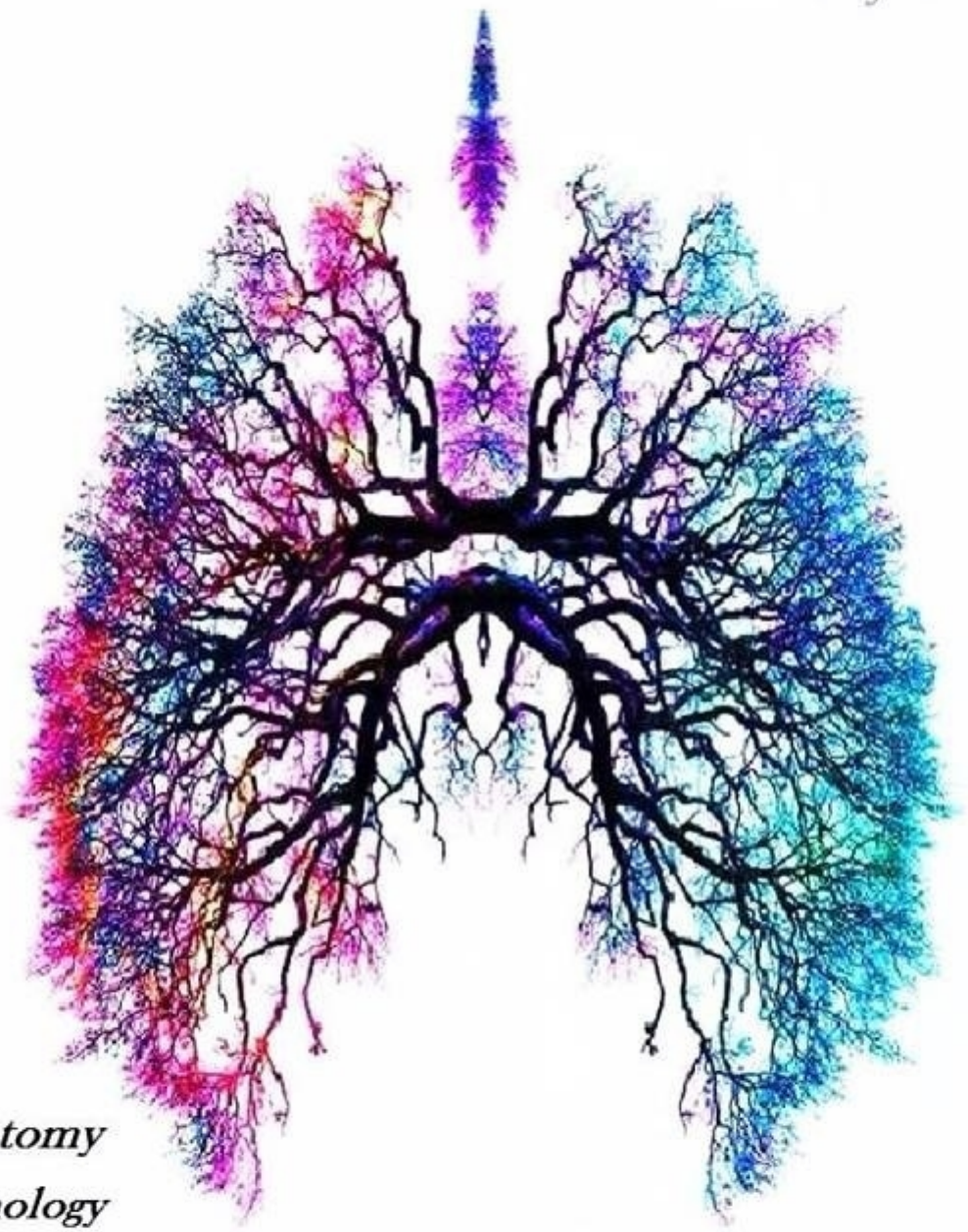
RESPIRATORY SYSTEM

Cover by: Aseil Khatib



- Anatomy
- Pathology
- **Physiology**
- Pharmacology
- Microbiology
- PBL

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Lecture # 2
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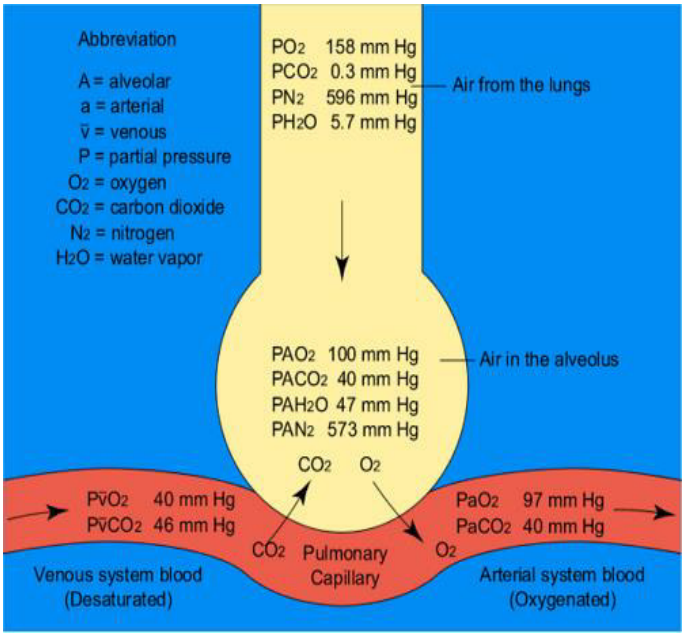
Mechanism of breathing

In this picture we can see the different partial pressures of oxygen in different compartments:



- Outside it's 160 mmHg
- in the anatomical space it's 150 mmHg
- in the alveoli it's 100 mmHg
- in the venous end of the capillary it's 40 mmHg

(The pressure gradient of O2 between the alveoli and the venous end is 60 mmHg)

- in the arterial end it's 100 mmHg (The PCO2is 40 mmHg)
- the intracellular pressure as you know is equal to the venous end.

Now we are going to discuss the mechanics of breathing and how we normally breathe.

- Breathing means the entry of air from outside to inside from atmospheric pressure to alveolar pressure.

- **Tidal volume:** is the normal air flow we take in each time we inhale and it's around 0.5 Liters.

- **Respiratory rate:** We breath 12 times per minute

- **Respiratory minute ventilation = respiratory rate (RR) × tidal volume**

$$= 12 \times 5$$

$$= 6 \text{ Liters}$$

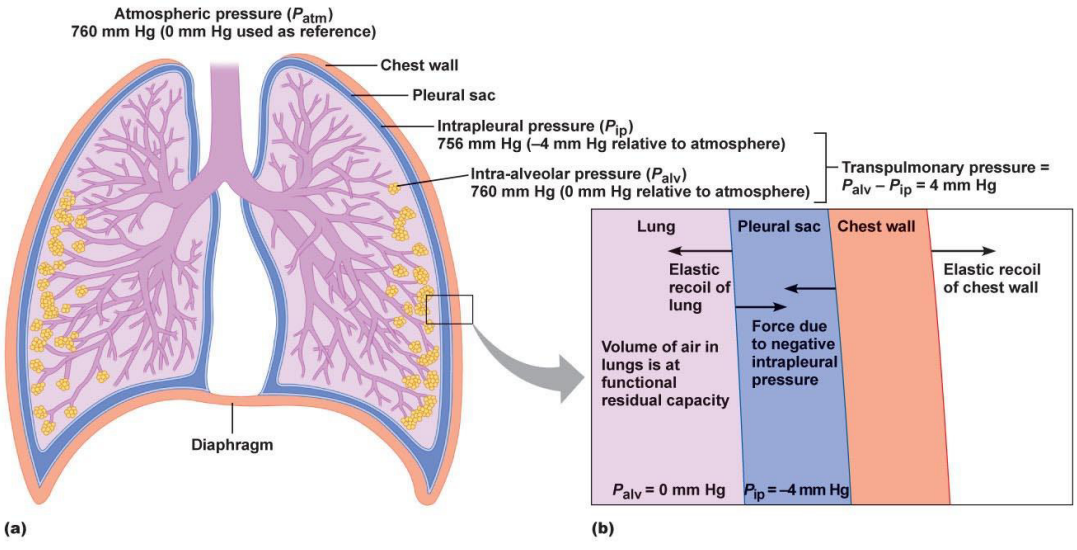
Written by Shaima AL-Haj

Respiratory minute ventilation is similar to the cardiac output which is the product of heart rate and stroke volume (70 beats per minute X 70 mL = 5 Liters and they are almost equal regarding air and blood volume.

In order for flow to occur you always need Ohm's law which says that the flow is directly proportional with the driving force and inversely proportional to the resistance.

- ✓ Flow of charges is a current and the driving force here is the voltage difference.
- ✓ The driving force for the flow of glucose (how many mole of glucose per minute) is concentration difference (ΔC).
- ✓ The driving force for the flow of Sodium (Na) is electrochemical gradient because it's charged and has both electrical and chemical forces.
- ✓ Driving force for flow of blood is the pressure difference between two points.
- ✓ And the flow of air is also controlled by the pressure difference.

Looking at the conditions in this room; the pressure outside the body is zero which is equal to 760 mmHg, the normal atmospheric pressure. (-1) is equal to 759 mmHg, while (+1) is equal to 761 mmHg.



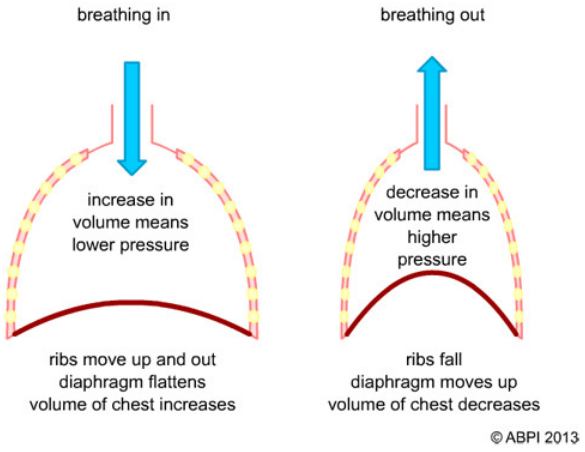
Air flow = (atmospheric pressure- alveolar pressure)/airway resistance
Resistance in the airways starts from nose, pharynx, larynx, trachea and the first 22 divisions until we reach the alveoli, which is the lung.

At rest, the pressure in the airways is atmospheric pressure. Zero – zero = zero so there is no flow of air. To generate driving force you have two options: either you make atmospheric pressure more than alveolar pressure or make alveolar pressure less than atmospheric pressure.

Normally we cannot manipulate the atmospheric pressure so we resort to making the alveolar pressure less than outside pressure. We make it 759 mmHg (-1) which is an enough driving force through the airway.

The lung is like a balloon inside another balloon, the external balloon is the thorax and the internal balloon is the lung, and between them is the plural cavity. Pressure in the plural cavity is -4 mmHg. The lung is a balloon and has an elastic characteristic which is responsible for its recoil tendency that causes collapse by a force equal to +4 mmHg. So to prevent the lung from collapsing we need an opposing force equal to collapsing force but in the opposite direction and this is equal to -4 mmHg (If the collapsing force is +14 the opposing force should be -14 and so on).

This -4 mmHg force can maintain the lung inflated partially and so even before you take in the tidal volume the lung already contains 2.2 Ls. After you take in the tidal volume it becomes 2.7 L , so the tidal volume is 0.5 L.



When the diaphragm underneath the lung contracts, it descends downward making the thoracic cavity larger. Increasing volume decreases the pressure according to Boyle's law, so the pressure that surrounds the lung instead of being -4 becomes -6 mmHg. The lung will automatically inflate because now it is surrounded by more negative pressure.

Again according to Boyle's law: because the lung volume increases, its inside pressure (intra –pulmonary pressure or intra- alveolar pressure) also drops and becomes -1 mmHg. -1 as we said is enough driving force to drive the tidal volume in (0.5 Ls). So the key statement today is that the inflated lung drives air in, it is not the air which inflates the lung (ليس الهواء من نفخ الرئة و لكن الرئة المنفوخة هي التي سحبت الهواء). Which happens first? First the lung inflates, second the air enters, this is the right sequence.

When enough air molecules enter and raise the pressure back to zero and no more driving force is present, inspiration stops. This means that at the end of inspiration the intra- alveolar pressure is zero. (لأنه صار صفر إنتهى)

During inspiration, the intra-alveolar pressure is sub-atmospheric because otherwise no driving force is present to initiate breathing. So our normal pattern of breathing is to make one end of the respiratory tube negative (-1), therefore this is called **negative pressure breathing** and it needs normal contraction of the diaphragm.

If the diaphragm for some reasons cannot contract then we cannot make the events mentioned earlier and we must make the outside pressure more than zero. This +1 mmHg it is enough driving force to allow air flow. How can we make this positive? By introducing a tube or what's called intubation that is connected to a machine called respirator. In the respirator the pressure is positive which pushes air in and then it's brought back to a negative value driving air out. This is called **Positive pressure breathing** which is an artificial breathing pattern and needs a ventilator.

As you remember, before taking a breath there is 2.2 Ls in the lungs, after taking in the tidal volume it becomes 2.7 Ls. On top of this tidal volume a person still can add three additional liters during deep inspiration. This is called **inspiratory reserve volume** (IRV) because it happens during inspiration. The final lung volume becomes 5.7 Ls.

Also before taking in the tidal volume, a person can still can exhale forcefully and empty some of the air in the lungs, this is called the **expiratory reserve volume** (ERV). However, even if you try to empty your lungs some air still remains in it and it's called the **residual volume** (RV). Those volumes differ between old and young people but as an average they're 50-50, meaning the ERV is about 1 L and so is the RV.

Up until now we have 4 volumes that do not overlap:
1- residual volume (RV)
2- expiratory reserve volume (ERV)
3- tidal volume
4- inspiratory reserve volume (IRV)

The addition of 2 or 3 or 4 of these volumes mentioned above results in a new volume and this is what we call **capacity**. The Lung has different capacities according to how many volumes are added (you can add a maximum of 4 and a minimum of 2).

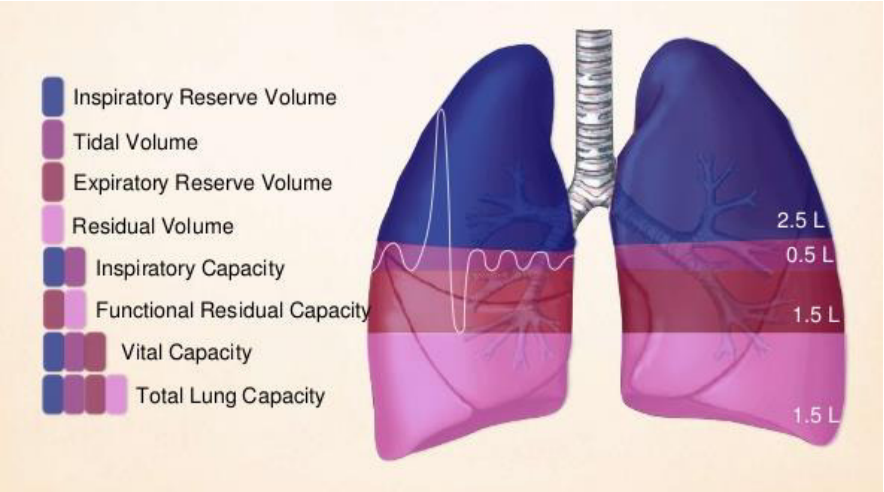
- **Tidal volume + IRV = Inspiratory capacity**

- **ERV + RV = functional residual capacity (FRC)**

- **Vital capacity** is the volume of air which you can exhale forcefully following forceful inspiration, meaning you first fill your lungs to the maximum and second you empty your lung to the maximum. During expiration, you are going to exhale 3 volumes: IRV, tidal volume and ERV.
IRV + tidal volume + ERV= Vital capaity

- **Total lung capacity (TLC)** which is the maximum volume of air that can be taken. This is the addition of the 4 volumes together.
IRV + tidal volume + ERV+ RV = Total lung capacity

So we have 4 volumes and 4 capacities, study this picture to get the full idea.



For air to enter the lungs a driving force needs to overcome airway resistance and this will produce air flow. The desired flow is the respiratory minute ventilation and the driving force is just 1 mmHg which is so small.

If we compare between air flow and blood flow:
Blood flows from the left ventricle (point A) back to the right ventricle (point B) and this happens because of pressure differences. The blood volume to circulate is 5 Ls which is the cardiac output (Q1), and this volume is almost equal to the volume of air going through the respiratory system.

As we said the driving force for blood flow is pressure difference. The mean arterial pressure (point A) is equal to 2/3 diastolic pressure and 1/3 systolic pressure and it's 100 mmHg. The pressure at point B is zero. So pressure difference (Δp) equals 100 mmHg.

This driving force works against TPR which is the resistance the blood is going to face while moving from the arteries to arterioles to capillaries to venules to veins. If you add these resistances you end up with the total peripheral vascular resistance.

So blood flow needs 100 mmHg to overcome resistance while the same flow of air needs only 1 mmHg to overcome the airway resistance, and this means that the air way resistance is just 1% of total peripheral vascular resistance. (High resistance needs high force)

Now we know that the airway resistance is so small and negligible, starting from the nose to the pharynx, larynx, trachea and 22 branches. In all these divisions the resistance is equal to 1% of TPR.

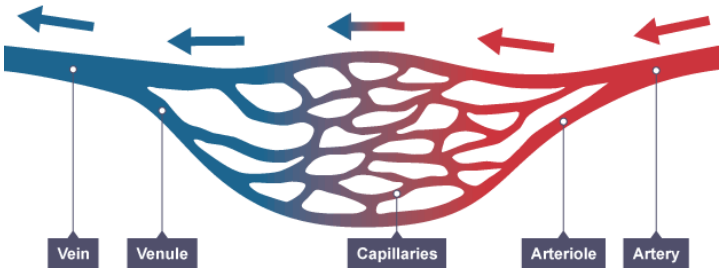
❖ We have 4 take home messages about airway resistance:

1- Air way resistance is so small: and this is evident because you don't feel like forcing air into your lungs when you breathe and you need a very small driving force.

2- Air way resistance resides in large air ways: this small resistance resides where, in the large airways or in the small airways?

For example the vascular resistance mostly resides in the arterioles. How did we determine this? By comparing the driving forces against the vascular resistance while keeping the blood flow through the different vessels equal to Q1. The higher the driving force means the higher the resistance.

Even though a capillary has a smaller diameter than an arteriole, when we compare resistance we consider the whole capillary bed and its cross sectional area and not just 1 individual capillary. The amount of blood in the arteriole divides over the entire bed, and since the cross sectional area of a capillary bed is way larger than that of an arteriole, the resistance in capillaries is less and the driving force in them is less as well.



Now as for the airway resistance:

- 40% of resistance comes from upper airways
- Another 40% comes from the first 7 generations especially in generation number 4.
- 20% from bronchioles.
- 0% from generation number 15 and beyond because of their huge cross sectional area and this means they offer very little resistance.

Note: conductance is the opposite of resistance

***3-* Pathophysiology of the airway resistance comes from small airways:** In case of increased airway resistance up to 10X we need to increase the pressure 10X as well to maintain the same flow. This additional increase comes from where, from large airways or small airways? Any increase in airway resistance comes from small airways because of the following reasons:

- ✓ Large air ways are supported by cartilage which is like a bony material that prevents the structure from being collapsed. The trachea for example will not collapse because of this protection. Cartilage remnants are found up until generation 12. From generation13 and beyond they are left as bronchioles on their own, meaning they have no support anymore.

- ✓ After generation 13, the diameter of the airways becomes smaller and can be easily obstructed. Larger airways like the trachea don't get obstructed by mucous easily unlike the smaller ones.

- ✓ Smaller airways are also surrounded by smooth muscle cells that when irritated they contract they narrow the lumen. This is **broncho-constriction**. An example is parasympathetic stimulation.
- ✓ Since airways are lined from inside with epithelium and goblet cells that secret mucous, this mucous might plug the airways. If the small airways are closed, there is no chance for air to reach the alveoli.
- ✓ If the epithelium becomes inflamed, inflammation causes edema (swelling) which means more narrowing of the lumen. We have so many different potential problems in the small air ways.

4-It is more difficult to exhale than to inhale ,so patients start wheezing during expiration: when a patient is suffering from an increase in airway resistance, is he going to face a problem mainly during inspiration or expiration?

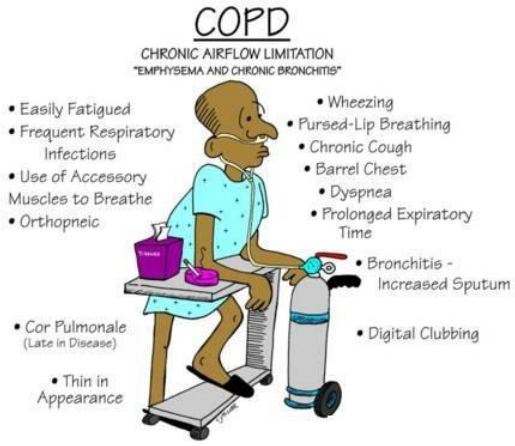
- In normal conditions

<u>During inspiration</u>: the diaphragm descends increasing the volume of the thoracic cavity. The pressure in the pleural cavity goes down from -4 to -6 mmHg. This is an opening pressure, meaning it opens the airways even more. The pressure in the lungs becomes -1 and air molecules go inside until the pressure becomes zero mmHg and this stops inspiration. At zero pressure the lungs have a larger volume.

<u>During expiration</u>: the lungs need to go back to their normal size, so the diaphragm relaxes and ascends decreasing the volume of the thoracic cavity. Less lung volume means the pressure in the pleural cavity increases from -6 to-4 mmHg. This is a closing pressure which closes the airways. The pressure in the lung becomes +1 due to this compression and air molecules go out until the pressure becomes zero again and expiration stops.

- In cases of increased airway resistance

We said flow = driving force (ΔP) /resistance (R), any increase in resistance will increase ΔP. A driving force of +1 mmHg is not enough to push air out of the lungs during expiration. We cannot modulate outside pressure, but we can modulate the driving force and it becomes +10 instead of +1. So you are going to push harder against the lung and pressure in the pleural cavity becomes more positive. Positive pressure is a closing pressure. We already have obstruction due to increased resistance, and during expiration this positive pressure obstructs airways even more and sometimes even closes them.



Patients with such conditions know how to push air out of their lungs without closing their airways. This is called breathing through pursed lips. This breathing technique produces back pressure inside the airways in order to keep them open longer and get air out of the lungs.

Inspiration is **active** meaning the contraction of diaphragm muscles uses ATP. Expiration is **passive** and you don't have to pay for it. However if you have increased airway resistance, **expiration becomes active** and you utilize more energy for it than you do for inspiration. Normal people only utilize 5% of the total ATP in our body to support the respiratory muscles but in cases of increased resistance they spend 25% and sometimes even 80 % of their energy on breathing. This leads to fatigue because there's no ATP for the rest of the body.

The Cardiac cycle is 0.8 seconds long, 0.3 is systole and 0.5 is diastole.

Heart rate is 60/0.8 = 75 beats per min

Respiratory cycle is 5 seconds long, 2 seconds for inspiration and 3 seconds for expiration.

Respiratory rate is 60/5 = 12 cycles per minute