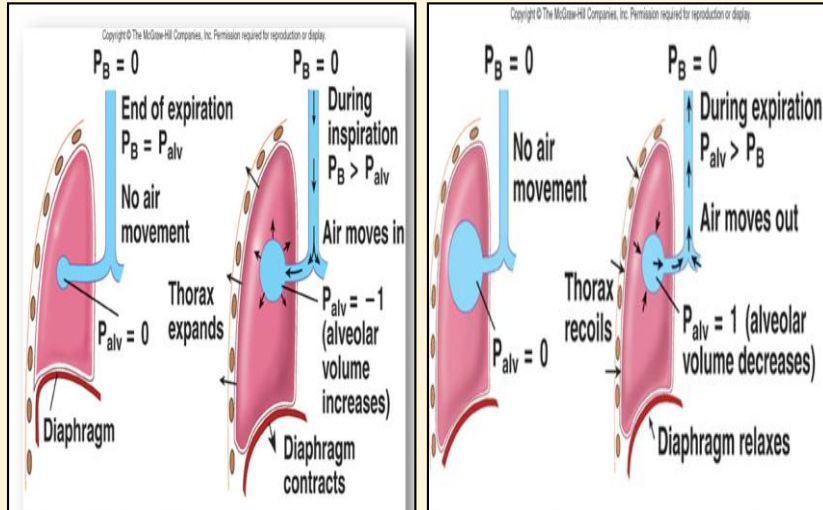


## Deep Forceful Breathing

- Deep Inspiration
  - During deep forceful inhalation accessory muscles of inspiration participate to increase size of thoracic cavity
    - Sternocleidomastoid – elevate sternum
    - Scalenes – elevate first two ribs
    - Pectoralis minor – elevate 3<sup>rd</sup>–5<sup>th</sup> ribs
- Deep Expiration
  - Expiration during forceful breathing is active process
  - Muscles of exhalation increase pressure in abdomen and thorax
    - Abdominal muscles
    - Internal intercostals

all the muscles that elevate the chest cage are classified as muscles of inspiration, and those muscles that depress the chest cage are classified as muscles of expiration. The most important muscles that raise the rib cage are the *external intercostals*, but others that help are the (1) *sternocleidomastoid* muscles, which lift upward on the sternum; (2) *anterior serrati*, which lift many of the ribs; and (3) *scaleni*, which lift the first two ribs. The muscles that pull the rib cage downward during expiration are mainly the (1) *abdominal recti*, which have the powerful effect of pulling downward on the lower ribs at the same time that they and other abdominal muscles also compress the abdominal contents upward against the diaphragm, and (2) *internal intercostals*.

Air will flow from a region of high pressure to one of low pressure-- the bigger the difference, the faster the flow



**Pressure changes in the lungs during breathing**

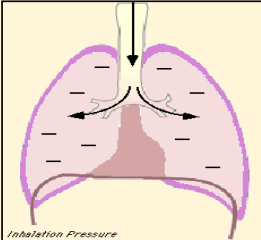
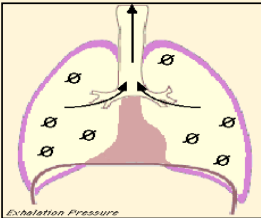
**1-Intra-alveolar (intrapulmonary pressure)**

Between breathes = zero pressure

During inspiration = (-1 mmHg). air (tidal volume) flow from outside to inside the lungs).

At the end of inspiration = zero. air flow stops.

During expiration = (+1 mmHg). air flow out of the Lungs

**1-Intra-alveolar (intrapulmonary pressure )or Alveolar Pressure**

*Alveolar pressure* is the pressure of the air inside the lung alveoli. When the glottis is open and no air is flowing into or out of the lungs, the pressures in all parts of the respiratory tree, all the way to the alveoli, are equal to atmospheric pressure, which is considered to be zero reference pressure in the airways—that is, 0 cm water pressure. To cause inward flow of air into the alveoli during inspiration, the pressure in the alveoli must fall to a value slightly below atmospheric pressure (below 0). The second curve (labeled “alveolar pressure”) of Figure 37-2 demonstrates that during normal inspiration, alveolar pressure decreases to about -1 centimeters of water. This slight negative pressure is enough to pull 0.5 liter of air into the lungs in the 2 seconds required for normal quiet inspiration.

During expiration, opposite pressures occur: The alveolar pressure rises to about +1 centimeter of water, and this forces the 0.5 liter of inspired air out of the lungs during the 2 to 3 seconds of expiration.



- **2-Intrapleural pressure (IPP):**

Pressure in the pleural space

is negative with respect to atmospheric pressure at the end of normal expiration( -5cmH<sub>2</sub>O).

- Why negative??:

1- The lung's elastic tissue causes it to recoil, while that of the chest wall causes it to expand. Because of these 2 opposing forces the pressure in the pleural cavity becomes negative.

2-The pleural space is a potential space, empty due to continuous suction of fluids by lymphatics.

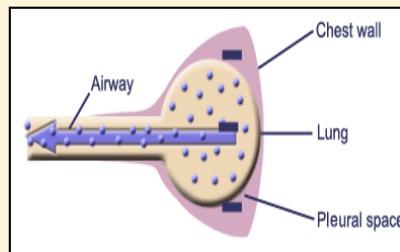
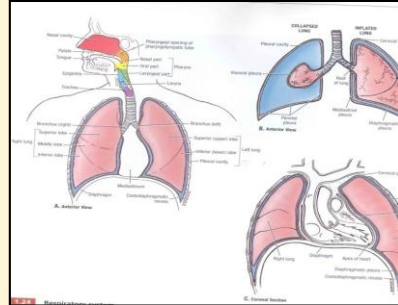
### **Pleural Pressure and Its Changes During Respiration or **Intrapleural pressure (IPP):****

Pleural pressure is the pressure of the fluid in the thin space between the lung pleura and the chest wall pleura. As noted earlier, this is normally a slight suction, which means a slightly *negative* pressure. The normal pleural pressure at the beginning of inspiration is about -5 centimeters of water, which is the amount of suction required to hold the lungs open to their resting level. Then, during normal inspiration, expansion of the chest cage pulls outward on the lungs with greater force and creates more negative pressure, to an average of about -7.5 centimeters of water.

**Figure 37-2** Changes in lung volume, alveolar pressure, pleural pressure, and transpulmonary pressure during... These relationships between pleural pressure and changing lung volume are demonstrated in Figure 37-2, showing in the lower panel the increasing negativity of the pleural pressure from -5 to -7.5 during inspiration and in the upper panel an increase in lung volume of 0.5 liter. Then, during expiration, the events are essentially reversed.

### Values of IPP

- (-5) cm H<sub>2</sub>O during resting position between breathes, and it becomes more -ve (-7.5) cm H<sub>2</sub>O during resting inspiration.
- Forced ventilation  
Insp. :-20 to -40 cm H<sub>2</sub>O  
Exp. : + 30 cm H<sub>2</sub>O



### 3-Transpulmonary pressure (TPp) (Extending Pressure)

- *The difference between the alveolar pressure (Palv) and the pleural pressure (Ppl).*

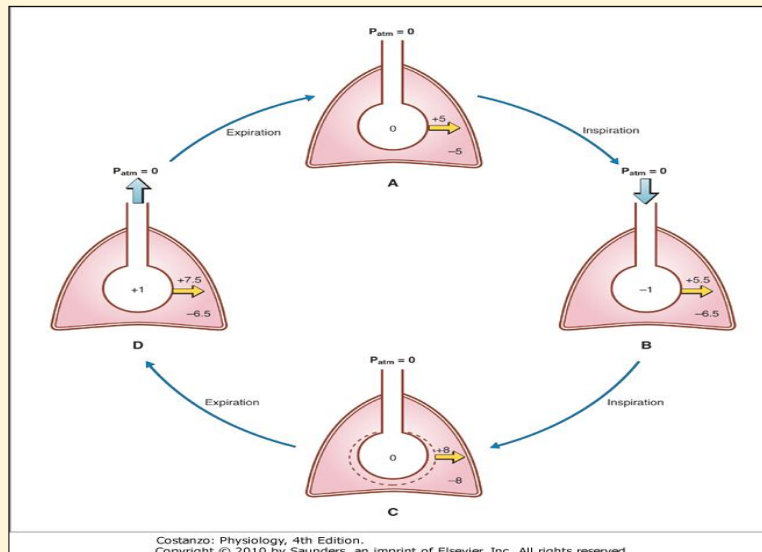
$$TPp = P_{alv} - P_{pl}$$

- *It is a measure of the elastic forces in the lungs that tend to collapse the lungs (**the recoil pressure**).*
  - It prevents lung collapse.
- The bigger the volume of the lung the higher will be its tendency to recoil.

### Transpulmonary Pressure.

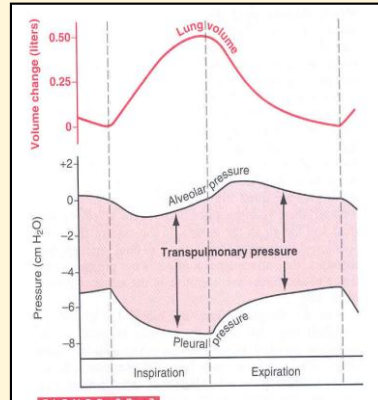
Finally, note in [Figure 37-2](#) the difference between the alveolar pressure and the pleural pressure. This is called the *transpulmonary pressure*. It is the pressure difference between that in the alveoli and that on the outer surfaces of the lungs, and it is a measure of the elastic forces in the lungs that tend to collapse the lungs at each instant of respiration, called the *recoil pressure*.

pressure and volume relationships in a single respiratory cycle.



## V<sub>c</sub> ( **Compliance of the lung** ) in a single respiratory cycle

E.g. two rubber bands, thin and thick. The thin rubber band easily stretched, and is very distensible and compliant. The thick rubber band difficult to stretch and is less distensible and compliant.



### **Compliance of the Lungs**

The extent to which the lungs will expand for each unit increase in transpulmonary pressure (if enough time is allowed to reach equilibrium) is called the lung compliance. The total compliance of both lungs together in the normal adult human being averages about 200 milliliters of air per centimeter of water transpulmonary pressure. That is, every time the transpulmonary pressure increases 1 centimeter of water, the lung volume, after 10 to 20 seconds, will expand 200 milliliters.

- Is defined as, the ratio of the change in the lung volume produced per unit change in the distending pressure.
- The extent to which the lungs expand for each unit increase in the transpulmonary pressure.

- $CL = \frac{\text{Volume change } (\Delta V)}{\text{Transpulmonary pressure change } (\Delta P)}$

- $CL = \frac{(\Delta V)}{(\Delta P)}$

### Cont...compliance of lung

- For both lungs in adult = 200 ml of air /cm H<sub>2</sub>O.
- For lungs and thorax together = 110 ml/cm H<sub>2</sub>O.
- Is reduced in pulmonary fibrosis , pulmonary edema, diseases of the chest wall ( kyphosis, scoliosis)
- Emphysema increases the compliance of the lungs because it destroys the alveolar septal tissue rich with elastic fibers that normally opposes lung expansion.

