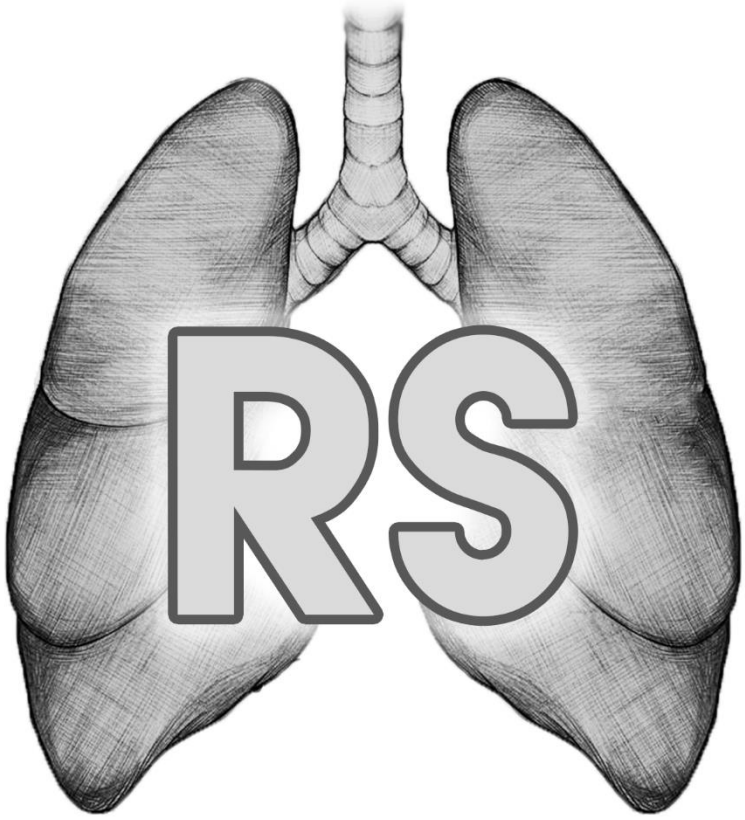


PHYSIOLOGY

☑ Sheet ○ Slide ○ Handout

Number

4

Subject

Lung Compliance

Done By

Nada Hajjaj

Corrected by

Alma Jarkas

Doctor

Yanal Shafagoj

Date: **00/00/2016**

Price:

***This sheet was written according to section 1 recording.**

❖ Before we start with this lecture's topic, few notes regarding **bronchial asthma** will be discussed:

- Why don't we consider bronchial asthma as part of COPDs?
  - Because it's usually acute disease that resolves by the age of puberty. But if it lasts for a long time it enters COPD category.

- How do we know whether bronchial asthma is reversible or irreversible?
  - We measure FEV1 before and after giving bronchodilator (B2 agonist), if we noticed improvement (increase) by 12% [approximately 200 ml increase] then it is reversible. If less than that we give cortisone inhaler (remember that asthma causes increase in mucous secretion and bronchoconstriction, in addition to inflammation of the epithelium and subepithelial layers and cortisone is an anti-inflammatory drug, then we repeat the test after two weeks to see the prognosis of the disease.

✓ We can give an asthma patient:
1) Bronchodilator.
2) Mucolytic drug: to destroy the mucous plugs which are solid and need to be lysed.
3) Glucocorticoids.

Note: Cough suppressants are contraindicated in every productive cough {any cough that secretes sputum} because we need the sputum to go out not to be locked in. And in children we consider every cough a productive cough (they can't express whether the cough is productive or not). So it is contraindicated in children.

❖ Lung Compliance:

- Lung is an elastic structure, which means two things:

1- It is compliant: capable to change its shape to a certain degree

2- It has recoil tendency: the recoil collapse tendency in value equals +4 mmHg, we have an intrapleural pressure which equals to it but in an opposite direction (-4 mmHg) that prevents the lung from collapsing.

Note: If we remove the negative intrapleural pressure, the lungs tend to collapse to their **minimal volume** and that's what happen in pneumothorax [intrapleural pressure become 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 have a problem understanding the previous concept please read what's in this box:

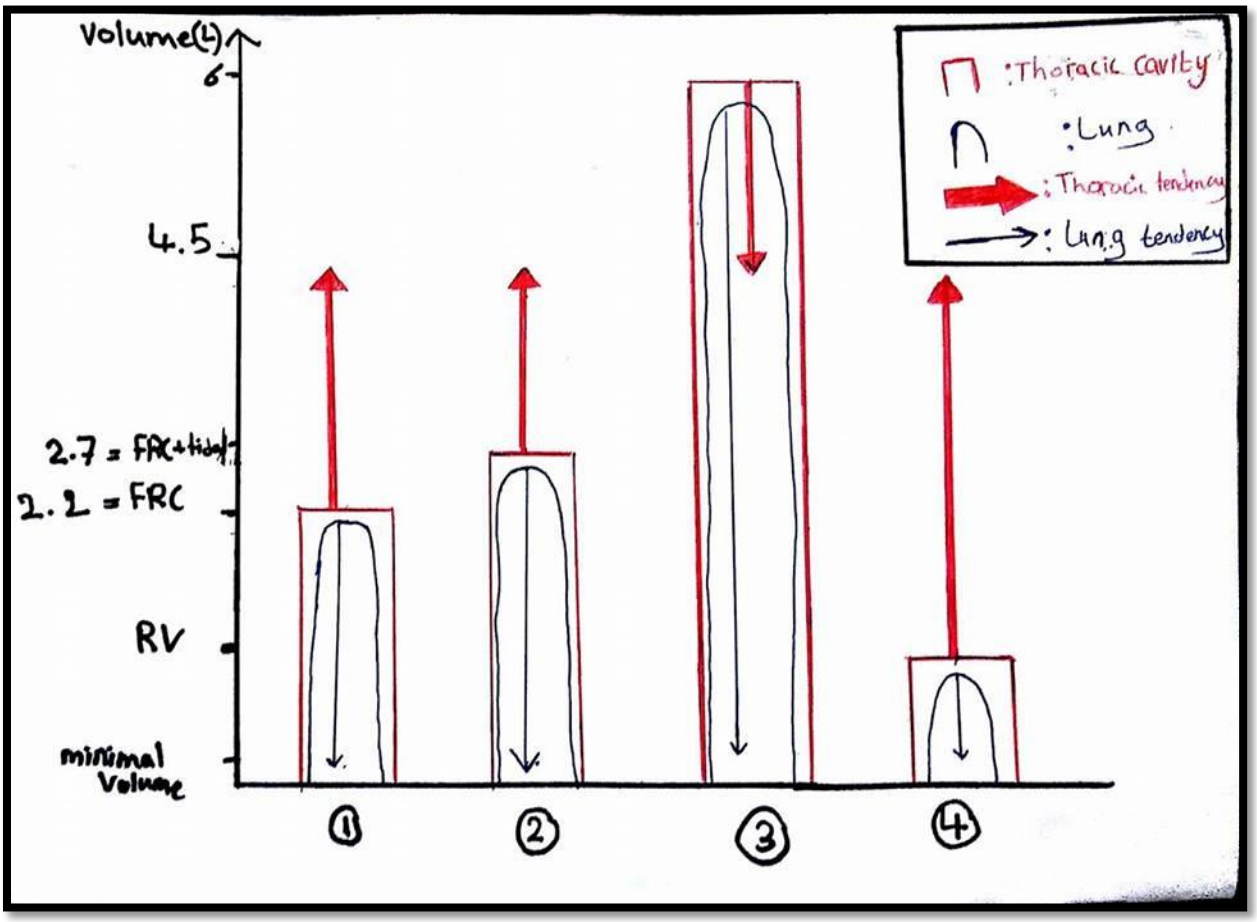
"To understand the consequences of pneumothorax, it must be recognized that, normally, the intrapleural space has a negative pressure(less than the atmospheric pressure).This ***negative intrapleural pressure*** *is created by two opposing elastic forces pulling on the intrapleural space: The lungs with their elastic properties, tend to collapse, and the chest wall, with its elastic properties, tends to spring out. When these two opposing forces pull on the intrapleural space, a negative pressure, or vacuum, is created. In turn, this negative intrapleural pressure oppose the natural tendency of the lungs to collapse and the chest wall to spring out."*

Cosatnzo, chapter 5, page 196.

What do we mean by the minimal volume?

- It is the resting 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.

- ✓ So we conclude that Lung tends to collapse. In contrast, thoracic cavity 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:



At 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%*6=4.5 liters.

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).

And these forces a+b are equal in value and opposite in direction. Thus, <u>the system (lung-thorax system) is at rest</u> at a volume equal to FRC.

At state ②: 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 is expiration considered a passive process (remember it also doesn't need contraction of muscles).

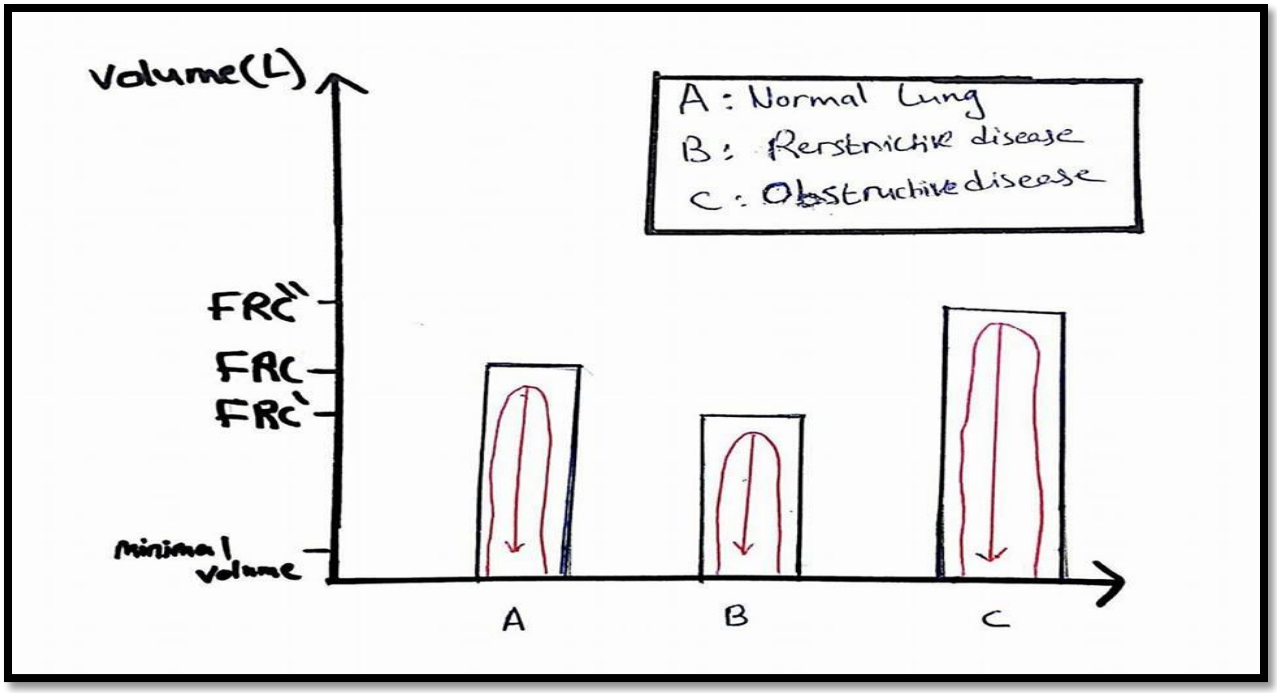
At state ③: The recoil tendency of the lungs is huge, the thorax now has tendency to collapse, because its volume now is larger than its resting volume. Both structure are tending to collapse, and the system tends to collapse in a huge force.

**Try to fill your lungs to the maximum, then close your mouth and nose and relax your muscles the air will go out غصباً عنّك.

In order to reach state ④: you need to apply force have to expire the expiratory reserve volume (ERV). Notice the volume of the lungs here is reserve 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 usually active and expiration is passive).

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

Note: In restrictive lung diseases like fibrosis, the collapsing tendency of the lung increases, our lungs adapt themselves by decreasing FRC to FRC', decreasing its collapsing tendency and keeping the system at rest.
On the other hand, in obstructive lung diseases like emphysema, the compliance of lungs increases and the collapsing tendency decreases, our lung adapt themselves by increasing FRC to FRC", increasing its collapsing tendency and keeping the system at rest. {See the figure next page}



- **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 causes 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, and that work is equal to the change in volume times the change in pressure.

W = ∆V * ∆P

- 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 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).

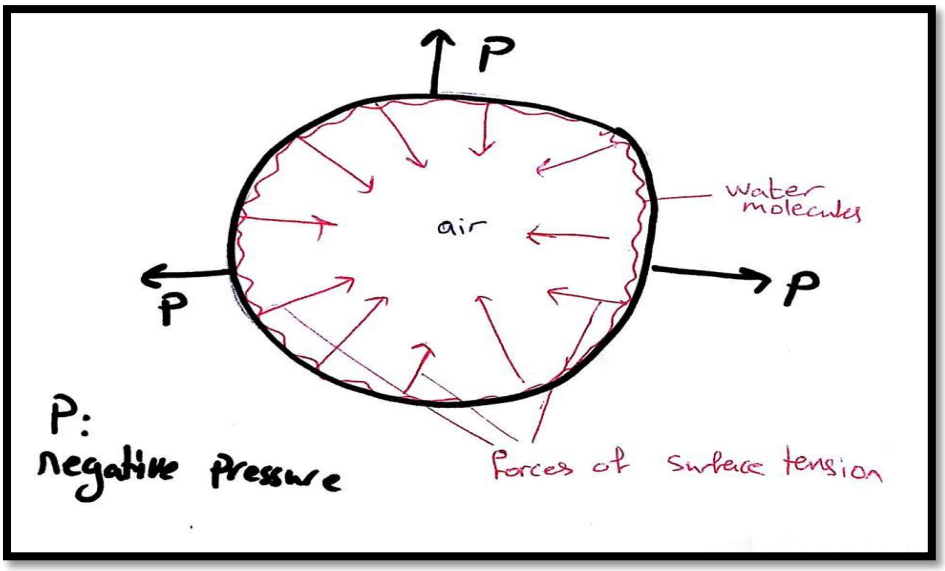
Viscosity of tissues 20% (the interstitial tissue oppose the movement during expiration and inspiration {just like the gum oppose your motion when you try to change its shape}.

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 its 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. (فقاعة الهواء تنفجر إلى الداخل وليس إلى الخارج).



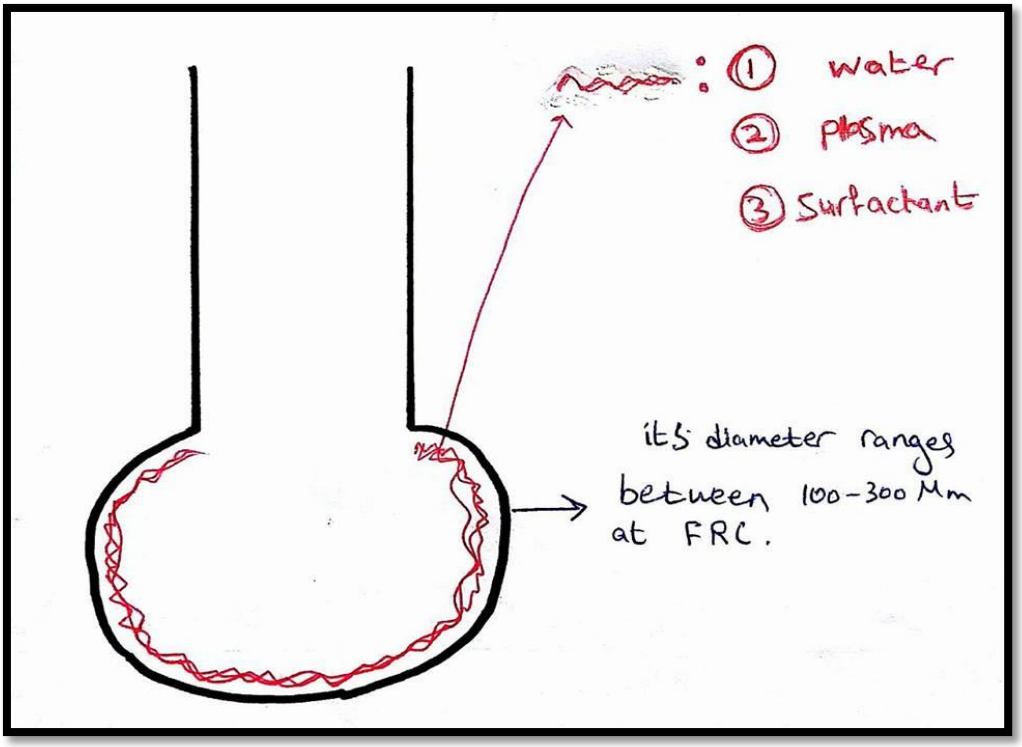
*If we apply pressure in the opposite direction we'll be able to keep the air bubble stable for a very long time, now the question is: what is the magnitude of that pressure?

*We apply LePlace's law to know it:

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

*It's increased as we increase the tension and it's increased as we decrease the radius because we're bringing water molecules close to each other making the attraction easier. [We expect that we need larger negative pressure to overcome the surface tension in a baby because his alveolar radius is way smaller].

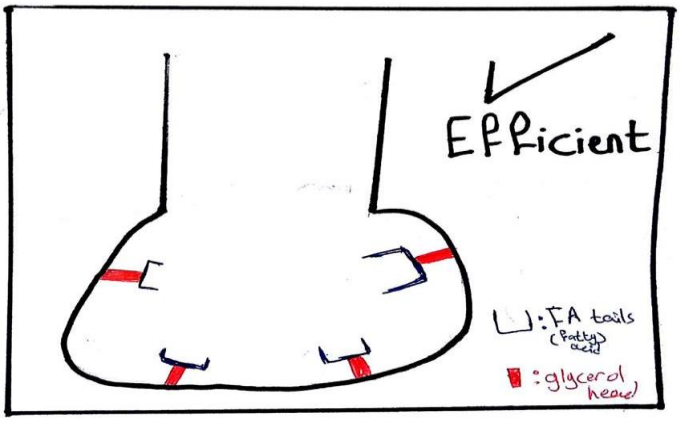
- Let's move the same concept to the alveolus and ask a question:
  What is the volume of negative pressure needed to overcome the tension if the alveolus was lined by: {see the figure below}
  1) Water vapor (H_2O)? It equals -23 mmHg
  2) Plasma (less polar than water)? It equals -13 mmHg
  3) Surfactant (along with water vapor)? It equals -4 mmHg



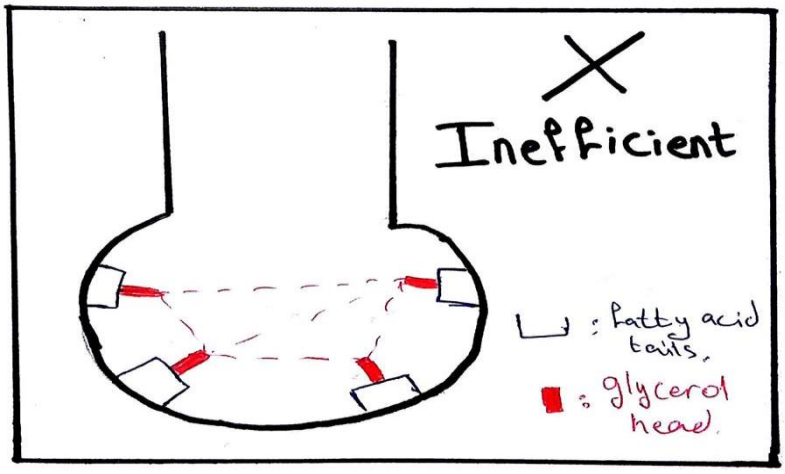
- What is the surfactant and how does it cause decrease in the negative pressure needed to overcome the tension???

  - It reduces the surface tension of water.
  - It's considered a glycolipoprotein.
  - 90% lipids, 8% protein, 2% carbohydrates
  - Non polar (not a specific thing to say).
  - Its lipid portion is mainly made of phospholipids which consist of a glycerol head (backbone) which is polar, and two fatty acid tails.

  - In order to the surfactant be capable of doing its job right, the glycerol head needs to be directed away from the air and other phospholipids molecules:



  - If the glycerol head was directed to the center, it will attracts the other glycerol head and thus losing the function desired from it.



It's not only the concentration of surfactant that matters but also the orientation of it.

- The surfactant is considered extremely important because it reduces the negative pressure needed to overcome the tension, thus saving us a lot of ATP. Need an explanation? Keep reading…

  As we've said before, **work = ΔP * ΔV

  ΔV: Tidal volume

  ΔP: The pressure we need to overcome.

**so surfactant decreases the pressure needed to inflate the lungs, as a result decreases the work (decreasing oxygen and ATP consumption). And that's the reason why the lung is considered an efficient machine, as it only consumes 2-3% of the oxygen it produces. If the surfactant was deficient Oxygen consumption will increase up to 20-50% leaving small amount to other tissues resulting in muscle fatigue and death.

The doctor mentioned the meaning of respiratory-exchange ratio:

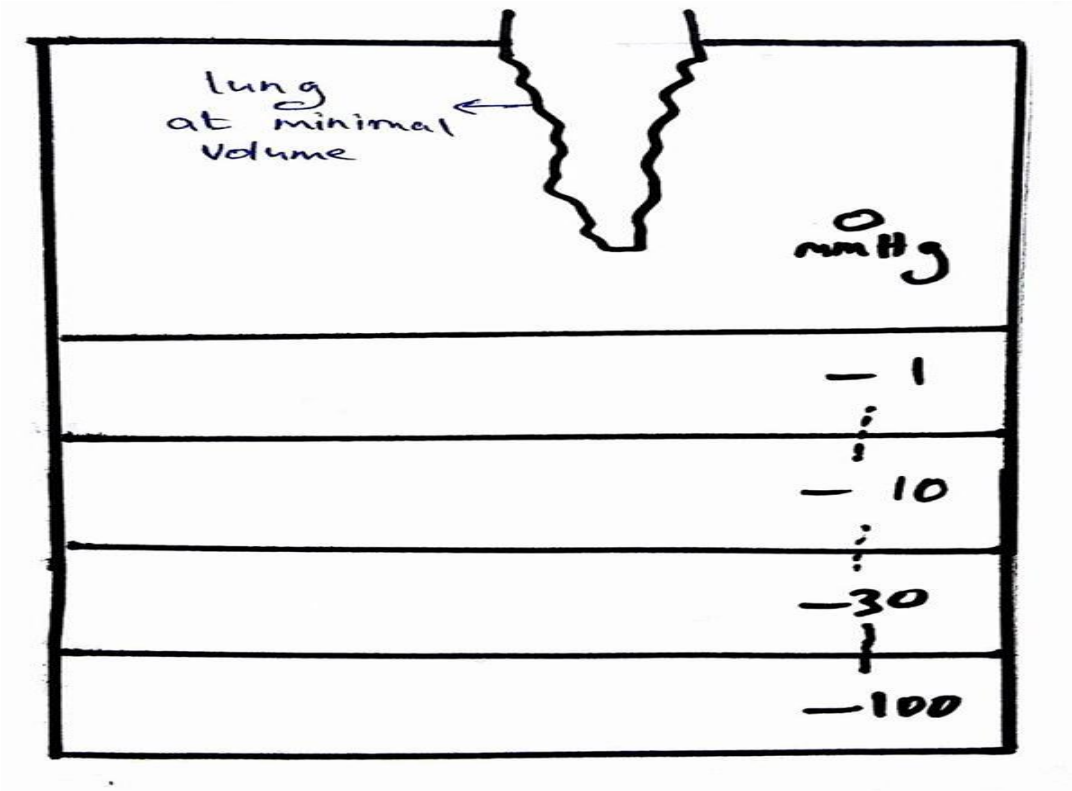
Respiratory-exchange ratio=rate of O_2 consumption /rate of CO_2 production

= 250 ml/min / 200 ml/min = 0.8

- **How did we know that the static elastic forces are:**
  1/3 elastic fibers and 2/3 Surface tension??

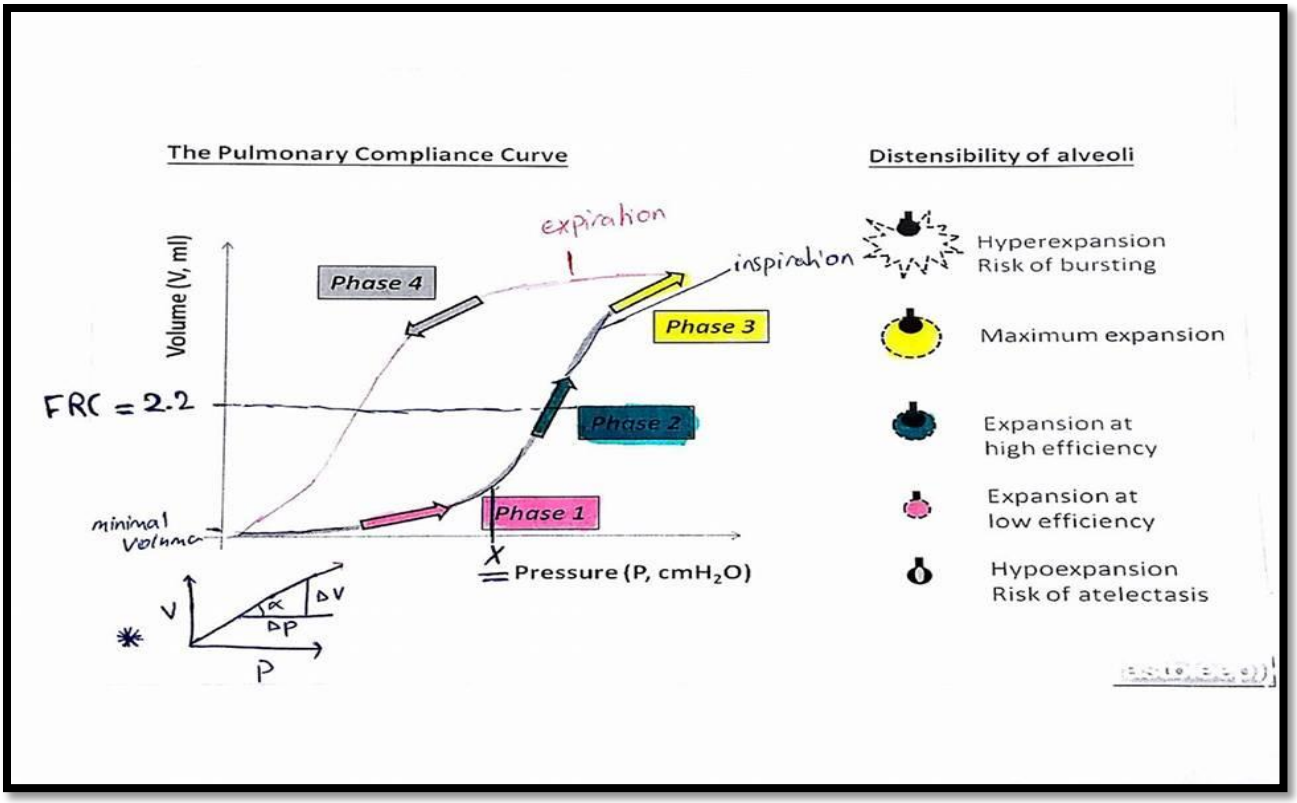
  ** We did the following experiment:

  -We put the lung in its minimal volume in a container and we change the volume of the container to increase the negativity of the pressure, and we observe the changes in lung volume.



  -And then we draw the pressure-volume curve which is illustrated next page: ⇨

  -After that we repeat the same experiment but after filling the lung with saline to eliminate the surface tension factor. {It's okay if you didn't understand the last sentence, it will be explained in details in the next sheet which is written by the amazing Reem Akiely (حُط إجريك بمي باردة)}



- Notice that as we move to the right the negativity of the pressure increases.
- The slope of the curve = tan α = ∆V / ∆P = compliance
  * Compliance is how much volume change I can get per unit change in inflation pressure (the negative pressure).
  *Too much volume change {when we apply certain force}→ compliant structure.
  *Very little volume change {when we apply the same force}→ not compliant (rigid) structure.
- Phase 1: the curve is barely elevated, too little change in volume for a huge change in pressure and a lot of oxygen and ATP consumption → not compliant.
- At certain critical pressure, a critical opening pressure, (Point X in the figure) the lungs become suddenly compliant. (بالضَّبط زي ما الذرة بتصير بُشار فَجأة بالطنجرة)
- Phase 2: we apply little force and get huge change in volume → compliant.

Notice that FRC resides within phase 2, so our inspiration plus expiration take place in a very compliant lung making breathing an easy process and that's a gift from god.

- Phase 3: you cannot inflate already inflated lung → not compliant.

- The expiration curve (phase 4) takes a whole different shorter curve and that is called hysteresis. The only explanation for that is: the surfactant is distributed not in a proper way during inspiration making surface tension higher, and in a proper way during expiration making the surface tension lesser (but it's the same concentration). So to reach a certain volume during expiration you need lesser negative pressure than during inspiration and the curve will be shifted to the left.

- **Few Important repeated notes:**

  ****** If the collapsing tendency is extremely high (it happens when the surfactant is absent), we go to the minimal volume each time we expire and it's extremely difficult to reopen the lungs at the minimal volume (phase1, low compliance), thus, if a premature baby was suffering from a lack of surfactant, each time he will expire he'll go to the minimal volume and when he try to inspire he'll consume huge amount of ATP (80% of his entire ATP available) as a result of that he'll suffer from muscle fatigue (not enough ATP is left for the rest of his body).

  ******As we said before, we cannot inflate already inflated alveoli.
  If for some reason our apical alveoli were inflated and are basal alveoli were not, when we inhale some air, it will go to our partial inflated alveoli (basal) and not to our inflated alveoli (apical). Resulting in an abnormal unequal distributed air.

  **** Infant respiratory distress syndrome:**
  Affects premature babies who have two problems:
  1) The tension is too high because no surfactant is present.
  2) The radius of alveoli is too small.

  $\Delta P = 2T / r$ → 1+2 will result in a very high pressure which equals -30 mmHg and that is impossible for a premature baby with no muscles to handle, thus, he will die because of muscle fatigue within 24 hours.

Special thanks goes to Bayan Nidal <3

The End