Important: these are LECTURE NOTES written by our colleague Shaimaa, it is not the original sheet.

- it's important that we don't waste lots of energy to breathe.
  - the energy we use is very little... the work of breathing which is equal to change of volume x unit change of pressure
    - change of pressure therefore would be more than expected in increased work
    - for example -1 to drive air way, but to drive air out is not ok. +1 is not enough, +10 means you havemore breathing work
    - the work, we use ATP to overcome two types of forces: elsti forces (70% of work), this comes in two forms: surface tension (2/3( and elastic fibers (1/3)... these elastic forces are static (vs. dynamic) when there is no airflow.
    - secondly, non-elastic forces which make 30% of the work and is of two forms as well: airway resistance (Which we will talk about today) makes up 80% of these forces and tissue viscosity is 20%...
    - elstic force smanifested when there is no airflow; with airflow you need additional forces to drive in and out... it is difficult to drive something vs keeping it at the same place (static vs dynamic)... think of isometric contraction of the muscle while carrying something
    - think of this example: when you are breathing, you air inflating the balloon and to do this, let's assume it's closed... to keep it inflated you need an opposig force (the negative pressure) the balloon is tending to collapse with +10, you need -10 pressure. However let's assume an open balloon and you have an airway. To drive air in, you have to overcome the rsistance in the airway... when there is no airflow, you cancel the airways, you only care about the collapsing tendency of the balloon... therefore during airflow, you have to overcome 1) collapsing force of the balloon and 2) the airway resistance
    - think of clay: stretching it needs force, and to return it you need a force as well:: non-elastic... vs. alveoli: there is surface tension = collapsing force.
therefore two types of forces... one is static and another is
dynamic

- static forces is elastic forces which takes 70% of the total work and
dynamic takes 30% in normal cases; and each have two components...
  viscosity will oppose motion in either direction; collapsing forces you
  should apply force in one direction and in another it is passive...

- dynamic: 80% airway resistance and 20% from viscosity...

- today we talk about airway resistance...

- a person who is a chronic smoker: to test his airway resistance due to
  smoking, we perform the following:
  
  - he expired which has 3 phases in a t vs volume graph
    
    1: he has exhaling, compare to the LV: rapid ejection and
    filling:: the first part leaves when high flow occurs.... the
    first 25% of the VC, the first L will leave easily in both
    normal people and patients... the last L will leave with
difficulty in both as well. Therefore, the middle 50% is
    where the difference lies.

    therefore if we cancel the first and last 25%, we end up
    comparing the middle portion and here we can see the difference.

    for example FEV 1 / FVC = 75% and the normal is 80%. 5% is
    not that significant, or so it’s assumed...

    but if you cancel the first and last 25% and just take the
    middle: mid-mean expiratory flow you will see the
    difference here might be double. This is why this is a more
    sensitive test: the flow was diluted in the first flow and
    slow flow in the last... you are taking the middle one.

    middle vital capacity / t = 3.5 L/sec normal

    this test is sensitive, but perhaps we can look at something
    more sensitive.

- let’s take a lung with an intra-pleural pressure of -8 at the apex and at
  the base -2... therefore the alveoli are more inflated at the apex than
  those in the base that are partially inflated.
- when you take 500 mL ventilation as you remember from the compliance curve, at high volume the lung is not compliant. You cannot inflate an already inflated alveoli. Which means that most of the 500 mL goes to the base rather than the apex. Therefore, ventilation of the base is more than that of the apex.

- Secondly, the heart: It's much easier for the heart to eject the blood to the base than to apex (due to gravity, in standing position). The perfusion of base is more than that of the apex... These are in their absolute terms, what I care about is relative. Absolute ventilation/perfusion is insignificant, you care about the perfusion/ventilation ration (and wasted perfusion or ventilation).

- Let's take a curve of perfusion going from the base to apex (base: max, apex: min).

  - Ventilation is also higher in the base... ventilation/perfusion ration is less than 1 (you have more blood in the base).
  
  - In the apex it is more than 1, more ventilation than perfusion.

  - What does this mean?

  - The apical alveolus: where we have more ventilation than perfusion. What would you expect the pO2 to be? (thrombosis: no perfusion, pO2 = 150 (like that of the outside air)... but with a bit of perfusion, it would be between 100-150, close to 130 as an example... what would be the pCO2? Less than 40 and the pH2O: 47 and pN2 = the remaining...

  - Let's go down to the base where have more blood the ventilation. The pO2 here would be < 100, close to 90 mmHg.

  - Ask the patient to breathe pure oxygen (100% oxygen) ... most of this oxygen is going to the base because it is higher than that of the base (absolute ventilation). Secondly, perfusion of apex is more than that of the base....

  - Missing information.
• when we take 500 mL, assume 400 mL went down and 100 mL above. The 100 mL are not going to dilute the air like those of the 400 mL, therefore the nitrogen of the 400 mL will decrease... at the end of tidal volume, pN of the base < apex. While exhaling, we analyze two things: nitrogen in expired air + the volume. --> Volume vs. nitrogen graph

• the first phase has no nitrogen, because it is pure oxygen of the ADS. Then it slowly begins to increase, which comes from both the apex and the base: the air is coming from both. But if a person has a slight degree of increased airway resistance, which will close first? Basal or apical bronchioles? The apical are surrounded by more negative pressure (-8).... while inspiring, the basal one will close, so the last portion of the air comes from the apex, and this air contains more nitrogen- you can see early changes in the airway resistance.... so the last increase of nitrogen (closing point) comes from the apex, and the basal bronchioles close.

• closing volume, a very sensitive test which can predict early obstruction in the airways. Before we understand the closing volume, we must also understand other things

• the first of which is the intra-pleural pressure is not equal at different regions of the lung: at the apex more negative than the base... -8 int he apex means that the alveoli at the FRC (before taking Vt) are already inflated. When you talk about already inflated alveoli you go back to the compliance curve where the two ends of the curve, they are not compliant at small nor large volumes. Not compliant when the alveoli are partially inflated, completely deflated or almost completely inflated: the lung is no longer compliant

• you take 500 mL which go to the alveoli down to the base because it is ..... ... ****

• due to gravity: more to the base > apex.
more ventilation and perfusion at the base but the ration of v/ p, we will realize that the perfusion is more in the apex, less at the base.

more air: composition of the alveoli air is closer to that of the air (apex).

when you take in 500 mL, 400 mL go down (pure oxygen), they will dilute the basal air more because the 400 > 100. So the nitrogen at the end of Vt is less and the apex will be diluted but in a smaller percentage... so you have two areas of high and low nitrogen and two different pressures, one where airways are not prone to close (the apex) and the others are susceptible to closing if you make the intra-pleural pressure less negative.

if the other airways are stenotic, the airways are prone to close in the base than that of the apex.

when we inhale, pure oxygen in the ADS which is also exhaled (pure oxygen, not nitrogen).

the air in the second phase: gradually increase in nitrogen.

3rd phase: constant level of nitrogen.

in actuality, it is 4 phases...

at the end of expiration, intra-pleural pressure less negative and narrowing --> basal airways close, the air comes purely from apex which has higher nitrogen and so suddenly (phase 4) nitrogen rises up.... therefore the volume difference between 3 and 4 is called the closure volume: after the basal bronchioles close, which should be 10% of the VC, sometimes normal people reach closing volume at the RV: meaning that the last mL is coming from the apex and base.

but at the age of 40 >10%, close to 20% of the VC.

we spoke of airway resistance... now, to talk about the clinical pathologies: COPD (emphysema -- too much air in the lungs and 2) chronic bronchitis)...
- pure airway resistance: bronchial asthma, which becomes chronic and becomes classified as COPD (early stages are not COPD)

- most of the asthmatic patients recover totally (90%) especially when they start with the pathology at a young age.

- emphysema and chronic bronchitis overlap: every patient suffering from emphysema has some sort of chronic bronchitis, but most of the clinical picture comes from emphysema if the initial pathology is that

- emphysema is a very complicated disease because it involves destruction of the alveoli, which causes the destruction of the respiratory membrane, the area available for diffusion (surface area), destruction of the capillaries → destruction of the barrier to be ventilated → increase pulmonary vascular resistance. the area is reduced, resistance is high... increased resistance, you must increase driving force to maintain a normal CO, driving force can be increased by increase pulmonary arterial pressure which normally fluctuates between 8-25 mmHg (diastolic and systolic respectively and the mean is 2/4 of the systolic)... pulmonary vascular resistance is only 1/7th of the systemic vascular resistance and we know that because delta p/ resistance = Q1 = Q2....
  - Q2 = 100/ TPR... calculations.

- so destruction of one capillary reduces the area, increases resistance, increases pressure, increases afterload of the RV, increasing its work... RV will undergo hypertrophy and then dilate....

- RV hypertrophy +/- heart failure (which will eventually develop in its absence) due to lung disease/problem = origin will reside in the lung, regardless of the cause is called cor pulmonale...

- therefore our issue in emphysema is not a pure obstructive disease but a cardiac disease: area available for perfusion is reduced, therefore we must test the diffusion capacity of the lung: how much oxygen is allowed to diffuse from the alveoli to the blood? in this case, the diffusion capacity of the lung is less than normal... increase pulmonary vascular resistance/increased arterial pressure/enlarged RV.