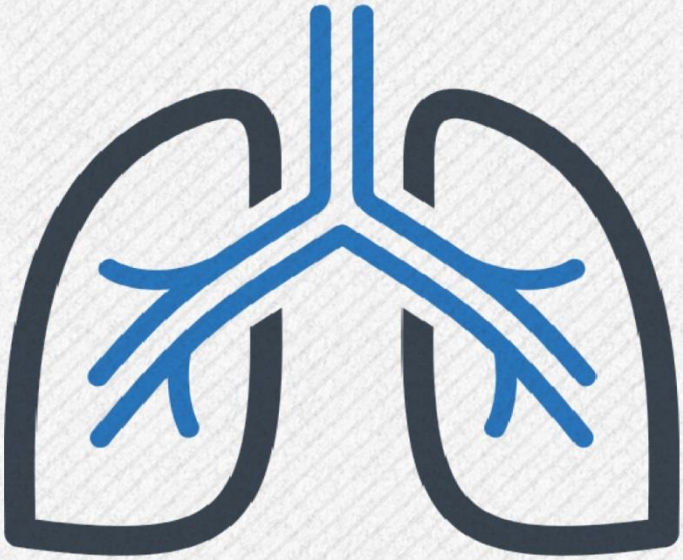

Respiratory system physiology

sheet handout slides

Number	6
Doctor	Yanal
Done by	Obada zalat
corrected by	Farah Al-huniti

Lung Compliance

The minimal volume of the lung

- Also called the resting volume or the unstressed volume; which is the volume at which the alveoli doesn't tend to collapse.

- It equals **150 ml** for the lungs.

- But how can I reach this volume?

-By removing remove the negative intrapleural pressure, the lungs tend to collapse to their minimal volume, and this cant happened in physiological state of the lungs; because after a forcefully expiration 1.1 L of air will remains in the lungs (which is the residual volume), so we can't reach the 150ml (the minimal volume).

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The Lung-thoracic cavity system

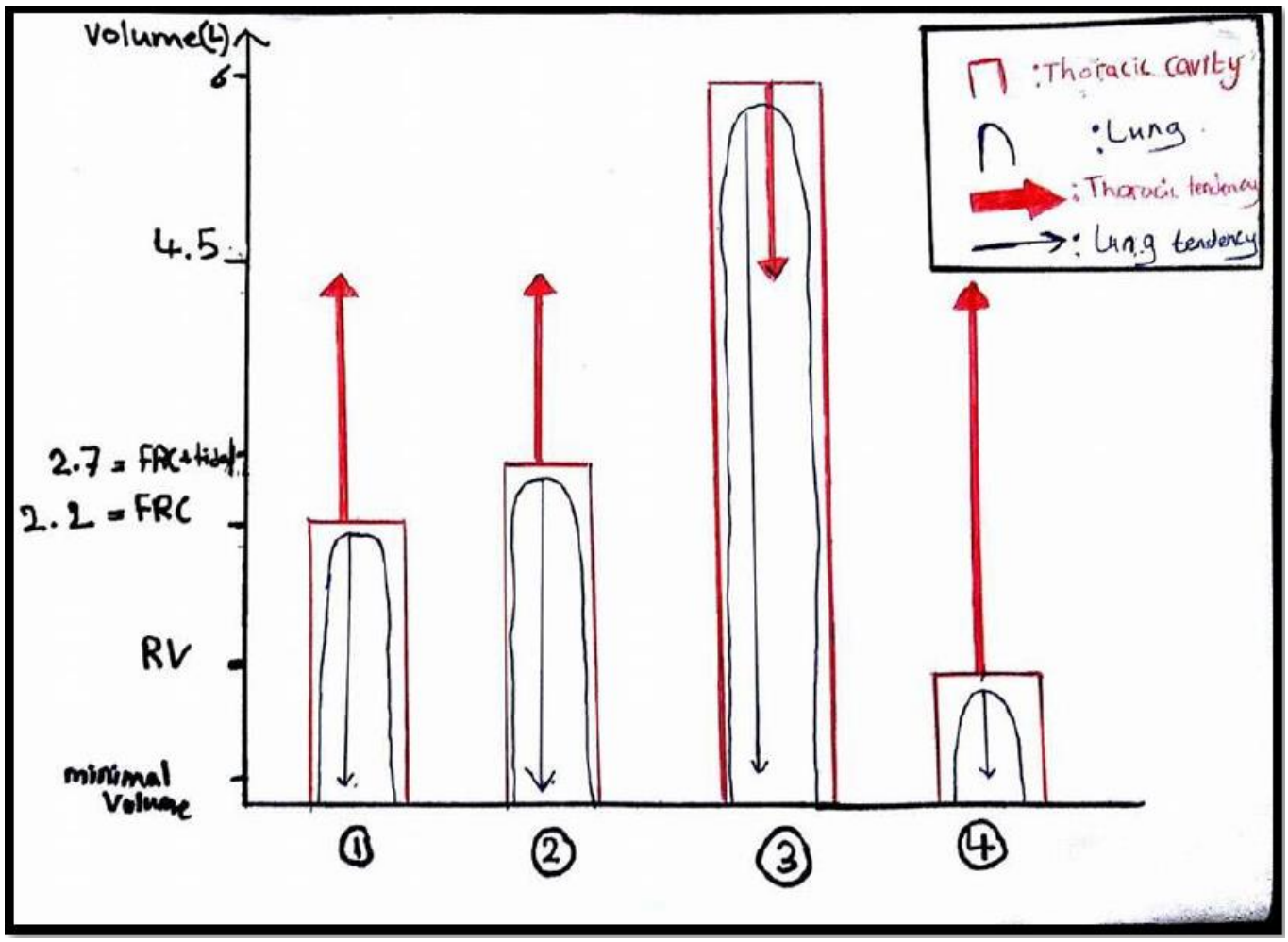
- actually we are dealing with two cavities, and each of them has its own resting volume.

- The thoracic cavity resting volume is 4.5 L (which equals 75% of TLC, 75%*6=4.5 liters), and this means that at the FRC (2.2L) the thoracic cavity is compressed.

- When the volume equals the FRC (2.2 L), the lung has the tendency to collapse, and the thoracic cavity has the tendency to expand, but the expanding and collapsing forces are equal, so the system is at rest (**system is neither tending to collapse nor to expand**)

- So, if we want to have a definition for the FRC; we can say that **"it is the resting volume of the lung-thorax system"**

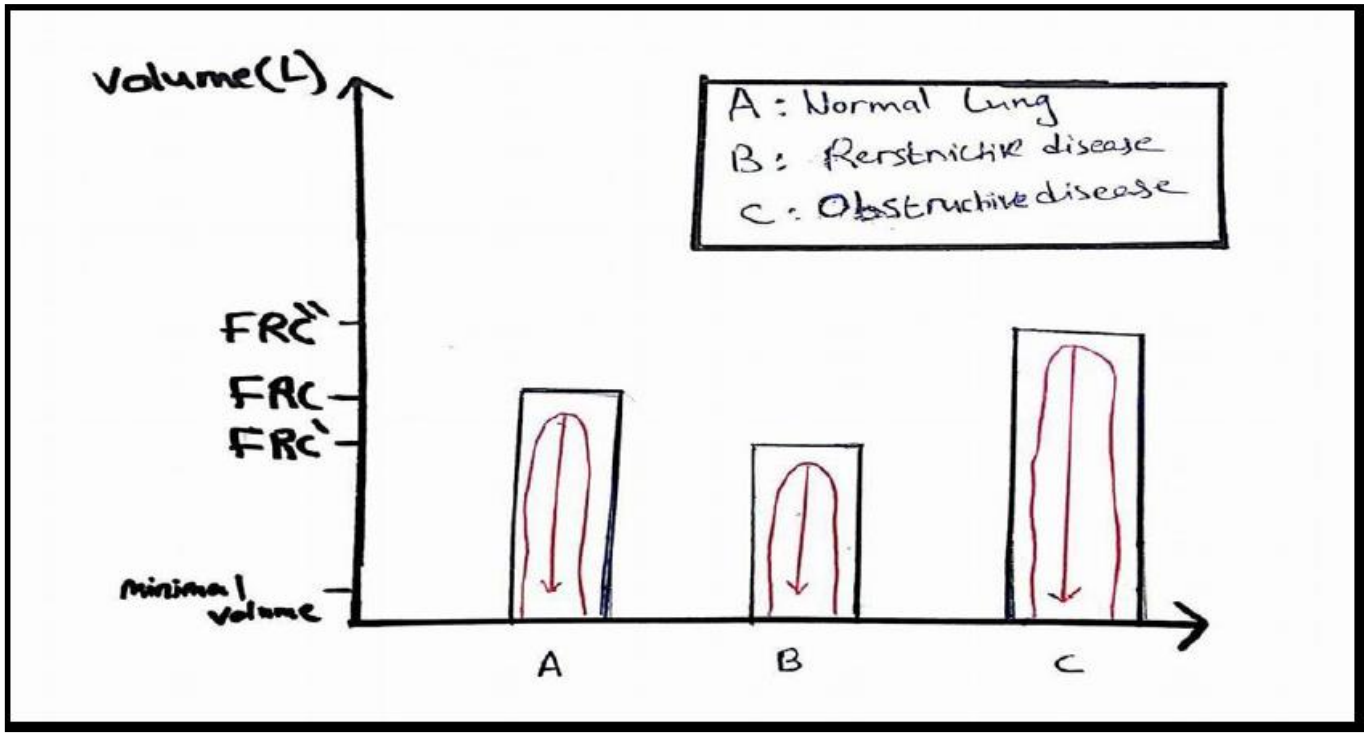
- For any elastic structure, if we want to move it from its resting state we need to apply force, but in order to bring it back to its resting state we don't need any thing (it's a passive process), and we can apply this principle on the lungs, let's see:



○As we move the lung from the FRC (as we taking the tidal volume), I need to apply force. Then, the tendency of the lung to collapse increases, as well the expanding tendency of the thorax decreases, so the system has the tendency to collapse, , and that can explain why the expiration is passive process, in contrast the inspiration is active (state 2).

○If you filled the lung to the maximum, tendency of collapse of the lung is huge, as well the thorax also tending to collapse (in order to reach its minimal volume), so the system now has a huge tendency to collapse (state 3).

○If I want to exhale to the residual volume, I need to apply force. Then, the tendency of the lung to collapse decreases, as well the expanding tendency of the thorax increases, so the system is tending to expand, so the inspiration here is passive (state 4).



○This figure illustrate the FRC- lung abnormalities relation:

○In restrictive lung diseases like fibrosis, the collapsing tendency of the lung increases, but the expanding tendency of the thorax is normal, so our system is now not at rest, and in order to bring it to the normal state, the lung adapt by decreasing the FRC to **FRC`**, decreasing the collapsing tendency and keeping the lung 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 .

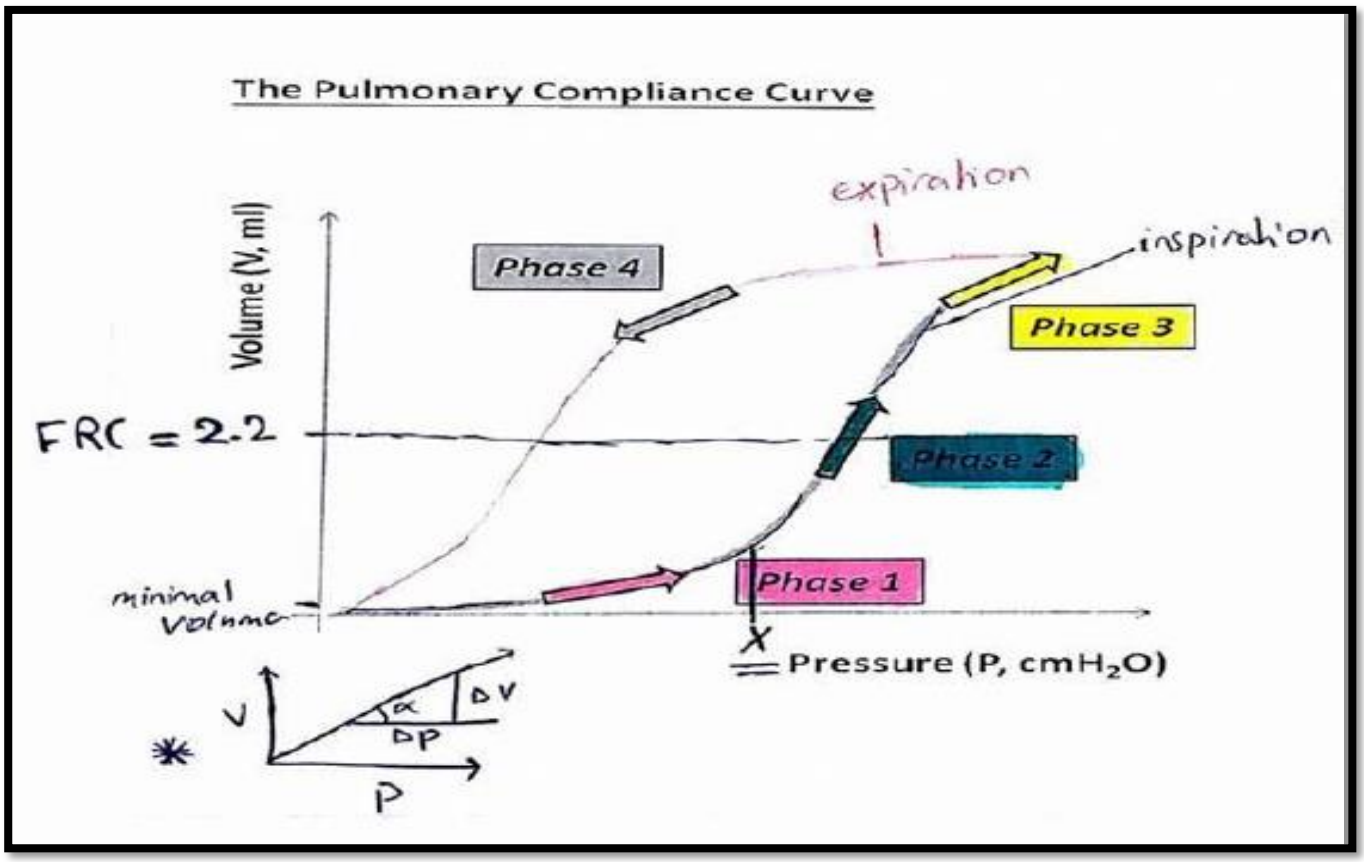
○So, whenever you see a patient with high FRC, you should know that his lung has low tendency to collapse (such as in obstructive disease), as well as if you see a patient with low FRC, you should know that his lung has a high tendency to collapse (such as in restrictive disease).

♦How we can inflate the lungs

volume of the container (actually we increasing it), and this will lead to increase the negativity of the pressure; according to Boyle's law. And this will inflate the lungs. But what is the relation between the pressure of that container and the volume of the lungs?!

- The answer will be clear after discussing this curve

◯Now let's have a look on this curve:



◯This curve shows the compliance of the lung (volume – pressure relationship)

◯Notice that as we move to the right, the pressure becomes more negative

◯It's very important to know that this curve is a static curve, which means that I measure the volume at each change in the pressure step by step, look at this example to understand what I mean:
If I left a chair, and I want to measure the static tension in my biceps, I will measure it while I'm lifting the chair only, not during lifting the chair and moving with it

◯So, we start decreasing the pressure around the lungs ((by increasing the volume in the thorax)), and we monitor the change in the volume of the lung, and we will have the following:

◯At the first the curve is barely elevated, too little change in volume for a huge change in pressure and a lot of ATP consumption; **not compliant** (Phase 1)

Remember that……
-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.

◯At **critical pressure**, the lung will become very compliant, then if we apply little force, we will get huge change in volume > compliant (Phase 2)

◯At the end of the curve; you cannot inflate already inflated lung (This is the Maximal point) > not compliant (Phase 3)

◯So, don't try to inflate already inflated lung (Phase 3), but always try to inflate **partially inflated** lung (Phase 2)

♦ **How to deflate the lung**

◯In order to deflate the lung, we need less negative pressure (positive pressure) around the lung, so I start to decrease the volume around it.

◯The logic said that I should retrain the same pathway of inspiration for expiration, but this is not applied here!!

◯The backward process is different than the forward process and this is called **Hysteresis.**

♦**NOW how can I read this curve** (the compliance curve of the lung)**?**
-We draw a line from the volume (Y-axis), for example at 4L, and then we look at the pressure that needed to keep the lungs uncollapsed during inflation and during deflation.
So, we can see that at inflation we need more negative pressure (more force)

to keep the lungs uncollapsed than in the deflation, but how can that exist?

◯Simply, the key is the **surfactant orientation**

◯But before talking about the mechanism let's talk about the structure
-Surfactant is considered a glycolipoprotein; 90% lipids, 8% protein, 2% carbohydrate
- Lipid portion is mainly made of **phospholipids** which consist of a glycerol head (backbone) which is polar, and two fatty acid tails.

◯So, if the surfactant oriented in a way that the non-polar fatty acid tails facing each others; there will be no surface attraction, as well the tension will decrease, so the negative pressure needed is less **(-4 mmhg is enough)**

◯But if the glycerol head was directed to the center, it will attracts the other glycerol head and thus increase in the surface tension, and we need more negative pressure **(-6 mmhg).**

◯So, not only the concentration of the surfactant that matter, but also the orientation of it.

◯During inflation, the surfactant is not oriented in a proper way, so it's not effective in reducing the surface tension, and thus we need much force to inflate the lung

◯On the other hand, during deflation you compress the lung, the surfactant will be oriented in a proper way, so less surface tension as well less negative pressure.

◯In order to know some abnormalities related to that curve (the compliance of the lung) we study the deflation curve because it is less complicated.

◯For example, if the curve shifted to left, which means that the patient has higher compliance, so he might suffer from emphysema. In contrast if the curve shifted to the right, this means less complains, so he might suffer from fibrosis.

RDS (Respiratory Distress Syndrome)

♦IRDS (Infant Respiratory Distress Syndrome)

◯If we have a premature baby, this means he has immature lungs, so there is no surfactant; thus too much surface tension (high collapsing tendency), and this will make the baby every time he exhale, he goes to the minimal volume, and each time he want to re-inhale, he will go throw the whole path of inspiration (from phase1 to phase 3).

◯That will consume too much energy; to re-open the alveoli. In addition to that, this baby needs **(-30mmgh)** pressure instead of (-4mmhg) in normal human.

◯This (-30mmhg) is a very dangerous pressure, but why?

◯First we will take a little bit about the capillaries physiology:
-Normally, we have filtration and reabsorption happened at the wall of capillaries, and these events is controlled by what we called **starling forces**

- Starling forces are 4 forces, and they are:

①**Pc**; Capillary Hydrostatic pressure - a filtration force (pushes fluid outside the capillary)

***it equals (10 mmhg) in pulmonary capillaries**

②**πc**; osmotic force due to plasma protein concentration – a reabsorption force,

***it equals (28mmhg) in all capillaries of the body**

③**Pi**; interstitial fluid Hydrostatic pressure - if negative, it's a filtration force, and if positive, it's a reabsorption force.

***it equals (-5mmgh) in the lungs >>represent the intraprular pressure**

④**πi**; osmotic force due to interstitial fluid protein concentration - a filtration force.

***it equals (14 mmhg)**

◯So, we have three forces that have filtration force to the outside of the capilaries, and they equals **29mmhg,** in contrast to **28 mmhg** reabsorption force, that means we have **+1mmgh net pressure (more filtration than reabsorption),** but because the presence of lymphatics, we can deal with extra amount of fluids

◯So, in our case (the pre-mature baby), the Pi will become (-30mmhg), that means we have (10+14+**30**)= **54mmhg!!** toward outside, in comparsion with 28mmgh. The net will be **26mmhg outward**, and this exseed the capacity of the lynphatics, so this baby will have **pulmonary edema**, that will lead to >>>**death**.

◯Now, we have a full idea about the surfactant functions, which are:

①Decreases the surface tention

②Decreases the collapsing forces

③Decreases Work of breathing

④Makes the surface tension, volume dependant

⑤Prevents the occurance of pulmonary edema

◯During the gestation period, the baby needs; prolactin, estrogen, thyroxin (T4) and glucocorticoids (cortisone) in order to produce surfactant.

◯We can predict the lung maturity via using lung markers, which could be taken from the amniotic fluid (by amniocentesis), and then we can decide the maturity of the lung depending on the marker ratio such as:

①Lecithin to sphingomyelin ratio If > **2**; the lung is mature

②Phosphatidylglycerol If present… the lung is mature

③Surfactant to albumin ratio; this is the most important lung marker.

Surfactant (mg) / albumin (g) - If > **55**; the lung is mature -
35 – 55(intermediate) - If < **35**; the lung is immature

◯If the lung is not mature, I will try to delay the delivery as I can (even one day makes a different).

◯Also, I will give the mother two shots of **dexamethasone** (glucocorticosteroid), and I will advise this mother to deliver in a very advance center (where they can take care of this premature baby)

◯Some notes about steroids:

-Naturally occurring steroids in human bodies are of three categories:

① C18: has 18 carbon atoms (like estrogen)

② C19: has 19 carbon atoms (like testosterone)

③ C21: has 21 carbon atoms (like aldosterone and cortisone)

-Dexamethasone has 22 carbon atoms, which mean that it's a **synthetic steroid**.

◯So, before birth we can give **two shots** of Dexamethasone for the mother, but after birth, what we can do?

◯Actually, this baby has many problems such as pulmonary hypertension; because of hypoxemia that induce vasoconstriction in the pulmonary arteries , and this hypertension will lead to open of **Ductus arteriousus (**between the aorta and the pulmonary artery), so we will have mixing of venous and arterial blood >>more **hypoxemia** and **acidosis** (because of shifting from aerobic respiration to anaerobic due to hypoxia, which leads to accumulation of lactic acids>>acidosis)

◯All these problems will make his respiratory rate 60 breath/min, so he fighting for air and he will die after few hours, unless we interfere, but how?

①We provide the baby with CPAP (Continuous Positive Airway Pressure)

②We provide the baby with PEEP (Positive End-Expiratory Pressure); we connect the baby to a ventilator

-The baby will be kept on the PEEP method (maybe for two weeks, three weeks, or even more; depending on the case), until his condition improves and surfactant becomes enough.

GOOD LUCK