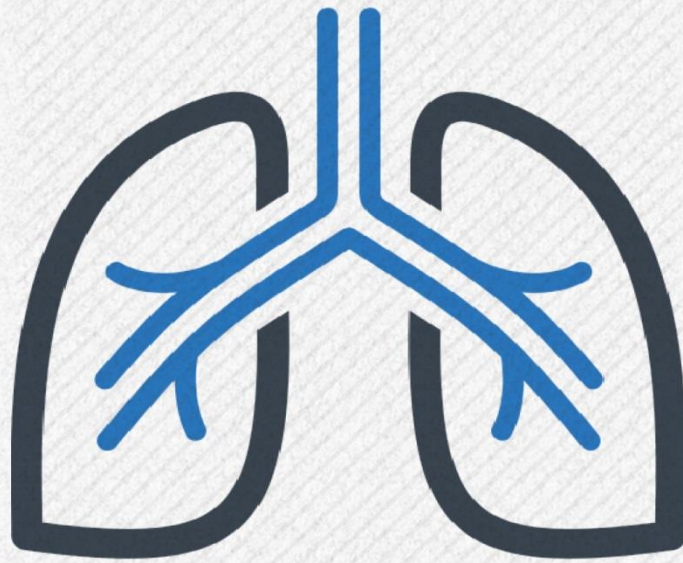




Respiratory system physiology



sheet



handout



slides

Number

5

Doctor

Yanal

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Before we start....

- This sheet is written according to section 2 recording.
 - The order of ideas is a little bit different from that in the recording.
 - You will find some additional information and details, trust me it's all for your benefits (I hope so ... 😊)
 - I put some information from last year's sheets, slides and Costanzo book fifth edition pages (194- 200).
-

Our topic for this sheet will be about **Lung compliance.....**

We learnt that the lung can be thought of as an elastic balloon, so what do we mean by elastic?

Lung Elasticity is the ability of the lung to recoil and come back to its resting state or resting volume when it's inflated, that means the lung has a recoil tendency.

The recoil collapsing tendency of the lung equals +4 mmHg (at FRC), we have an intrapleural pressure which is equal to it but in the opposite direction (-4 mmHg) that prevents the lung from collapsing.

****Let's talk about intrapleural pressure...**

The intrapleural space normally has negative pressure (less than atmospheric)

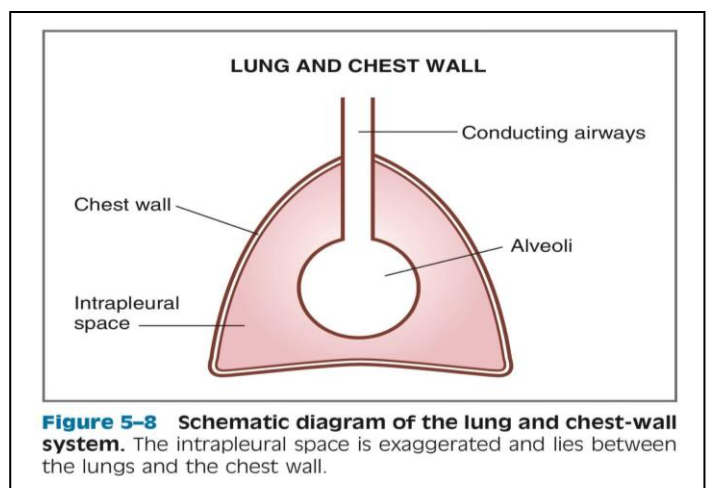
- this pressure is created by two opposing elastic forces pulling on the intrapleural space which are:

- 1- The lungs with their elastic properties, tend to collapse.
- 2- The chest wall with its elastic properties, tend to spring out.

When these two opposing forces pull on the intrapleural space, this negative intrapleural pressure is produced (P_{ip}).

- In turn this negative intrapleural pressure opposes the natural tendency of the lung to collapse and chest wall to spring out.

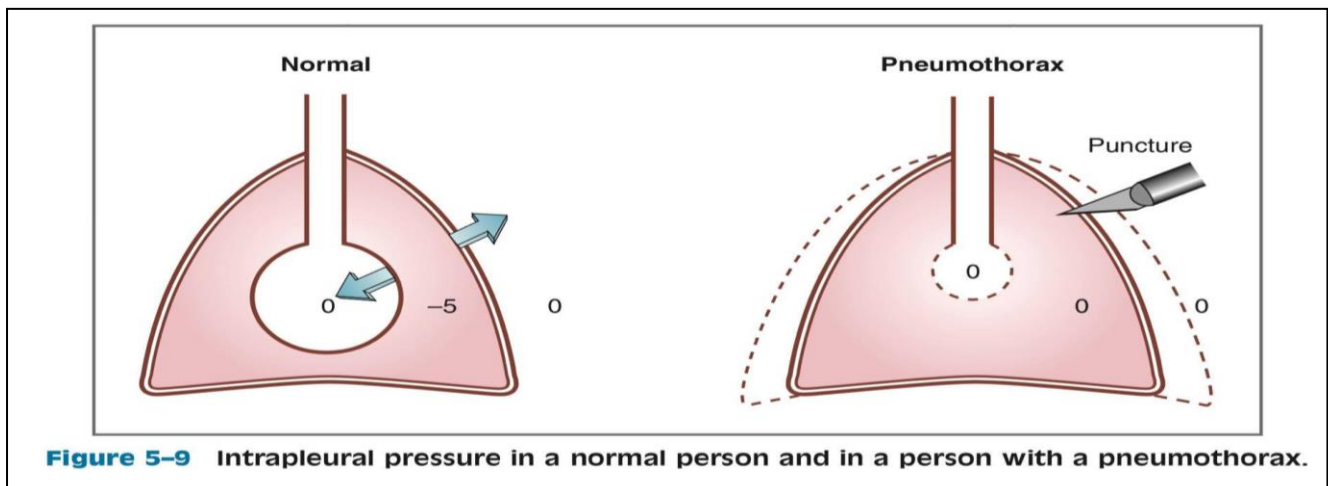
- So you must know by now that P_{ip} , Prevents the lungs from collapsing and the chest wall from Spreading out.



***So** what will happen if we remove this negative intrapleural pressure ?

Like when a sharp object punctures the intrapleural space, this space is introduced to the atmospheric air & is abnormally filled with air (this is called pneumothorax), what will happen is that the lungs tend to collapse to their **minimal volume**, (intrapleural pressure becomes equal to the atmospheric pressure =0 mmHg)

In easier words, the lungs tend to collapse like a balloon and the chest wall tends to spring out, it's got all these little muscles on it trying to pull it away from the center of the body.



If you still have trouble picturing why the chest would want to spring out, think of the chest wall as a spring that you normally contain by compressing it between your fingers. Of course the real chest wall is "contained" by the negative intrapleural pressure, rather than the force of your fingers.

If you release your fingers or eliminate the negative intrapleural pressure, the spring or the chest wall springs out.

We mentioned something about resting volume, so what do we mean by it?

- It is also called the minimal volume which is the volume at which the structure doesn't tend to expand nor collapse.
- To change the resting volume you need to apply forces. (Although bringing the structure to its resting volume is a passive process)

- It equals 150ml air in the lung, (Remember: this is not anatomic dead space volume even though it is 150 ml !!), so at FRC the lung will be partially collapsed.
- It is also known as unstressed volume of the lung.
- Normally we don't reach the minimal volume, we could only reach the residual volume but not less than that, so lungs don't reach their resting state.
- MV(Minimal Volume) is used for medico legal purposes, How?

Let's assume that there is a newborn and he took his first breath, so there is MV in his lungs but unfortunately he died after a while, if we take his lungs and we put in a water pool, this lungs will float (because it contains MV) , while in another baby born dead (stillbirth), the baby didn't take a first breath and his lungs contain no air (not even the MV), so if we place his lungs in the water it will sink.

*So the absence or presence of MV allows us to know if the baby was born dead or if someone killed him after birth.

we said that we call the minimal volume by the unstressed volume, what do we mean by unstressed volume?

In the cardiovascular system there is also what we call unstressed volume, and since we have unstressed volume we also have stressed volume, let's explain them:

1-The unstressed volume is the volume of blood in the vasculature that produces no pressure (contained mostly in the veins), it's worth to know that if the vessels contain only the unstressed volume (abnormally), there will be no pressure in them and therefore no force or tension so no blood flow and the blood can't go back to the heart (no venous return & of course no cardiac output).

2- The stressed volume is the volume that produces pressure by stretching the elastic fibers in blood vessels walls (it's hold by the arteries), and the role of the sympathetic nervous system is to keep this volume.

** The total volume of blood in cardiovascular system is the sum of stressed and unstressed volume plus whatever contained in the heart.

Costanzo page 131

Follow up →

To calculate these volumes:

we know the veins contain 3L → 2.5L are the unstressed volume, we can measure it by taking an empty vein (no pressure in it) and then by filling it with blood until pressure begins to develop in the wall of the vessel (at that point we have reached the upper limit of the unstressed volume, and if we exceed that volume, stress (pressure) will develop in it).

→ 0.5L, it's the stressed volume.

The arteries contain 750 ml → 500 ml is the unstressed volume, pressure is zero.

→ 250 ml is the stressed volume.

** it's the same concept in the lungs.

** So what do we mean by compliance?

- It is the change in lung volume per unit change in transpulmonary pressure. (**compliance = $\Delta V / \Delta P$**)

You have to be familiar with these concepts:

-intrpleural pressure (explained before)

- alveolar pressure: also called intra-pulmonary pressure, is the pressure of air inside the lung alveoli. When there is no air flowing into or out of the lungs, alveolar pressure is equal to atmospheric pressure (zero cmH₂O).

I recommend to you watching this animation (just to understand the concept) :

http://highered.mheducation.com/sites/0072507470/student_view0/chapter23/animation_alveolar_pressure_changes_during_inspiration_and_expiration.html

- Transmural pressure : → it's the pressure exerted across the wall, and it's the pressure that determines the inflation and deflation, it can be:

-zero → at rest / negative → deflation / positive → inflation

→ It's equal to the pressure inside minus pressure outside
= P inside – P outside

so if this transmural pressure was the difference between alveolar pressure and interpleural pressure it will be called **transpulmonary pressure.

**So transpulmonary pressure = P inside (alveolar) – P outside (interpleural)

COMPLIANCE is the ability of the lung to stretch.

- you must know that Inflating one balloon is easier than inflating two balloons, one inside the other, the two balloons are the lung and the thorax, so the compliance of the lungs in vivo (we inflate both lungs and thorax) is less than the compliance of the lungs in vitro (since we inflate the lungs only).

- also COMPLIANCE = $1/\text{ELASTANCE}$ (inverse relation)

-AN ELASTIC STRUCTURE MUST BE AUTOMATICALLY COMPLIANT. A COMPLIANT STRUCTURE NEEDS NOT TO BE ELASTIC. For the lung to be efficient compliant, it must return back to resting volume following inspiration, otherwise it keeps inflating. GUM is compliant but not elastic and a RUBBER BAND is compliant and elastic.

Specific compliance = C/FRC to correct for differences in lung volume between a child and an adult.

$C_L = 200\text{ml/cm H}_2\text{O}$. For the lung alone

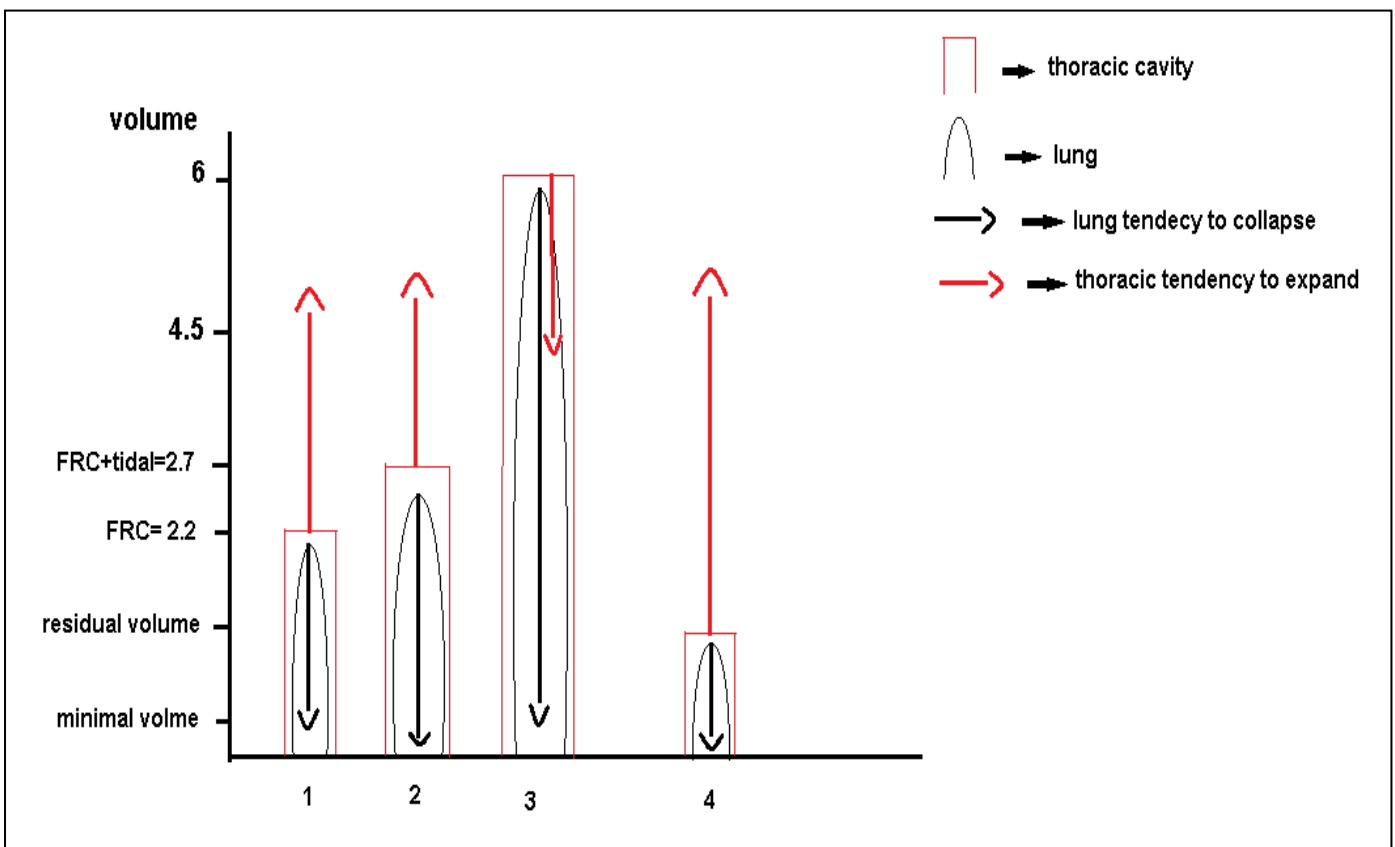
$C_W = 200\text{ml/cm H}_2\text{O}$. For the thoracic wall alone

$C_S = 100\text{ml/cm H}_2\text{O}$. S stands for lung-thorax system (For both)

From slides

The resting volume of the thorax is 4.5L, so at FRC the thorax will be compressed (FRC= 2.2 L)

** So we conclude that (at FRC) the Lung tends to collapse. In contrast, thoracic cavity (at FRC) tends to expand. Let's see the relation between these two tendencies and try to look at the thoracic cavity and lung as a whole system. Look carefully at this figure:



* **state 1:** When the lungs are full of functional residual capacity:

- a) The lung continuously tries to reach its resting volume (recoil tendency).
- b) The thorax also tries to reach its resting volume which equals 75% of TLC
 $= 75\% \times 6 = 4.5$ liters.

And these forces a+b are equal in value and opposite in direction, so the net force is Zero. Thus, the system (lung-thorax system) is at rest at a volume equal to FRC.

- so you should ,by now, be familiar with these terms:

- ❖ resting volume for the lungs equals 150 ml.
- ❖ resting volume for the thorax equals 4.5 L.
- ❖ resting volume for the lung-thorax system equals FRC= 2.2 L
(the system is at rest, remember the lungs don't reach its resting state)

* **state 2:** To overcome the resting state and since the lungs are elastic all I need is to apply force (active process), this force produced by adding the tidal volume to the FRC.

- So the recoil tendency of the lungs increases and the expanding tendency of the thorax decreases. The forces are still opposite but are no longer equal in magnitude.

The system tends to collapse passively.

That's another way to understand why expiration is considered a passive process (remember it doesn't need contraction of muscles in normal breathing).

* **state 3:** The recoil tendency of the lungs is huge, the thorax now has tendency to collapse, because its volume now is larger than its resting volume. Both structures are tending to collapse, and the system tends to collapse in a huge force.

Functional residual capacity (FRC): is the most important indicator and it is the volume of air present in the lungs just before inhaling tidal volume
 $FRC = (ERV + RV)$.

**Try to fill your lungs to the maximum (forceful inspiration), then close your mouth and nose and relax your muscles the air will go out غصباً عنك .

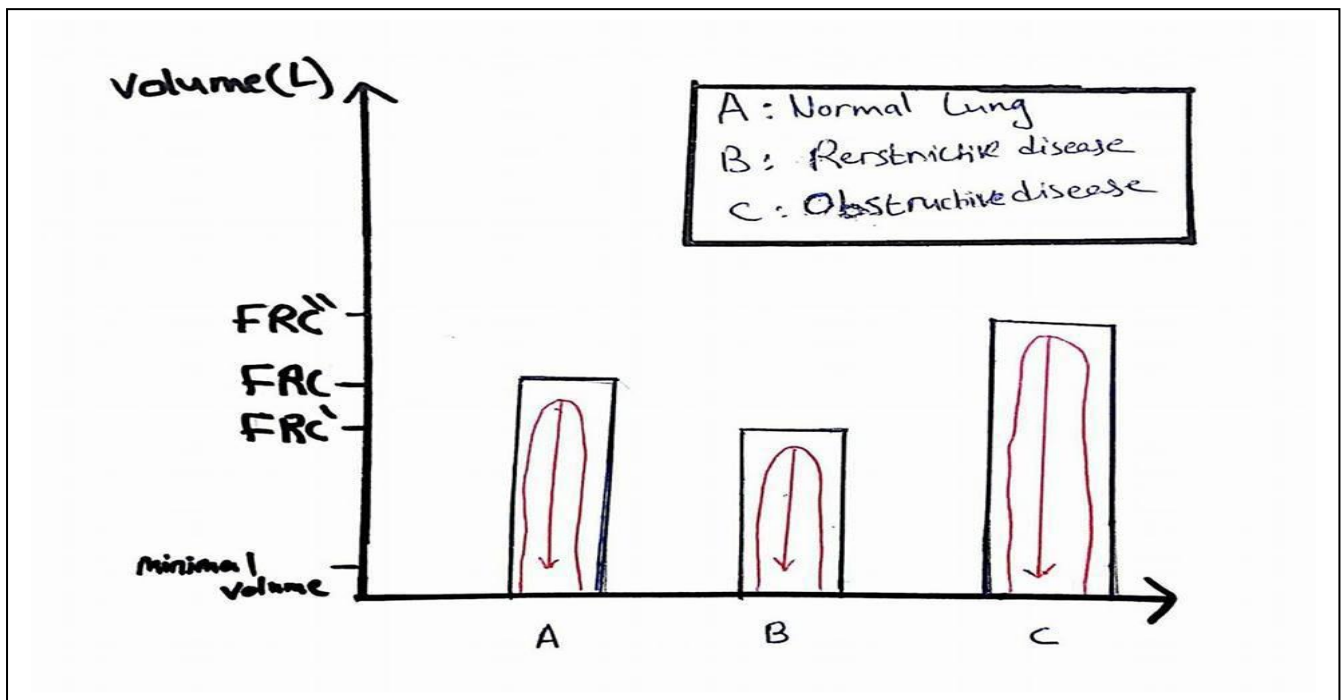
* **state 4:** In order to reach such a state you need to apply force to expire the expiratory reserve volume (ERV) using expiratory muscles, here the **expiration is active**.

- Notice the volume of the lungs here is the residual volume, the recoil tendency of the lung is decreased and the tendency of the thorax to expand is increased.

-**The system tends to expand**, so here the inspiration becomes a passive process.

(This doesn't happen normally in our lungs. inspiration is active and expiration is passive).

Note: We care mostly about the lung recoil capacity more than the thorax capacity to expand because thoracic abnormalities relating to its capacity are rare. (Although we may have deformities in the chest wall).



The figure above shows the following:

- A → Normal lung.
- B → In restrictive lung diseases like fibrosis, the collapsing tendency of the lung increases, our lungs adapt themselves by decreasing FRC to FRC' which results in decreasing their collapsing tendency and increasing the expanding force and thus keeping the system at rest (we reach a new equilibrium state).
- C → In obstructive lung diseases like emphysema, the compliance of lungs increases and the collapsing tendency decreases, our lungs adapt themselves by increasing FRC to FRC'' resulting in increasing their collapsing tendency and decreasing the expanding force and thus keeping the system at rest.

So to sum up, in clinical findings:

- if we have low FRC, this mean that we deal with restrictive disease (high collapsing force in the lungs).
- if we have high FRC, this mean that we deal with obstructive disease (low collapsing force in the lungs), like in emphysema where destruction of elastic fiber occur, so more compliance.

* The two extremes of anything are not good (الفضيلة تقع بين الرذيلتين)

This rule also applies in physiology.

- An example from the Respiratory System:

1- Too much compliance is bad:

In COPDs [like emphysema], the compliance is very high → not beneficial because it's difficult for the lungs to deflate.

Note: in COPDs, the Total Lung Capacity might reach 7- 7.5L (instead of the normal 6L)

2- Too little compliance is bad:

In restrictive lung diseases [like pulmonary fibrosis and RDS], the compliance is low → not beneficial because it is difficult for the patient to inflate his lungs.

Note: in restrictive lung diseases, TLC might only reach 4- 4.5L.

The forces that cause the recoil tendency of the lung:

We've said that the lung is elastic and has the tendency to recoil. What are the forces that cause this recoil tendency? We will start by discussing all the forces that we need to spend work on them during breathing:



❖ We spend work to breathe, so what do we mean by it?

We have already pointed out that during normal quiet breathing, all respiratory muscle contraction occurs during inspiration; expiration is almost entirely a passive process caused by elastic recoil of the lung and chest cage. Thus, under resting conditions, the respiratory muscles normally perform 'work' to cause inspiration but not expiration.

- the work is equal to the change in volume times the change in pressure.

$$W = \Delta V * \Delta P \rightarrow \Delta V : \text{Tidal volume} \quad / \quad \Delta P : \text{amount of pressure needed}$$

Energy required for inspiration:

During normal quiet respiration, only 3 to 5 percent of the total energy expended by the body is required for pulmonary ventilation. But during heavy exercise, the amount of energy required can increase as much as 50-folds, especially if the person has any degree of increased airway resistance or decreased pulmonary compliance. Therefore, one of the major limitations on the intensity of exercise that can be performed is the person's ability to provide enough muscle energy for the respiratory muscle alone.

Guyton twelfth edition, page 468

** so if I increase the pressure, the work will increase, therefore ATP consumption will increase, and this is bad.

❖ The work of breathing is spent on different forces:

1. 70% Static elastic forces :

(not dynamic, the force of bringing anything back to its original state).

- 2/3 Surface tension (we will discuss it in details in a second).
- 1/3 elastic fibers (if you want to inflate your lung, you have to stretch the elastic fibers and you need work to do that. So when these fibers are lost due to many causes as seen in emphysema, it becomes easier to inflate but harder to deflate because there won't be elastic recoil to help you "expiration becomes harder").

2. 30% Dynamic; non elastic:

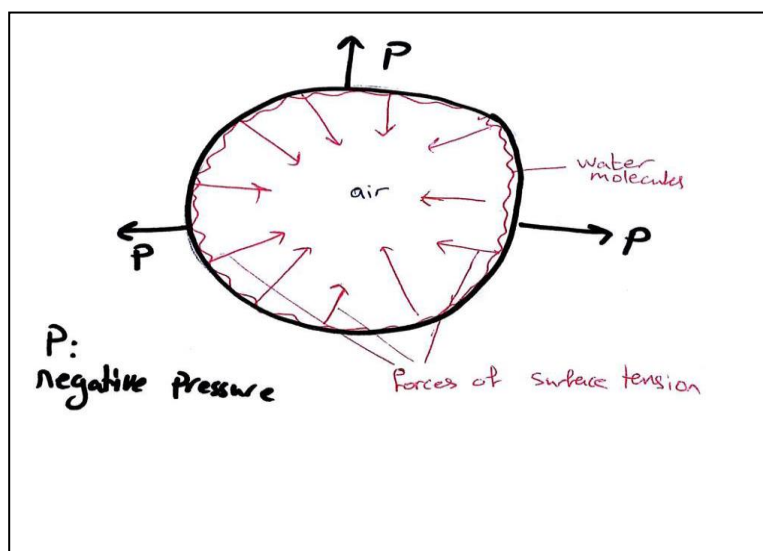
- Airway resistance 80% (manifested only during air movement, that's why it's considered dynamic).
- Viscosity of tissues 20% (the interstitial tissue opposes the movement during expiration and inspiration {just like the gum oppose your motion when you try to change its shape}).

Now, let's go back to the title I've put. What are the forces out of all I mentioned that cause the recoil tendency of the lungs?

They're the static non-dynamic elastic forces; surface tension and elastic fibers.

• Surface Tension:

- It's the intermolecular attraction between water molecules due to their polar nature through hydrogen bonds.
- Imagine we have an air bubble (walls are made by water molecules and inside is air) ("water-air interface"), the water molecules will try to come together bringing the bubble to the center causing it to collapse, surface tension is a collapsing force.



*If we apply pressure in the opposite direction we'll be able to keep the air bubble stable for a very long time, now the question is: what is the magnitude of that pressure?

We apply LaPlace's law to know it:

$$\Delta P = 2T / r$$

*the pressure is increased as we increase the tension and it's increased as we decrease the radius because we're bringing water molecules closer to each other making the attraction easier.

* We expect that we need a larger negative pressure to overcome the surface tension in a baby because his alveolar radius is way smaller.

* Let's move the same concept to the alveolus and ask a question:
What is the negative pressure needed to overcome the tension if the alveolus was lined by:

- 1) Water vapor (H₂O)? It equals -23 mmHg (when r equals 100 µm)
- 2) Interstitial fluid (less polar than water)? It equals -13 mmHg
- 3) Surfactant (along with water vapor)? It equals -4 mmHg

** Remember whenever you increase the collapsing tendency, the negative intrapleural pressure will increase.

➤ What is the surfactant (surface active agent) ?

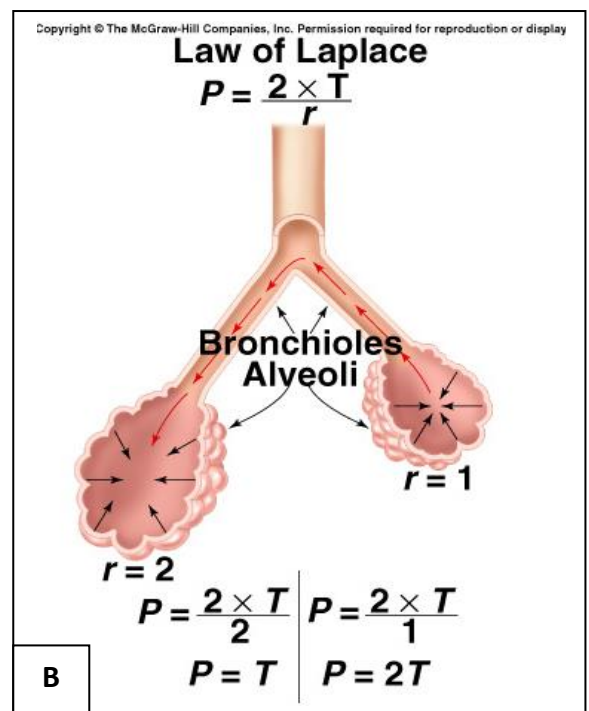
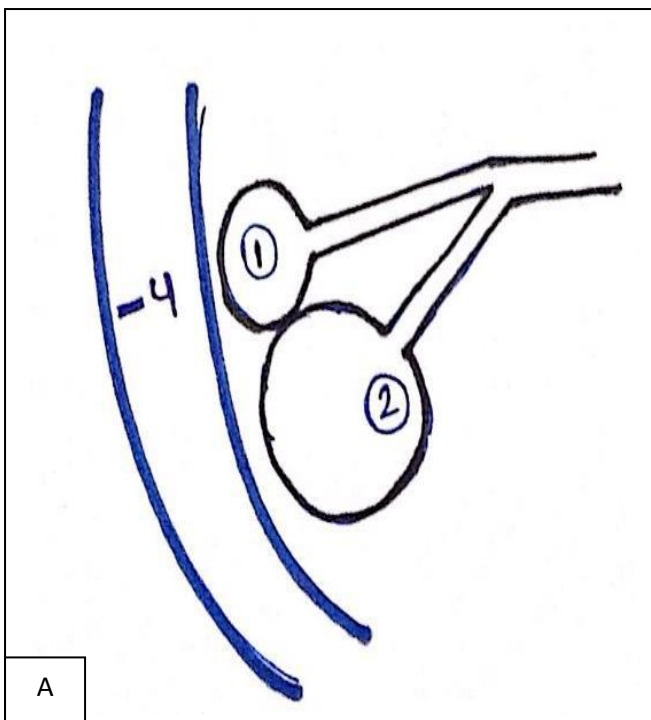
- It is produced by Alveolar type II cells.
- It is Glyco(2%)-lipo(90%)-protein(8%) plus calcium ions, so it's mainly composed of phospholipids.
- It has one hydrophilic head (glycerol moiety) and two parallel hydrophobic fatty acids tails (phospholipid) facing alveolar air. Thus, the hydrophobic portion is oriented towards the air, and the hydrophilic portion is facing the wall of the alveoli (hidden).
- Surfactant reduces surface tension forces by forming a monomolecular layer between aqueous fluid lining alveoli and air, preventing an air/water interface.
- Respiratory distress syndrome: occurs in premature newborns, their lungs collapse since they can't produce surfactant to reduce the collapsing force also they have minimal muscles so they can't produce forceful inspiration.

- If surfactant is absent, the collapsing forces would be huge, and thus, the negativity needed in the pleural cavity will be higher (for example; -14 instead of -4 in a specific region). Since more pressure is needed, more muscle contraction will be needed (to provide a higher force) so higher work of breathing. The opposite applies when surfactant is present.

Let's go back to LaPlace's law.....

According to it (see figure B):

- Pressure in pleural cavity is directly proportional to surface tension; and inversely proportional to radius of alveoli.
- If different alveoli of different sizes exist in the same region, the pressure that is needed to keep the smaller alveoli open, would be greater than that for the larger alveoli, if surface tension was the same in both, so the smaller alveoli will empty their content into the larger alveoli if the pressure was the same for both.



**** (see figure A)** but actually that doesn't apply in our lungs, let's assume there are small (1) and big (2) alveoli in the same region, both of them are surrounded by the same negative pressure (-4), so as a result & like we said above; the smaller alveolus should automatically collapse and empty its content into the larger alveolus (since we don't have a specific pressure for each one, for example, for the big one (-4) and for the smaller one (-6) to oppose the collapsing force, rather it's the same for both (-4)).

**** But such a situation normally doesn't occur (collapsing of small alveoli), so what happens??**

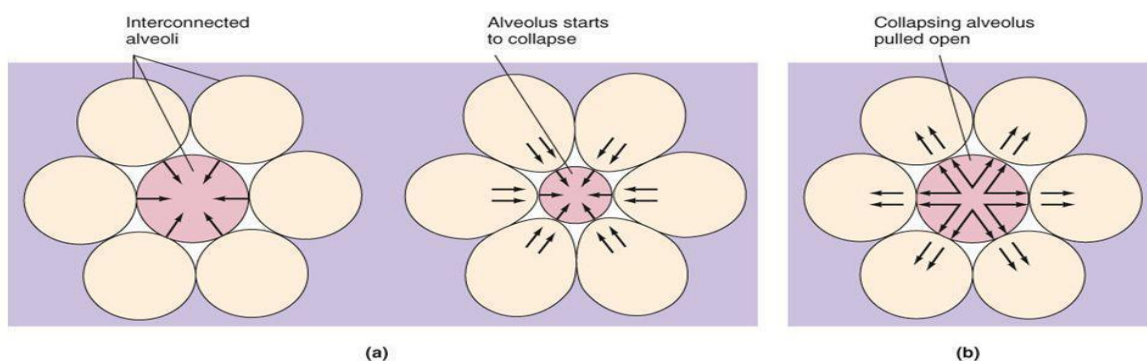
- Here comes the function of the surfactant since in reality, if the radius of the alveolus becomes smaller, the concentration of the surfactant increases. (the amount of surfactant is the same, but because the volume has decreased, the concentration of surfactant increases and thus becomes more effective in reducing surface tension).

And from that concept we have what we call Alveolar stability

****Alveolar stability:** small alveoli can coexist with large alveoli in the same region in the lung (same surrounding intrapleural pressure) due to two reasons:

- 1) Surfactant makes surface tension volume dependent.
- 2) Alveolar traction, also called "alveolar interdependence":
 - Alveoli are surrounded by other alveoli and interconnected by connective tissue.
 - If an alveolus starts to collapse, surrounding alveoli are stretched and they apply expanding forces on the collapsing alveolus (because the surrounding alveoli are trying to resist the extra stretching) thereby help to keep the collapsing alveolus open. This is called alveolar interdependence. Alveoli depend on each other (see the figure below).

**Another factor which helps keep the alveoli open is:
The Alveolar Interdependence**



If an **alveolus start to collapse** the **surrounding alveoli** are **stretched** and **then recoil** exerting **expanding forces** in the **collapsing alveolus** to **open** it

Important Notes:

1- When the radius decreases, the surface tension simultaneously decreases, to maintain the same needed inflation pressure (here, it's -4 in our example).

2- Surfactant makes surface tension volume-dependent:

- Smaller volume → less surface tension (because the surfactant would be more concentrated and thus more effective).
- Larger volume → higher surface tension.

3- We keep saying “in the same region” because different parts (regions) of the pleural cavity have different intrapleural negative pressures.

4- Loss of interdependence as a result of the loss of alveolar walls will cause a greater tendency to collapse.

Sorry for any mistakes

Please if you want and if you have free time, fill this form (I'm waiting for your opinions) 😊

<https://goo.gl/forms/vyWdTWMEFeIVHWi1>

