Mechanics of ventilation: static lung mechanics

Specific learning objectives:

- Elastic properties of lung and chest wall
- Compliance: measure of elastic properties of lung

- *An isolated lung tends to contract until eventually all the contained air is expelled.*
- *When the thoracic cage is opened it tends to expand to a volume about 1 l greater than functional residual capacity (FRC).*
- *In a relaxed subject with an open airway and no air flowing, the inward elastic recoil of the lungs is exactly balanced by the outward recoil of the thoracic cage.*

Air flow in lungs

- No muscles in the alveoli; air passively moves in/out of the lungs in response to pressure gradients.

- **Forces are present that resist the opening of the lungs** i.e. the alveoli:
  - The natural tendency of the lungs to recoil or collapse: **elastic recoil**
  - **The surface tension in alveoli**

- **What keeps the alveoli (lungs) expanded:**
  - Negative intra-pleural pressure
    - The space between the two pleural layers is always negative or sub-atmospheric. This tends to suck the lungs outward
  - Alveolar pressure
    - pressure within the alveoli themselves tend to keep the lungs inflated
    - Reduced surface tension within the alveoli

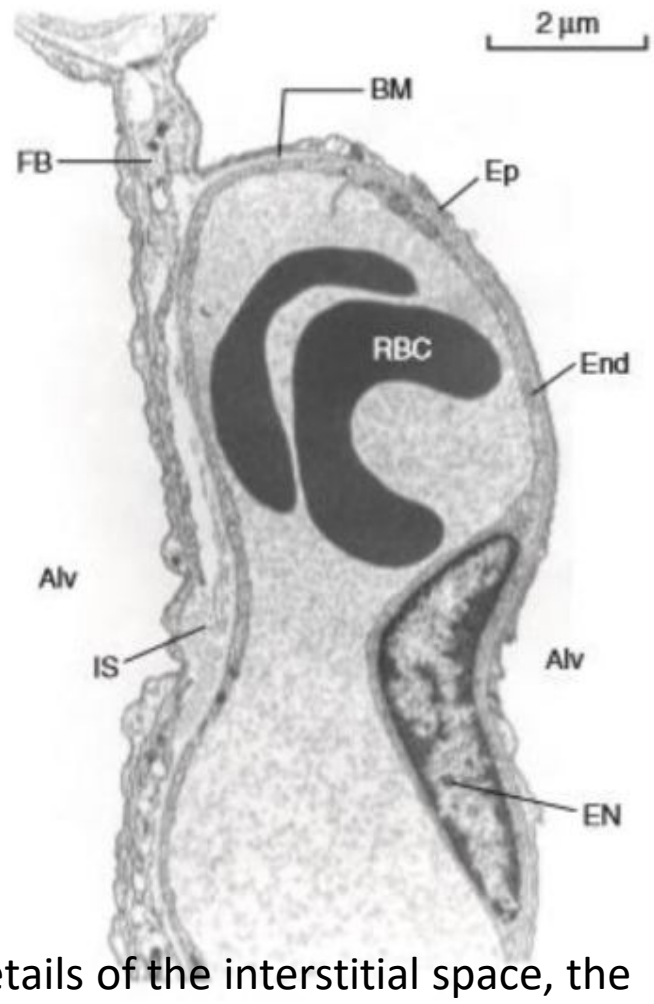
Why does an inflated lung want to recoil inward?

- Elastic recoil is the tendency to resist or oppose stretching.
- Lung volumes are determined by the balance between the lung's elastic recoil properties and the properties of the muscles of chest wall.
- When chest is opened, the lung recoils until the transpulmonary pressure is zero and the chest wall increases in size (60-70% of the vital capacity).
- Elastic recoil of the lung is directly related to lung stiffness, i.e., the stiffer the lung, the more elastic recoil.
- Elastic recoil pressure increases as the lung inflates.
- **The outward elastic recoil of the chest wall is greatest at residual volume, whereas the inward elastic recoil of the lung is greatest at** total lung capacity

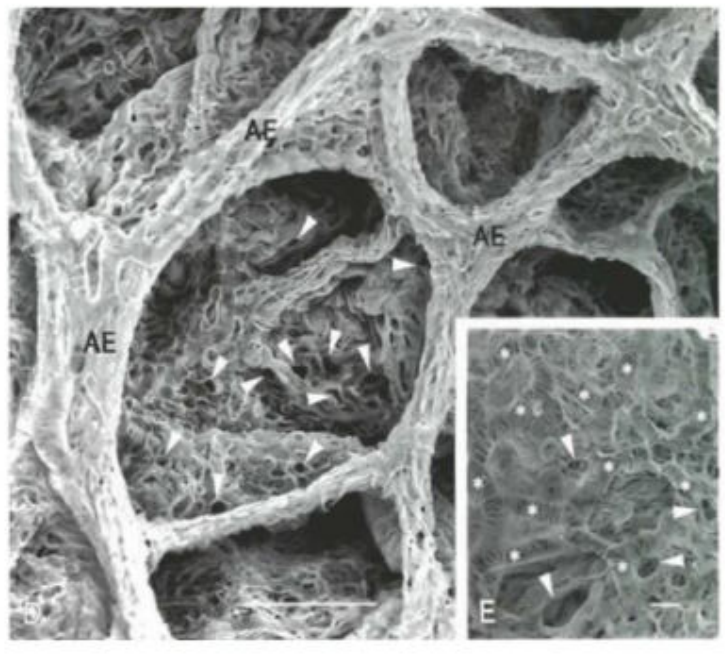
The framework of the lungs

- is made up of bundles of **elastic and collagen (type I,II,III,V,VI)** fibres that extend from the large airways down to the alveoli and across to the pleura and blood vessels.

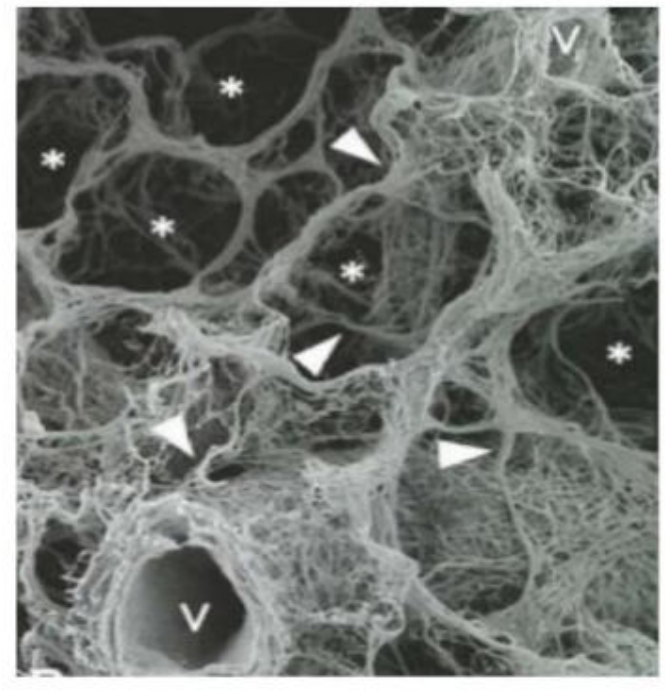
- Fibres are frequently in apposition and together they form the scaffolding of the lung resembling crumpled **wirenetting.**
- The connective tissue scaffold which forms the lung structure has three interconnected types of fibre: 1. **Axial spiral** fibres running from the hilum along the length of the airways 2. **Peripheral fibres** originating in the visceral pleura and spreading inwards into the lung tissue,3. **Septal fibres**, a network of which forms a basket-like structure of alveolar septa, through which are threaded the pulmonary capillaries, which are themselves a network
- Geometry: **nylon stocking.**
- Expansion of the lungs affects mainly the lung parenchyma. During an inspiration that starts from residual volume, the coils of the spiral fibres of the **alveolar ducts expand longitudinally; this enlarges the mouths of the alveoli that lie between the coils.**
- This stretches the alveolar septa, smoothens the undulations in alveolar walls, opens up of pleats in the septa and recruit previously collapsed alveoli.



Details of the interstitial space, the capillary endothelium and alveolar epithelium.



Electron micrographs of the collagen fibre network of rat lung



Electron micrographs of the elastin fibre In the rat lung

Elastic recoil: static recoil pressure

- **During inspiration**, contraction of respiratory muscles stretches the elastic and collagen tissues network of the lungs and pleura; it also overcomes the surface tension.
- The work that is done in stretching the lung is not dissipated as heat; instead the energy is stored in the structures, which have been stretched, then expended to drive the subsequent expiration.
- The static recoil pressure varies with lung volume .
- The slope of the relationship between pressure (Pst) and volume (V) describes the **distensibility** of the lungs. The slope at functional residual capacity is called the compliance of the lungs.

Elastic recoil of the lung affects compliance

The more elastic something is, the more it wants to return to its original shape
The more compliance something is, the less it wants to return to its original shape.

Lung compliance is defined as the change in lung volume per unit change in transpulmonary pressure Gradient.

- **Stiff lungs have a low compliance.**

Compliance is usually expressed in litres (or millilitres) per kilopascal (or centimetres of water) with a normal value of 1.5 $l.kPa^{-1}$ (150 $ml.cmH_2O^{-1}$).

Compliance may be described as static or dynamic depending on the method of measurement.

:Static compliance is measured after the lungs have been held at a fixed volume for as long as is practicable

:Dynamic compliance is usually measured in the course of normal rhythmic breathing.

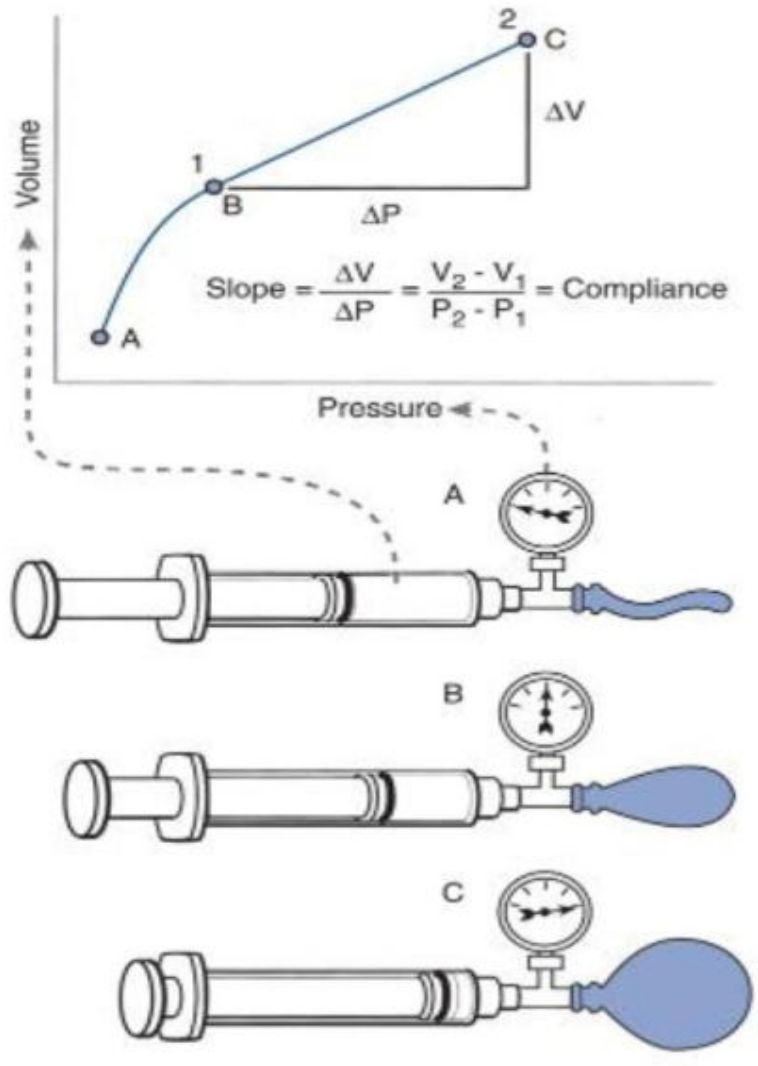
Compliance of the Lungs

- 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.**
- **It is a measure of elastic properties of the lung**
- **Reflects distensibility of lung**

- Compliance : change in volume per unit of pressure change:
- **C_L = ΔV (liters)/ΔP (cmH_2O)**

- The total compliance of both lungs together in the normal adult human averages about **200 milliliters of air per centimeter of water** transpulmonary pressure.

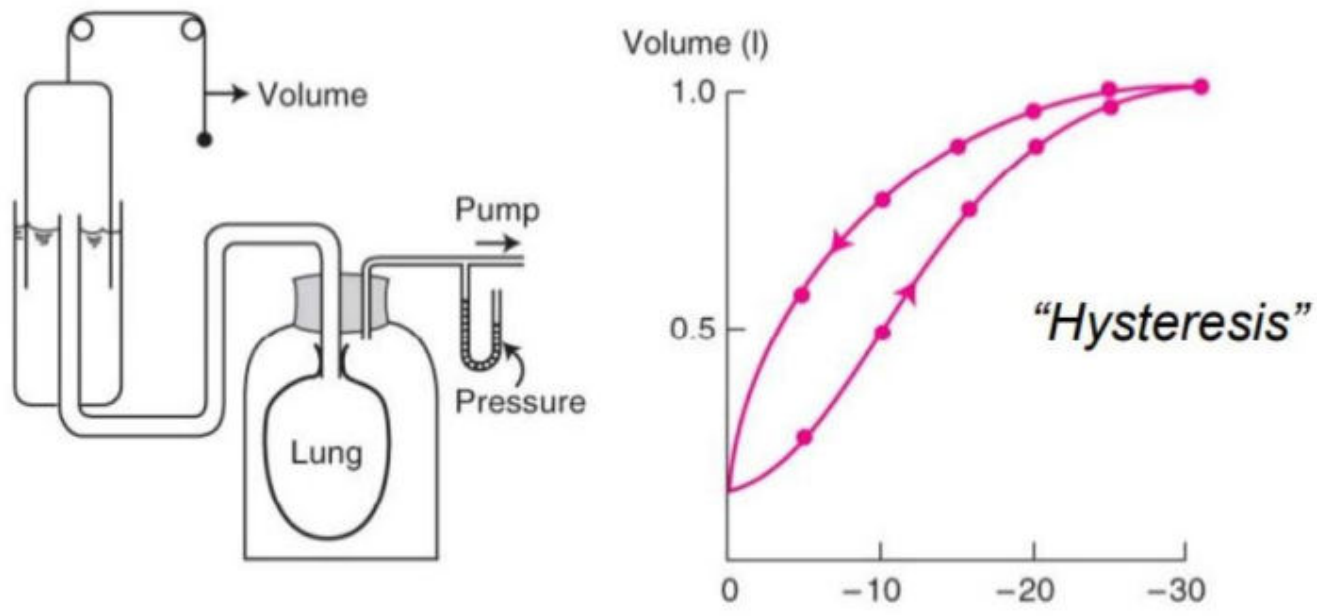
- That is, every time the transpulmonary pressure increases 1 centimeter of water, the lung volume, after 10 to 20 seconds, will expand 200 milliliters.
- To inspire a normal tidal volume of 500 ml, intrapleural pressure must fall by 2-3 cm H_2O



If the lungs are slowly inflated and then slowly deflated, the pressure/volume curve for static points during inflation differs from that obtained during deflation. The two curves form a loop, which becomes progressively broader as the tidal volume is increased.

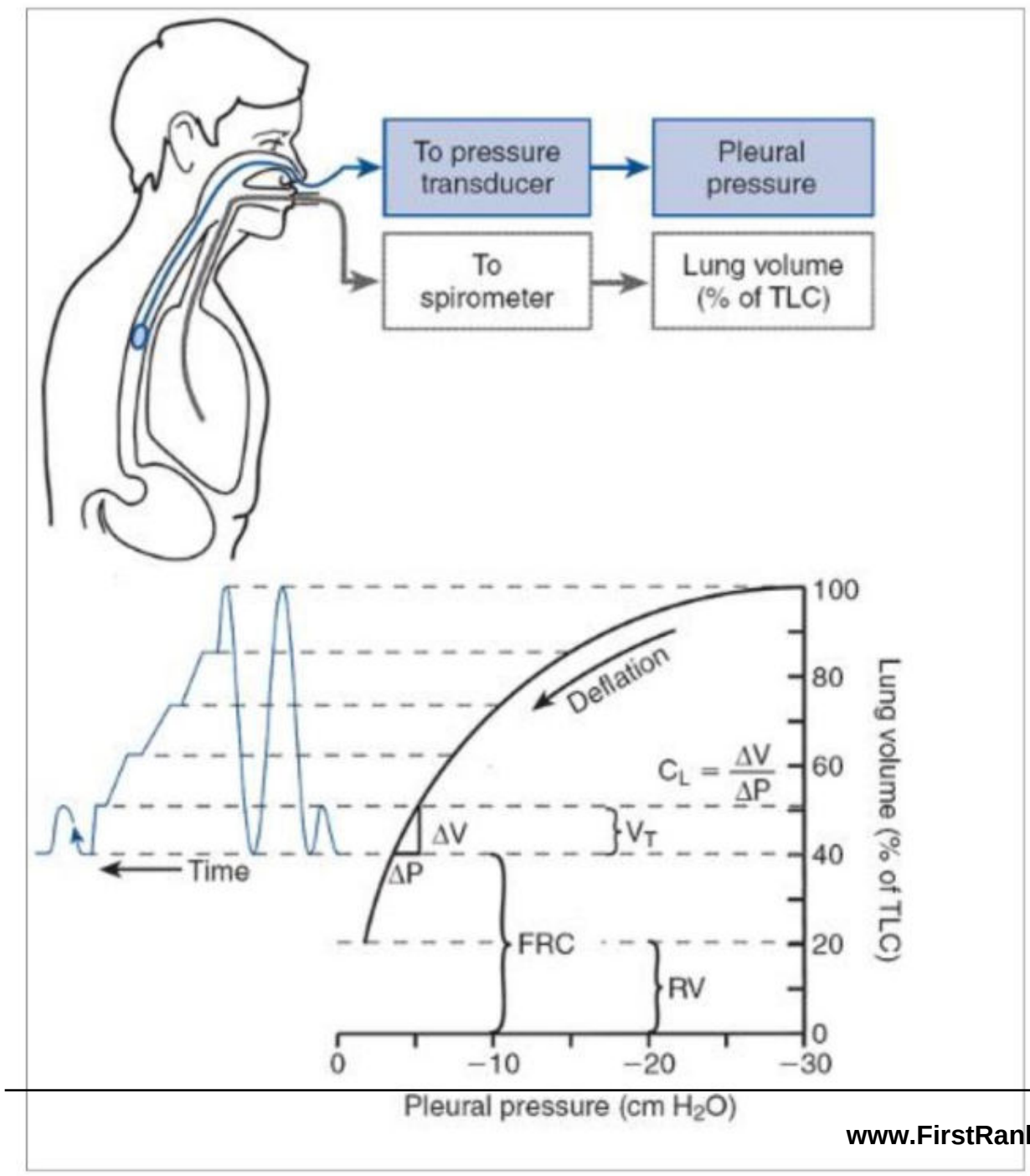
Rodney A. Rhoades, David R. Bell, **Medical Physiology: Principles for Clinical Medicine 3rd** Edition

$$\text{Compliance} = \frac{1}{\text{Elastance}} = \frac{\Delta\text{volume}}{\Delta\text{pressure}}$$



- As the pressure is reduced in steps, the volume increases.
- After the expanding pressure exceeds about 20 cm H_2O, the volume changes are less.
- Lung is much stiffer at higher volumes.

Nonlinear curve; slope of the P-V curve is considered i.e. **change in pressure over the litre above FRC on the descending limb of the curve**



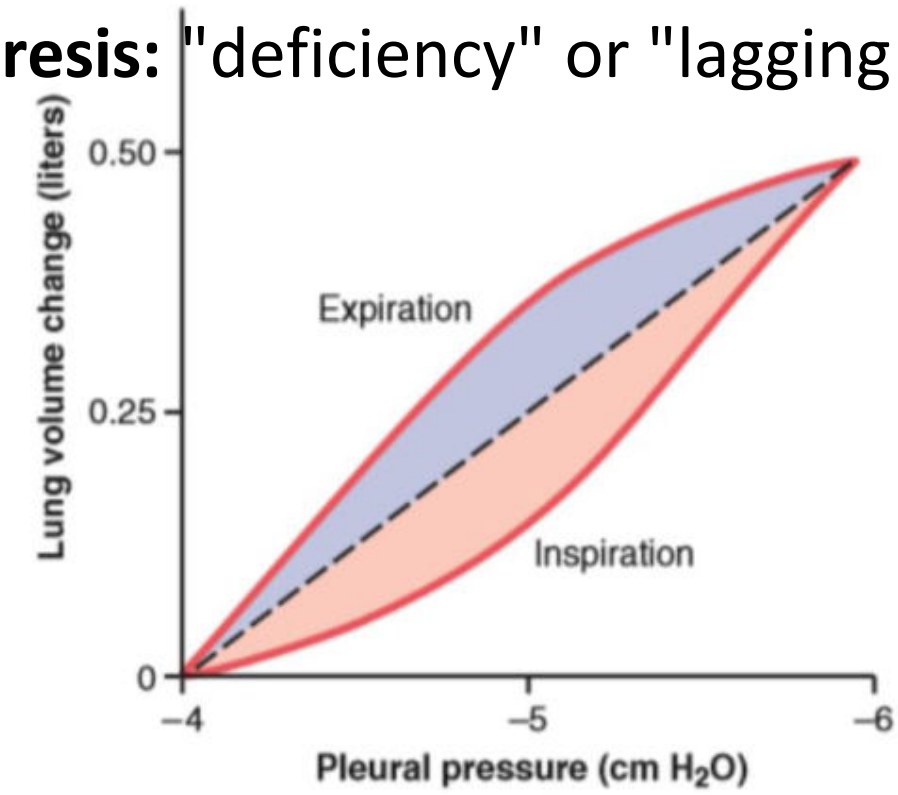
Lung compliance is measured from a pressure /volume curve.

The subject first inspires maximally to total lung capacity (TLC) and then expires slowly, while airflow is periodically stopped to simultaneously measure pleural pressure and lung volume.

Lung compliance (CL) is measured in L/cm H_2O.

Rodney A. Rhoades, David R. Bell, **Medical Physiology: Principles for Clinical Medicine 3rd** Edition

Hysteresis: "deficiency" or "lagging behind"

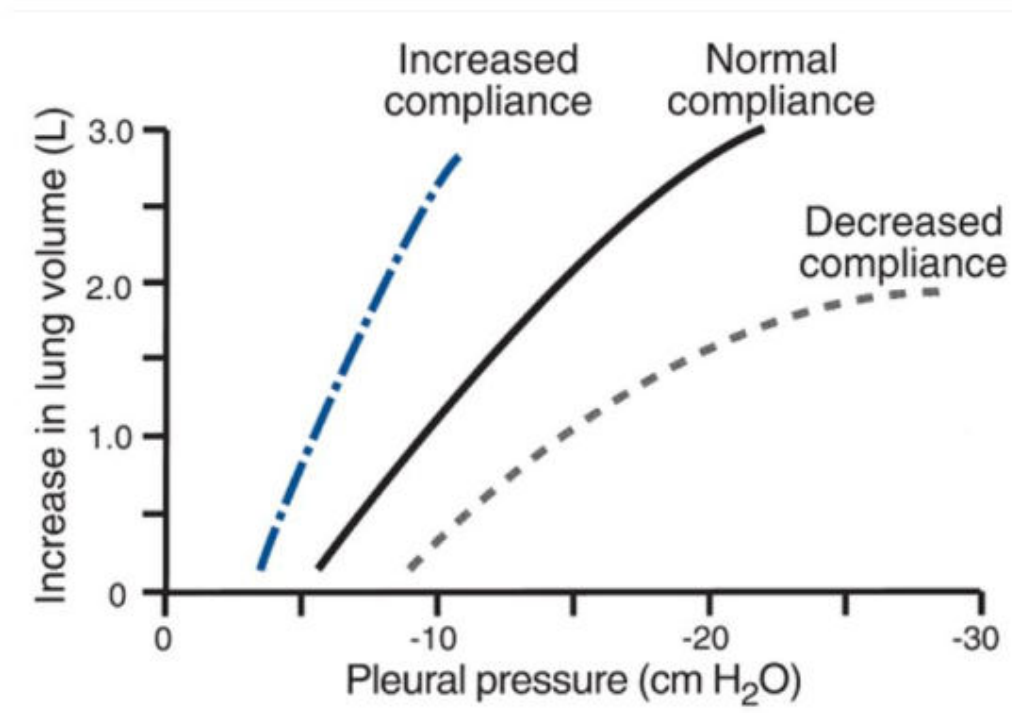


- At any given pressure, the volume on the descending limb of the pressure-volume curve exceeds that obtained while the lung was being expanded. The failure of the lung to follow the same course during deflation as it did during inflation is called hysteresis.

A more than the expected pressure is required during inflation and rather less than the expected recoil pressure is available during deflation. This resembles the behaviour of perished rubber or polyvinyl chloride, both of which are reluctant to accept deformation under stress but, once deformed, are again reluctant to assume their original shape.

The most important cause of the observed hysteresis in the intact lung : The surface tension of the alveolar lining fluid is greater at larger lung volume and also during inspiration than at the same lung volume during expiration.

Compliance in diseases



Patients with a chronic obstructive lung disease (COPD), such as emphysema, have abnormally high lung compliance.

Patients with restrictive diseases, such as respiratory distress syndrome, have abnormally low lung compliance.

Specific compliance

- Lung volume depends on the **body size** [(mouse v/s elephant) and if a person has one lung removed surgically] Therefore absolute values of compliance can not be used to compare lung compliance of different sized individuals.
- Compliance that has been adjusted for different lung volumes is called specific compliance.
- **Specific compliance** is change in volume per unit change in pressure/ initial volume.

- **Specific compliance** = Compliance divided by FRC
- Normal value = 0.8 (range 0.3 to 1.4) kPa^{-1} (0.08, range 0.03 to 0.14 cm H_2O^{-1}).
- Similar values in both sexes and all ages including neonates
- Is a measurement of the intrinsic elastic property of the lung tissue

Pressure/volume relationships of the lung plus thoracic age

- **Compliance** is analogous to electrical capacitance, and in the respiratory system the compliance of lungs and thoracic cage are in series.
- Therefore the total compliance of the system obeys the same relationship as that for capacitances in series

  1/Total compliance= 1/ Lung compliance+ 1/ thoracic compliance

- For its reciprocal, **elastance**, the relationship is then much simpler:
- **Total elastance**= lung elastance + thoracic cage elastance

Factors Affecting Lung Compliance

- ***Lung volume***. Larger animal species have thicker alveolar septa containing increased amounts of collagen and elastin resulting in larger alveolar diameters, so reducing the pressure needed to expand them. An elephant therefore has larger alveoli and higher compliance than a mouse.
- ***Posture***. Lung volume and compliance, changes with posture. Compared with the supine position, thoracic cage compliance is 30% greater in the seated subject and the total static compliance of the respiratory system is reduced by 60% in the prone position because of the diminished elasticity of the ribcage and diaphragm when prone.

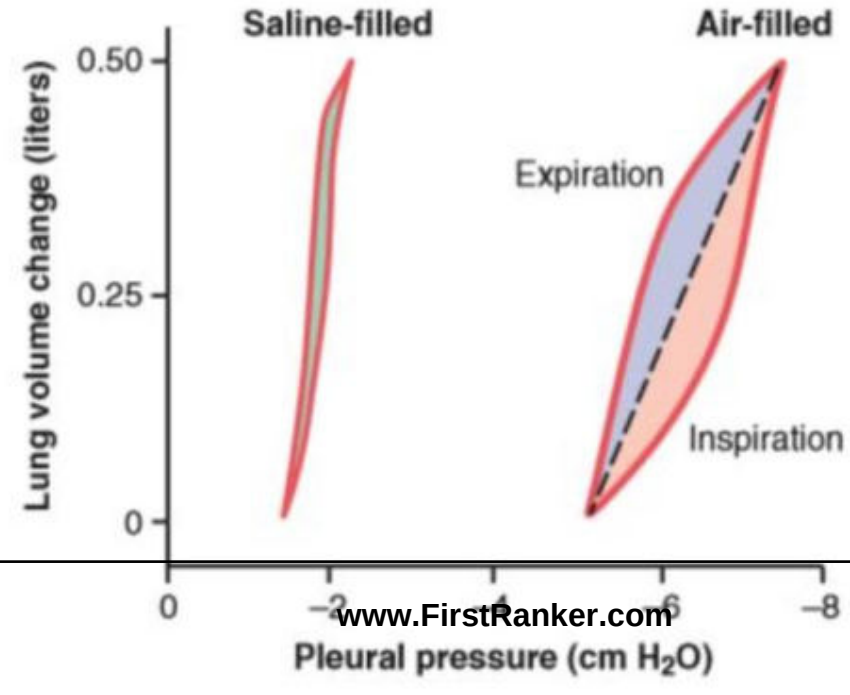
- ***Pulmonary blood volume***. The pulmonary blood vessels probably make an appreciable contribution to the stiffness of the lung. Pulmonary venous congestion is associated with reduced compliance.
- ***Age***. There is a small increase in lung compliance with increasing age, believed to be caused by changes to the structure of lung collagen and elastin.
- ***Bronchial smooth muscle tone***. *A*n infusion of methacholine sufficient to result in a doubling of airway resistance decreases dynamic compliance by 50%. The airways might contribute to overall compliance or bronchoconstriction could enhance time dependence and reduce dynamic compliance.
- ***Disease***. Important changes in lung pressure– volume relationships are found in some lung diseases

When the lungs are filled with air, there is an interface between the alveolar fluid and the air in the alveoli. In lungs filled with saline solution, there is no airfluid interface, and therefore, the surface tension effect is not present; only tissue elastic forces are operative in the lung filled with saline solution.

The transpleural pressures required to expand air filled lungs are about three times as great as those required to expand lungs filled with saline solution.

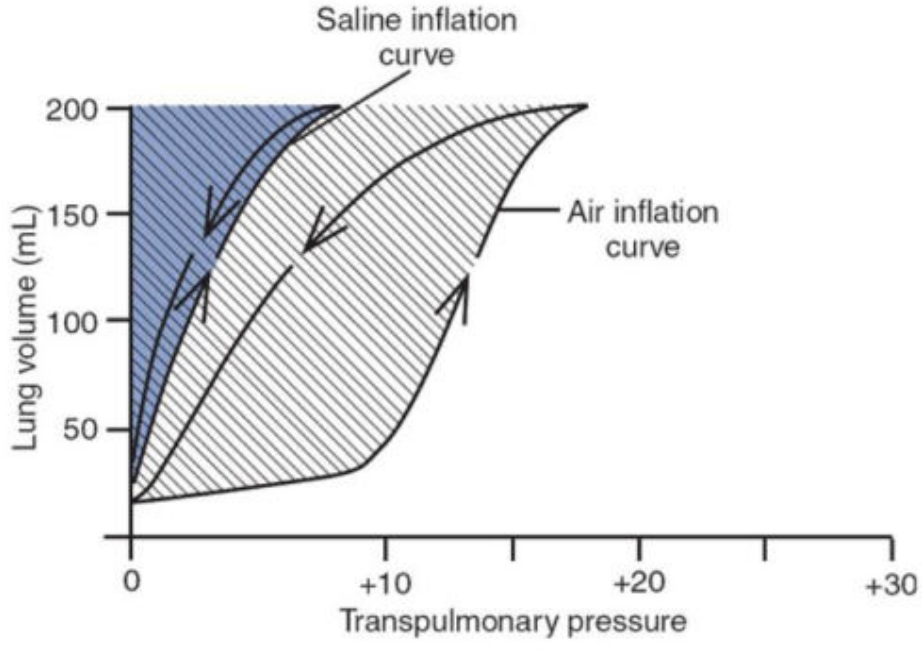
Thus, one can conclude that the tissue elastic forces tending to cause collapse of the air-filled lung represent only about one third of the total lung elasticity, whereas the fluid-air surface tension forces in the alveoli represent about two thirds.

The fluid -air surface tension elastic forces of the lungs also increase tremendously when the substance called surfactant is not present in the alveolar fluid.



Hysteresis is not present in the saline generated curves.

The difference between the saline and air curves is much smaller when lung volumes are small.



1. The slope of the deflation limb of the saline curve is much steeper than that of the air curve. This means that when surface tension is eliminated, the lung is far more compliant (more distensible).
2. The area to the left of each curve is equal to work, which can be defined as force (change in pressure) * distance (change in volume), the elastic forces and surface tension can be separated.

   The area to the left of the saline inflation curve: work required to overcome the elastic recoil of the lung tissue.

   The area to the left of the air inflation curve: work required to overcome both elastic tissue recoil and surface tension.

Area to the left of the air curve - Area to the left of the saline curve shows that approximately two thirds of the work required to inflate the lungs is needed to overcome surface tension.

Elastance

- Elastance is a measure of the work that has to be exerted by the muscles of inspiration to expand the lungs.
- **Elastance** is the **reciprocal** of compliance.
- It is the pressure change that is required to elicit a unit volume change and is expressed in kilopascals per litre.
- It is the collapsing force that develops in the lung as the lung expands.
- It always act to collapse the lungs. It is equivalent to collapsing force that builds up in a balloon completely inflated with air.
- Stiff lungs have a high elastance.
- Elastance is due to:

A. Collagen and elastic fibers within the lungs (minor component)

B. Surface tension of the alveoli (major component), Elastance is described by laplace law E=2T/R

What is Surface Tension?

- A molecular, cohesive (binding) force found at liquid-gas interfaces.
- Expressed in dynes/cm
- A liquid film that coats the interior of alveoli, causing air-liquid interfaces to assume a **spherical** shape.

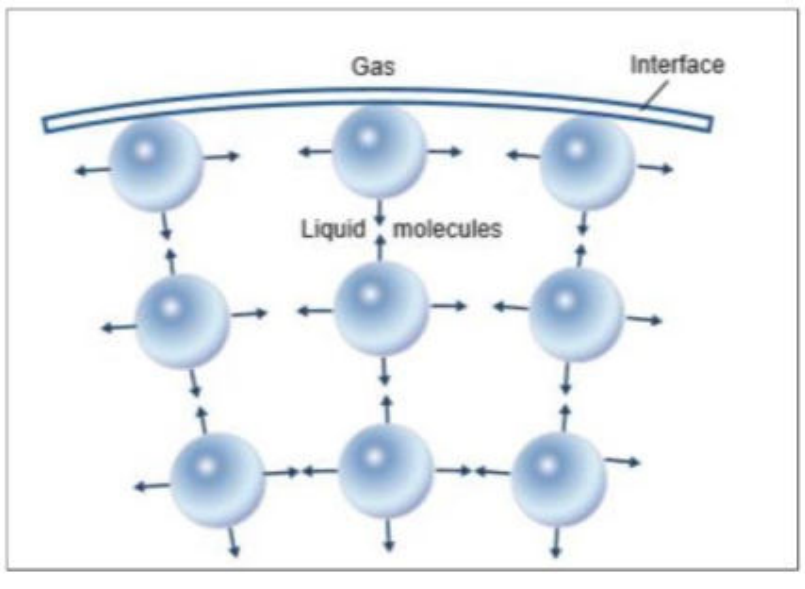
It is defined as the force acting across an imaginary line 1cm long in a liquid surface. This tension develops because the cohesive forces between adjacent liquid molecules are greater than the forces between the molecules of liquid and gas outside the surface.

Because of surface tension, the alveolus

- Resists stretching
- Recoils after stretching
- Favors reduced surface area (to shrink into a sphere)

- Surface tension at an air/water interface produces forces that tend to reduce the area of the interface.
- **The pressure inside a bubble is higher than the surrounding pressure by an amount depending on the surface tension of the liquid and the radius of curvature of the bubble according to the Laplace equation**: For a sphere like alveolus, the relationship between pressure with in the sphere and tension in the wall is given by **Laplace's Law**

- **P=2T/R**



The force of surface tension in a drop of liquid. Cohesive force attracts molecules inside the drop to one another. Cohesion can pull the outermost molecules inward only, creating a centrally directed force that tends to contract the liquid into a sphere.

~~Laplace's law~~

$$\text{pressure} = \frac{(2 \times \text{surface tension})}{\text{radius}}$$

- "The pressure inside a balloon is calculated by twice the surface tension, divided by the radius."

- Pressure to collapse generated by alveoli is inversely affected by radius of alveoli

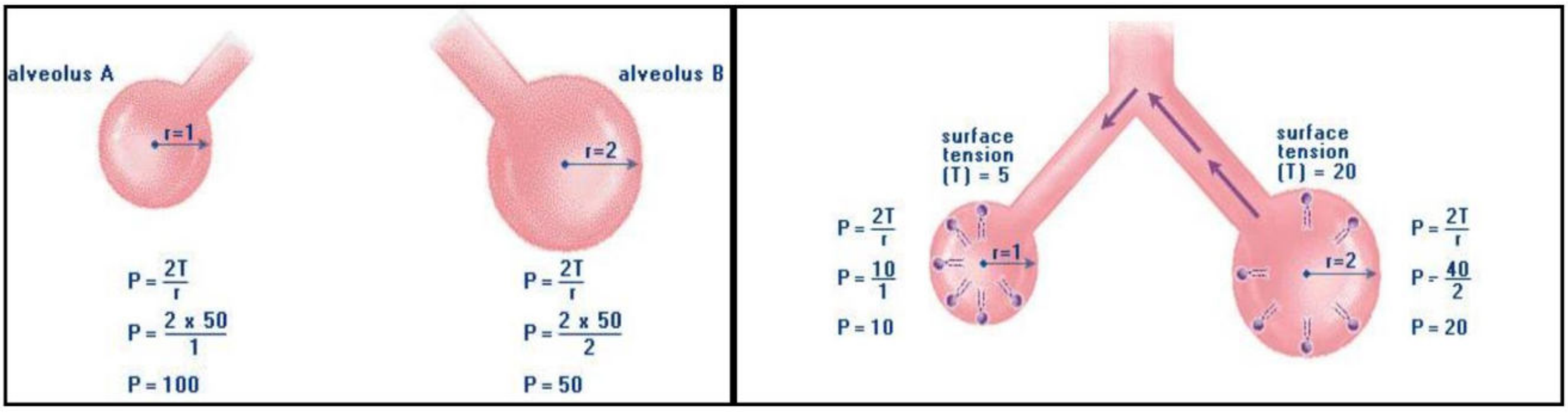
↑ Radius = ↓ Pressure to collapse

- The smaller a bubble, the higher the pressure acting on the bubble
- **Smaller alveoli have greater tendency to collapse**

If some alveoli were smaller and other large = smaller alveoli would tend to collapse and cause expansion of larger alveoli

That doesn't happen because:

- *Normally larger alveoli do not exist adjacent to small alveoli = because they share the same septal walls.*
- *All alveoli are surrounded by fibrous tissue septa that act as additional splints.*
- *Surfactant reduces surface tension =* ***as alveolus becomes smaller surfactant molecules are squeezed together increasing their concentration = reduces surface tension even more.***



Surfactant makes it possible for alveoli of different diameters that are connected in parallel to coexist and be stable at low lung volumes, by lowering surface tension proportionately more in the smaller alveoli

Effect of Alveolar Radius on the Pressure Caused by Surface Tension

The smaller the alveolus, the greater the alveolar pressure caused by the surface tension.

This phenomenon is especially significant in small premature babies, many of whom have alveoli with radii less than one quarter that of an adult person.

Further, surfactant does not normally begin to be secreted into the alveoli until between the sixth and seventh months of gestation, and in some cases, even later.

Therefore, many premature babies have little or no surfactant in the alveoli when they are born, and their lungs have an extreme tendency to collapse, sometimes as great as six to eight times that in a normal adult person.

This situation causes the condition called **respiratory distress syndrome of the newborn**.
It is fatal if not treated with strong measures, especially properly applied continuous positive pressure breathing.

The Alveolar Surfactant

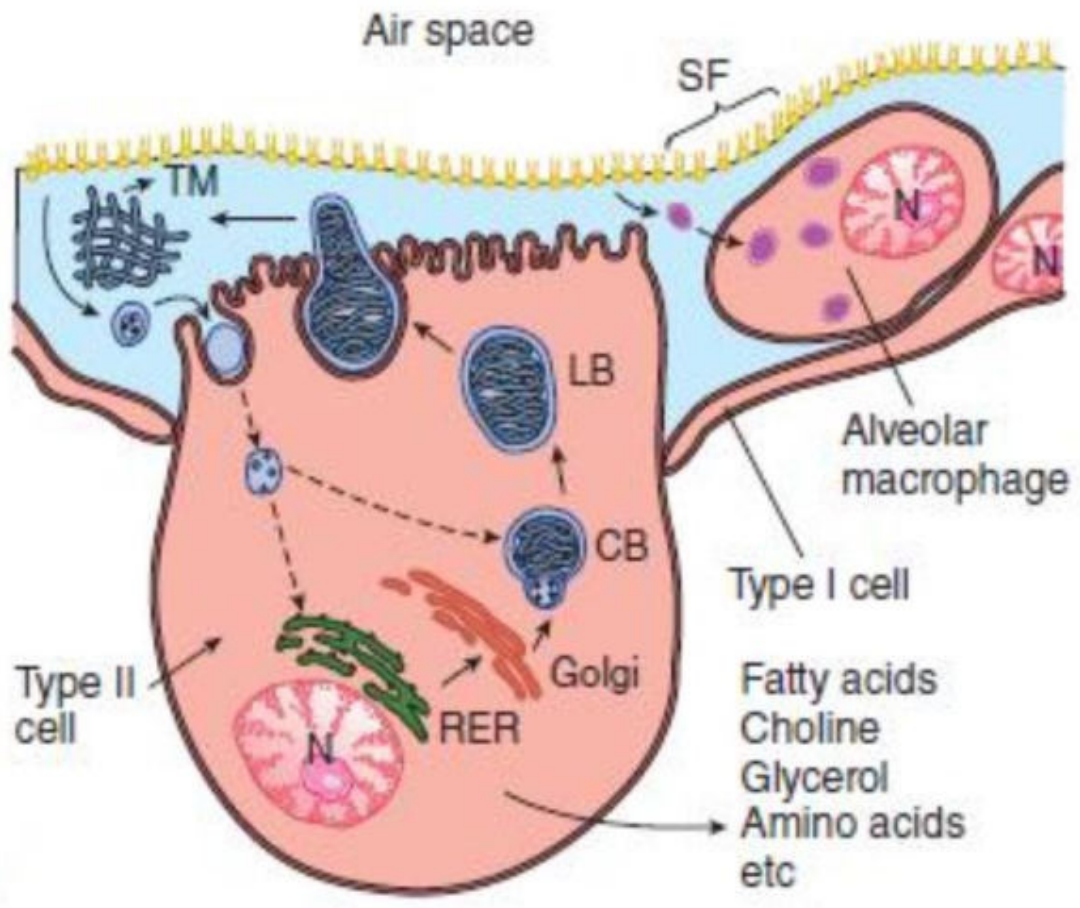
- It is a surface active agent in water, which means that it greatly reduces the surface tension of water. Approximately 90% of surfactant consists of lipids, and the remainder is proteins and small amounts of carbohydrate.
- Most of the lipid is phospholipid, of which 70% to 80% is dipalmitoyl phosphatidyl choline (DPPC), the main constituent responsible for the effect on surface tension.
- At low lung volumes, when the molecules are tightly compressed, some surfactant is squeezed out of the surface and forms micelles. On expansion (reinflation), new surfactant is required to form a new film that is spread on the alveolar surface lining.
- When surface area remains fairly constant **during quiet or shallow breathing**, the spreading of surfactant is often impaired.
- **A deep sigh or yawn** causes the lungs to inflate to a larger volume and new surfactant molecules spread onto the gas/ liquid interface.
- **Patients recovering from anesthesia are often encouraged to breathe** deeply to enhance the spreading of surfactant. Patients who have undergone abdominal or thoracic surgery often find it too painful to breathe deeply; poor surfactant spreading results, causing part of their lungs to become atelectatic.

Synthesis of Surfactant

- Both formed in and liberated from the alveolar epithelial type II cell.
- The lamellar bodies contain stored surfactant that is released into the alveolus by exocytosis in response to high volume lung inflation, increased ventilation rate or endocrine stimulation.
- After release, surfactant initially forms areas of a lattice structure termed tubular **myelin**, which is then reorganized into monolayered or multilayered surface films.
- The alveolar half-life of surfactant is 15 to 30 h with most of its components recycled by type II alveolar cells.
- They perform this function 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



- The released lamellar body (LB) material is converted to tubular myelin (TM), and the TM is the source of the phospholipid surface film (SF).
- Surfactant is taken up by endocytosis into alveolar macrophages and type II epithelial cells.

- Formation of the phospholipid film is greatly facilitated by the proteins in surfactant. This material contains four unique proteins: surfactant protein **(SP)-A, SP-B, SP-C and SP-D**.
- **SP-A** is a large glycoprotein. ***SP-A is intimately involved in controlling the surfactant present in the alveolus with type II alveolar cells having SP-A surface receptors, the stimulation of which exerts negative feedback on surfactant secretion and increases reuptake of surfactant components into the cell.***
- **SP-B and SP-C** are smaller proteins, which are the key protein members of the monomolecular film of surfactant, vital to the **stabilization** of the surfactant monolayer.
- A congenital lack of **SP-B** results in severe and progressive respiratory failure and genetic abnormalities of SP-C lead to pulmonary fibrosis in later life.
- Like SP-A, **SP-D** is a glycoprotein. It plays an important role in the organization of SP-B and SP-C into the surfactant layer.
- Both SP-A and SP-D are members of the collectin family of proteins that are involved in **innate immunity** in the conducting airway as well as in the alveoli, are involved in the control of surfactant release and in preventing pulmonary infection.

How does surfactant reduce the surface tension so much?

- The fatty acids are hydrophobic and generally straight, lying parallel to each other and projecting into the gas phase. The other end of the molecule is hydrophilic and lies within the alveolar lining fluid.

- When this occurs, their intermolecular repulsive forces oppose the normal attracting forces between the liquid surface molecules that are responsible for surface tension.

- The reduction in surface tension is greater when the film is compressed because the molecules of DPPC are then crowded closer together and repel each other more.

Physiological importance of surfactant

- A low surface tension in the alveoli increases the compliance of the lung and reduces the work of expanding it with each breath.
- Promotes alveolar stability and prevents collapsed at low volumes--small alveoli are prevented from getting smaller.
- Promotes “dry” alveoli--collapsed alveoli tend to draw fluid from pulmonary capillaries (edema-movement of fluid into alveoli; It has been calculated that if it were not present, the unopposed surface tension in the alveoli would produce a 20 m Hg force favoring transudation of fluid from the blood into the alveoli.

ELASTIC RECOIL OF THE THORACIC CAGE

- The thoracic cage comprises the ribcage and the diaphragm.
- Each is a muscular structure and can be considered as an elastic structure only when the muscles are relaxed, which is can be achieved only under the conditions of paralysis.

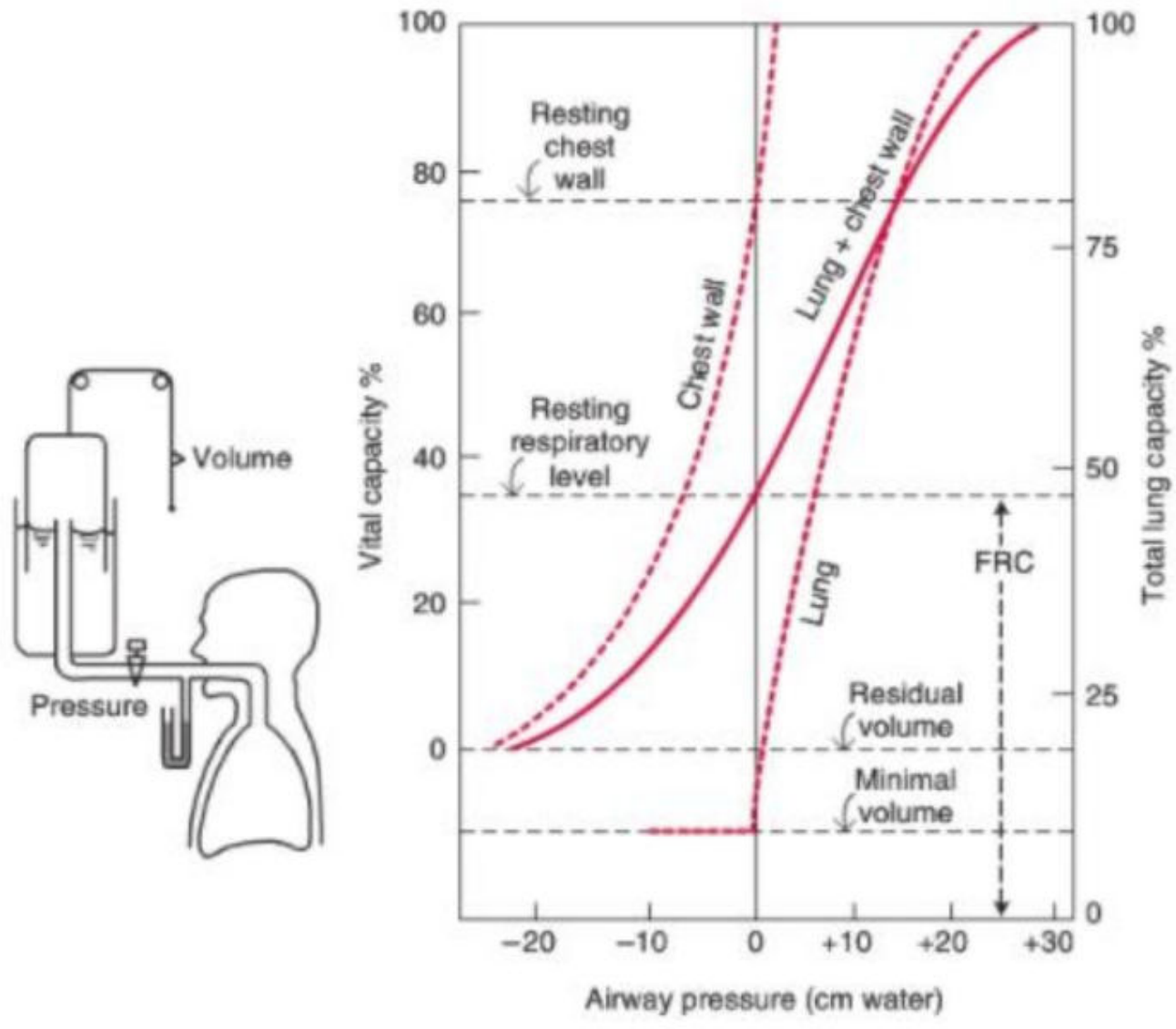
➢The compliance of lungs+thorax = 1/2 of lungs alone.

- To measure compliance, air is forced into the lungs a little at a time while recording lung pressures and volumes.

- The curve of airway pressure obtained in this way, plotted against volume, is the pressure–volume curve of the total respiratory system. The pressure is zero at a lung volume that corresponds to the volume of gas in the lungs at FRC (relaxation volume).

- This relaxation pressure is the sum of slightly negative pressure component from the chest wall (Pw) and a slightly positive pressure from the lungs (PL). PTR is positive at greater volumes and negative at smaller volumes.

Compliance of thorax and lung together



Relaxation pressure-volume curve for the total respiratory system

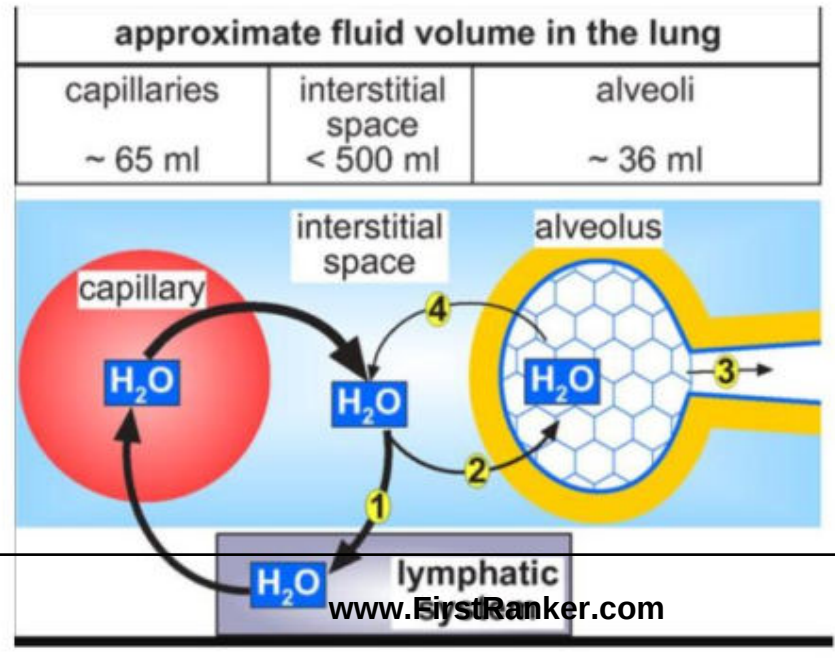
- The relaxation pressure of the total system is zero at functional residual capacity (FRC).
- As the volume of the system is increased the relaxation pressure increases, and the opposite occurs as the volume is decreased.
- The relaxation pressure-volume curve of the lung shows a positive pressure of 4 mmHg at FRC, and this is balanced by an equal and opposite outward expanding pressure developed by the chest wall.
- The relaxation curve for the chest wall alone crosses the zero pressure line at 70% of vital capacity. In other words below this volume the chest wall continues to develop a negative pressure, that is tends to expand, whereas above that volume a positive pressure is required to expand the chest wall

Factors Influencing Compliance of the Thoracic Cage

- Anatomical factors include the ribs and the state of ossification of the costal cartilages, which explains the progressive reduction in chest wall compliance with increasing age.
- Obesity and even pathological skin conditions : scarring of the skin overlying the front of the chest, for example, from burns, may impair breathing.
- A relaxed diaphragm simply transmits pressure from the abdomen that may be increased in obesity and abdominal distension.
- Compared with the supine position, thoracic cage compliance is 30% greater in the seated subject and the total static compliance of the respiratory system is reduced by 60% in the prone position because of the diminished elasticity of the ribcage and diaphragm when prone.

Alveolar lining fluid

- The intermediate surface area of the human lung is 130 $m^{2.}$ and is mostly constituted by the alveolar region. The average fluid surface height in the alveoli is 0.2 µm.
- The alveolar fluid content can be estimated to be ~36 ml.
- A 70-kg human needs ~2900 ml of water per day. ~725 ml/day of this water will be lost due to respiration.
- This indicates that the alveolar fluid volume is replaced ~20 times/day.
- Water loss due to respiration and its replacement from the internal water body content is a dynamic process that must be tightly regulated.



Front. Physiol., 22 May 2012

Alveolar interdependence