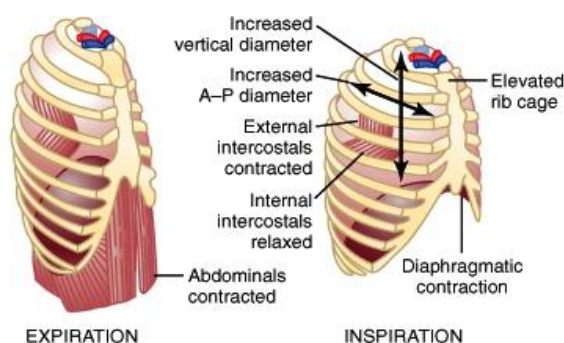




- What is the main function of the lung?
  - **Gas exchange is the main function which aims to provide oxygen for the tissues and get rid of CO<sub>2</sub>. This process is achieved by the following mechanisms:**
    - ✓ Lung mechanics (process of breathing).
    - ✓ Diffusion of O<sub>2</sub> and CO<sub>2</sub> across the alveolar-capillary membrane.
    - ✓ Transport of O<sub>2</sub> and CO<sub>2</sub> in blood and body fluids to the cells.
    - ✓ Chemical and neural control of ventilation.

- Movements of rib cage with respiration:
  - **At rest/expiration:** ribs move backward and downward.
  - **With inspiration:** ribs move forward and upward increasing antero-posterior space of thoracic cavity and allowing lungs to expand and be filled with air. In addition, chest volume is also increased vertically during inspiration by contraction of diaphragm (downward movement).

Notice that expiration is a PASSIVE process achieved by the elastic recoil of lungs and chest wall.



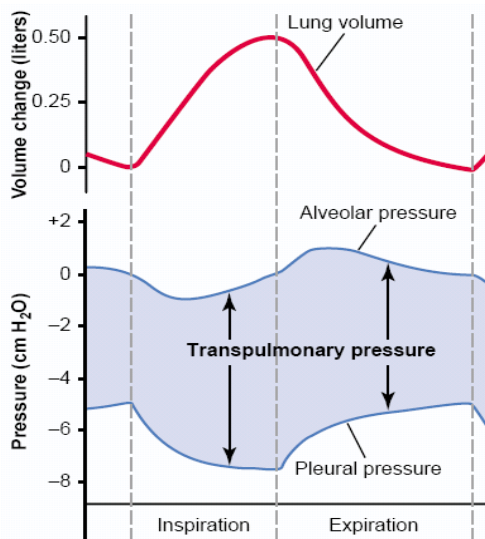
- Inspiratory and expiratory muscles:

Inspiratory muscles (increasing antero-posterior diameter of the chest by 20% with maximal inspiration)	Expiratory muscles (it is passive but with heavy exercise additional aid by some muscles is needed)
Diaphragm	Abdominal muscles (pull lower ribs down and compress abdomen upwards)
External intercostals muscles (elevate the rib cage)	Internal intercostal muscles (lower the rib cage)
Sternocleidomastoids (elevate the sternum)	
Anterior serrati (elevate several ribs)	
Scalenes (elevate first two ribs)	

- Pleural cavity:
  - Notice that pleura is composed of two layers (parietal and visceral pleurae) with a cavity between the two (pleural cavity) that is filled a fluid that has a mucoid character (allowing easy slippage of the moving lungs).
  - **This fluid is only few mls and if it increases** → the excess fluid will be pumped out by lymphatic thus creating a NEGATIVE intrapleural pressure (= -4 or -5 cmH<sub>2</sub>O).
- Pressures:
  - **Pleural pressure:** inspiration starts when chest expands and intrapleural pressure becomes more negative (reaching -7.5 cmH<sub>2</sub>O).
  - **Alveolar pressure:** at rest, alveolar pressure is atmospheric (= 0 cmH<sub>2</sub>O). For inspiration to occur, alveolar pressure must become negative (-1 cmH<sub>2</sub>O) and this is achieved by expansion of the chest wall. Therefore, a person can suck 500 ml of air in 2 seconds (this is known as the tidal volume). During expiration, alveolar pressure becomes positive (+1 cmH<sub>2</sub>O) to exhale that 500 ml of air in 1 second.
  - **Transpulmonary pressure (P<sub>L</sub>):** it represents the difference between alveolar pressure and pleural pressure (P<sub>L</sub> = P<sub>alv</sub> – P<sub>pl</sub>). It is simply a measurement of recoil pressure which drives elastic recoil of the lung at any instant.



- ✓  $P_L$  at beginning of inspiration =  $(0) - (-5) = +5$
- ✓  $P_L$  at beginning of expiration =  $(0) - (-7.5) = +7.5$  (there is a greater recoil pressure).



- **Therefore, breathing cycle occurs in the following order:**

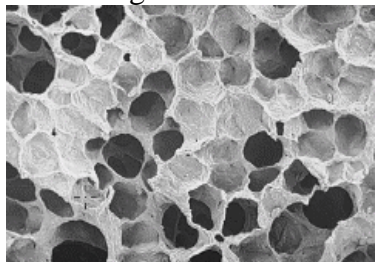
- Neural command to breath.
- Muscle contraction increasing chest volume.
- $P_{pl}$  becomes more negative (-7.5).
- Causes  $P_{alv}$  to become negative (-1).
- Air enters and  $P_{alv}$  increases (+1).
- Greater recoil (+7.5) causes air to exit.
- Cycle starts again.

- **Lung compliance:**

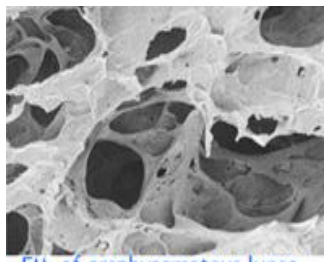
- **Definition:** it is the extent to which the lung expand for each unit change in transpulmonary pressure (200 ml of air per each cmH<sub>2</sub>O of change in transpulmonary pressure):

$$\checkmark \text{ Compliance (C)} = \frac{\Delta V}{\Delta P} = \frac{500}{2.5 \text{ or } 3} \cong 200 \text{ ml}$$

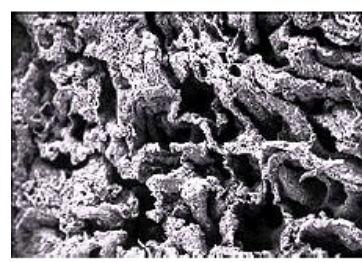
- Notice that the compliance of the combined lung-thorax system is almost one half of that of the lung alone.
- Process of respiration is composed of the following 3 components:
  - ✓ Compliance work (elastic work): during which you have to overcome elastic forces of lungs and tissues.
  - ✓ Tissue resistance work: in which you have to overcome viscosity of lungs and chest wall structures.
  - ✓ Airway resistance work.
- **Emphysema:** it is a component of Chronic Obstructive Pulmonary Disease (COPD) in which there is destruction of elastic tissue of lungs due to smoking. In this case, compliance increases as pressure needed for the same change in volume is less due to loss of elastic tissue.
- **Lung fibrosis:** in this condition there is increased elasticity due to fibrosis of lung tissues. Therefore, compliance decreases as pressure needed for same change in volume is greater.



EM of healthy lung tissue



EM of emphysematous lungs



EM of fibrotic lung tissue



- **Surfactant and surface tension:**

- When water forms a surface layer with air (fluid-air interface), the surface H<sub>2</sub>O molecules have a strong attraction.
- In alveoli, H<sub>2</sub>O attracts other H<sub>2</sub>O molecules and they force out air and attempt to collapse the alveolus.
- **Therefore, there are 2 elastic forces affecting the air-filled lung:**
  - ✓ Tissue elastic forces (elastin and collagen) tending to collapse the air-filled lung is only about (1/3 of total lung elasticity), while the surface tension forces in alveoli represent (2/3 of total lung elasticity).
- **Now you know why inspiration is an ACTIVE process because it has to overcome these to elastic forces.**
- **Notice that the smaller the alveoli the more is the surface tension (Pressure =  $\frac{2 \times \text{surface tension}}{\text{radius}}$ )**
- **Surfactant:** it is secreted by type-II alveolar cells and functions in decreasing the surface tension (which normally tends to collapse the alveoli). In premature babies, decreased synthesis of surfactant results in respiratory distress syndrome.

- **Important calculations:**

- **Minute respiratory volume = tidal volume x respiratory rate**
  - ✓ Tidal volume = 500 ml.
  - ✓ Respiratory rate = 12 breaths/minute.
  - ✓ Minute respiratory volume = 6 Liters.
- **Alveolar ventilation = Respiratory rate x (tidal volume – dead space)**
  - ✓ Normal dead space volume = 150 ml.
  - ✓ Alveolar ventilation = (12) x (500-150) = 4200
  - ✓ Dead space is divided into:
    - ❖ *Anatomic dead space:* in airway passages where there is no gas exchange.
    - ❖ *Physiologic dead space:* it is the space where there is no gas exchange due to impairment of ventilation/perfusion (usually there is a defect in blood flow).

- **Relation between respiratory air passages and Autonomic Nervous System (ANS):**

- **Sympathetic nervous system:** epinephrine and NE stimulate → β-adrenergic receptors thus resulting in → bronchodilation and vasoconstriction. Notice that few sympathetic nerve fibers reaches the lung, so that the effect of sympathetic nerve fibers stimulation is weak.
- **Parasympathetic nervous system:** vagus nerve (X) causes → bronchoconstriction and vasodilation.
- **Bronchoconstriction can be caused by:**
  - ✓ Irritants (nicotine and noxious gases (الغازات الضارة)).
  - ✓ Local lung factors: such as histamine which is released by mast cells when there is irritation resulting in allergic reaction.

- **Cartilage rings in upper airways keep them open (C-shaped cartilages). They disappear in bronchioles (less than 1.5 mm) so lung can expand easily.**

- **The greatest airway resistance is found in big bronchi near the trachea (because few of these bronchi are there compared to 65,000 parallel terminal bronchioles). Yet in disease conditions, these small bronchioles play greatest role in determining airflow resistance for 2 reasons:**

- **Because they are of small size, they can be easily occluded.**
- **They have greater percentage of smooth muscles so they constrict easily.**