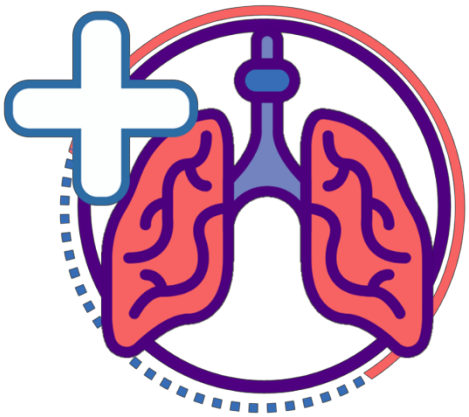

# RESPIRATORY SYSTEM

Physiology

Sheet  Slide

| | |
|---|---|
| Number: | 8 |
| Done by: | Doctor2018 |
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| Doctor: | Yanal Shafaqouj |

*To make a long story short, whenever there is no ventilation whatever the cause was {either bronchial or cardiac defects},* **(V/Q) Is zero.**

## Pulmonary Circulation

### Notes

- pressures in the Pulmonary Circulation are much lower than in thesystemic circulation.
  ***Pulmonary arterial pressure is between 12-15 mmHg, while aortic pressure is100 mmHg. Therefore we can conclude that the pulmonary vascular resistance is between 1/7 or 1/8 of the system vascular resistance.***
- Resistance in the pulmonary circulation is much lower than in the systemiccirculation.
- Pulmonary blood flow is equal to the cardiac output of the right ventricle,which equals the cardiac output of the left ventricle.
- Pulmonary artery pressure(Pa) = CO* pulmonary resistance

CO= 5L /Pa=14

**exercise effects on cardiac output:**

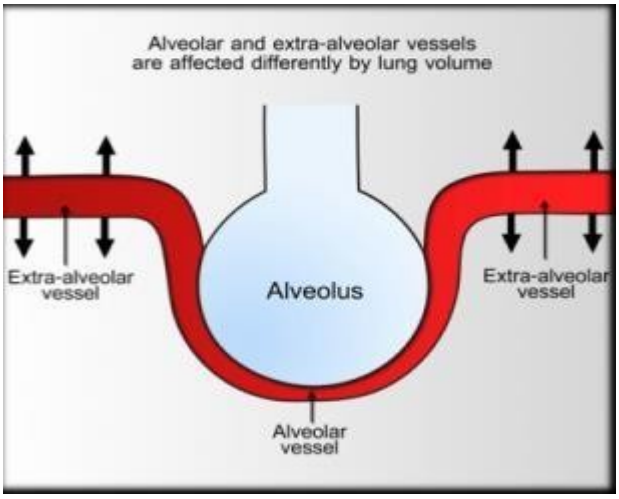
- An individual's blood pressure is represented by this equation →
  **P = Cardiac output * TPR**
  *During exercise the cardiac output increases, five times its normal value per say, yet the pressure remains constant or slightly increases, never ever increases by the same proportion because too much after load imposed on the ventricles will eventually lead to ventricular failure.*
  **The resistance had to decrease almost to the same proportion of the cardiac output increment*
  *two factors played a major role:*
  **a- Recruitment**: by increasing the number of opened capillaries.
  → Normally (2/3) of blood capillaries are closed and only (1/3) is open, during exercise they all open to increase the vasculature to the exercised muscles.

**Distension:** by distending all the capillaries and increasing the rate of flow; Expansion of their walls.

## Alveolar and Extra-Alveolar vessels

*This part of the lecture depends solely on this representation*

Each time you take a breath, the alveoli distend, compressing the part of the vessel that has direct contact with the surface of the alveolus (Alveolar vessel), the two tales of the vessel away from the center are called Extra Alveolar vessels.




So, whenever the pressure inside the alveolus become higher than that in the capillary, the latter will eventually close, and blood flow will decrease.

Whilst the alveolar pressure has increased, the pleural pressure will, by default, become more negative; thus, the extra-alveolar vessels will expand and distend.

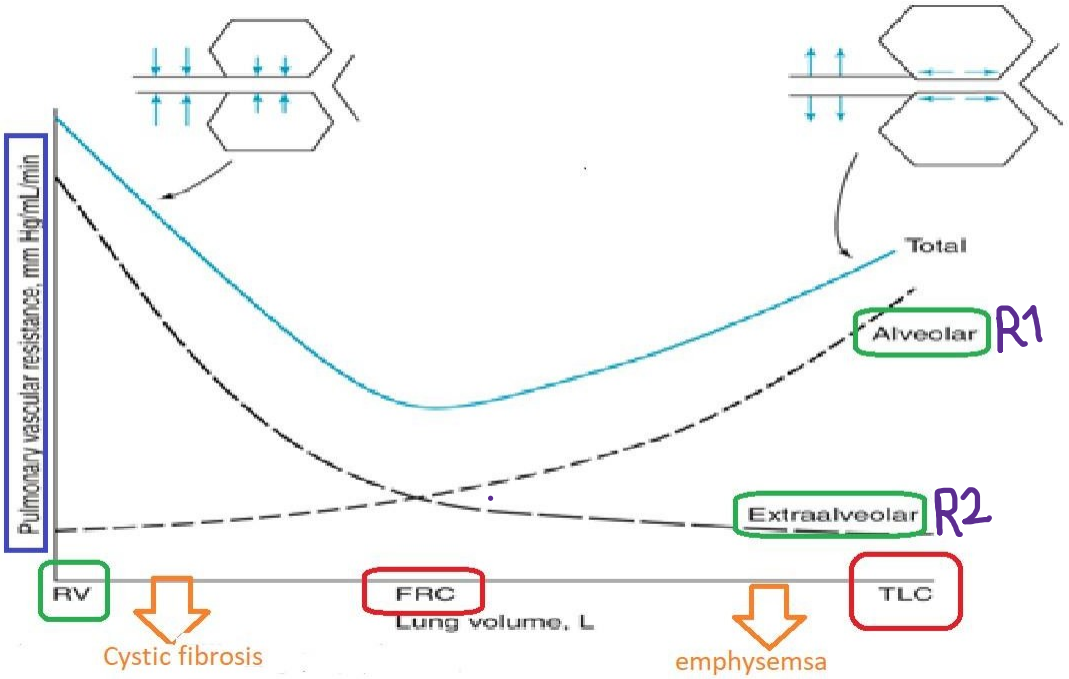
**→ To summarize:**

- alveolar air pressure➡️ compresses the alveolar capillary & distends the extra-alveolar capillary

- *Whenever: P.alveoli > P.capillary ( blood pressure in capillary)➡️ the capillary will be closed ➡️no blood flow*

The figure in the next page is of a great importance, it compares between the alveolar resistance and extra-alveolar resistance, and finally describing the mean pulmonary vascular resistance.

1) + alveolar volume ➡️ more negative pulmonary pressure ➡️ +Load on the heart
2) Total resistance =R1 +R2
3) TPR is lowest at FRC
4) + FRC➡️+TPR & + pleural Pressure ➡️extra load on the heart
5) + pleural pressure ➡️ +R1 / -R2




***What should you learn from this graph?***

1- *When you increase your alveolar volume by stretching their walls and taking deep breath, you will increase the volume, approaching **TLC** "total lung capacity"; thus, increasing the resistance in the alveolar part of the capillary due to higher negativity in pleural pressure, the extra-alveolar parts of the capillary will distend; thus, low resistance.*
2- *If pleural pressure was less negative, the extra-alveolar resistance would increase, while the alveolar resistance would decrease.*
3- *FRC, is the lung volume where the minimal pulmonary vascular resistance resides.*

*Pathological conditions that affect the curve:*

***** In Emphysema, FRC ⬆️ **"can't exhale"** → which increases alveolar Resistance and thus Total Pulmonary Resistance. While extra-alveolar resistance islow.

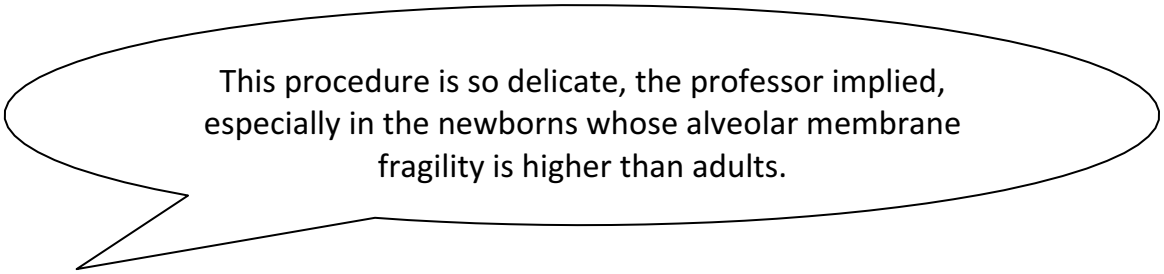
***+ R ➡️+P➡️pulmonary hypertension (>20 mmHg , might reach the systemic pressure)➡️cor pulmonale***

* In pulmonary fibrosis,FRC ⬇️,**"can't inhale"**→extra-alveolar resistance is higher and thus Total Pulmonary Resistance. While alveolarresistance is low.

always keep in mind the pleural pressure, is the hidden third party, contributing the most to what is going on. If it is less negative, -3 for example, the alveolus won't distend، alveolar Resistance is low. At the same time, extra alveolar Resistance will increase because of the compression the pleural pressure exerts on the extra alveolar the walls. On the other hand, if pleural pressure was -8, the alveolus will distend due to increased alveolar pressure, pleural pressure will drop, relieving the compression on the extra alveolar walls, distending as a result.

**Clinical correlation:**

*Positive pressure breathing is used to help the collapsed alveoli to distend. using a machine that changes the atmospheric pressure to positive, consequently developing a pressure gradient, driving air in. However, if the positive pressure was optimal; it would open the collapsed alveoli without affecting the alveolar vessels, but what if the positive pressure increased more?*
*Before it ruptures the wall of the alveolus it would definitely obstruct the alveolar vessel, no perfusion → (V/Q) = ∞.*

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**The flow in the capillaries, is it pulsatile or continuous**?

**Q: What is a Pulsatile flow?**
**A:** it is a pressure which develops a difference between systole and diastole, directly related to the cardiac cycle.

**Q: What is a Continuous flow?**
**A:** it is a pressure which remains the same during systole and diastole, not affected nor related to cardiac cycle.

**Q: So, is the flow in the capillaries pulsatile or continuous?**

**A: <u>the systemic</u>** capillaries pressure is neither pulsatile nor continuous, **it is intermittent**, meaning that on each side of the capillary there are **pre-capillary sphincters** which opens or closes due to local mediators such as O2, CO2, NO, H+ and many others. During exercise, these mediator's concentration changes {either increase or decrease, depending on the mediator} and the sphincter opens → increases flow → decreases resistance.

**So,** at the end of the arterioles and beginning of capillaries damping and disappearing of the pulse pressure starts, thus the difference between systolic and diastolic blood pressure, so called **Pulse Pressure**, is vanished.

**Q: But is this the same case in the capillaries of the lungs?**

**A:** capillaries of the lungs are near to the heart, the main pump, being affected by this short distance; there blood flow is **pulsatile.**

**Having said that the alveolus acquires *<u>systolic (pS), diastolic (pD) and alveolar pressure (pA),</u>* let us see how they are related …**

Pp= pS-pD
Normal Pp= 40
If increases (80 for example ) ➡️Arteriosclerosis
If decreases(20 for example)➡️bleeding ; the SNS is active, BP is normal, + HR.
↪Important in car accidents cases

## What if ?

- **pA > pS > pD (Zone 1) →** there will be *no flow,* alveolar air pressure (pA) is higher than pulmonary arterial pressure (pS, pA) during any part of cardiac cycle, <u>this zone does not exist in human lung normally.</u>

**Pathologically, it may exist; as the apex may be ventilated but not perfused, emphysema( COPD) and hemorrhage are examples. A third case mentioned in the slides is when breathing against positive intra-alveolar pressure, PEEP, we may obstruct blood flow to certain areas of the lung.**

- **pS > pA > pD (Zone 2) →** *intermittent flow,* systolic arterial pressure is higher than alveolar air pressure, but diastolic arterial pressure is below alveolar air pressure. <u>The flow is during systole only.</u>

**This zone exists in human lung at the apex in the upright position. When a person lies down all lung become zone 3.**

- **pS > pD > pA (Zone 3) →** *continuous flow*, pulmonary arterial pressure remains higher than alveolar air pressure at all times, this what happens mostly in our lungs.

**There is blood flow during systole and during diastole. During exercise all lung become zone 3 even the apex.**

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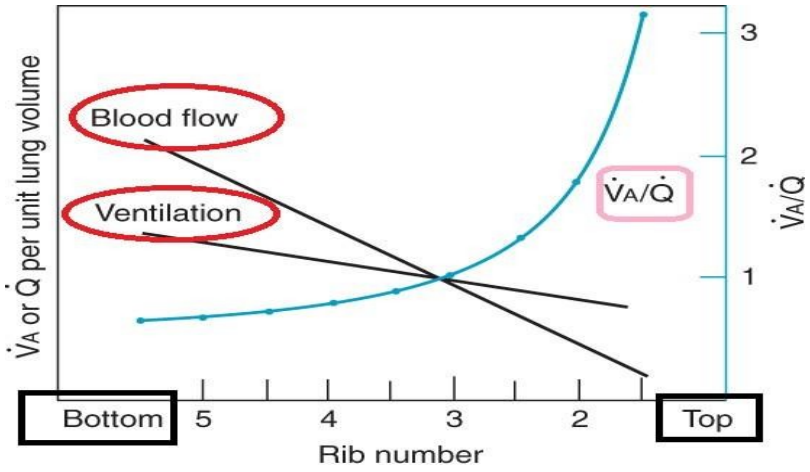
***(V/Q) ratio between the base and the apex is represented in the below curve.***

*Remember:*

*Qbase>Q apex*

*V base> Vapex*

*(V/Q) base < (V/Q) apex*




**Notice how are the variables represented.**
**Bottom=Base**
**Top=Apex**

- When ventilation exist but the perfusion is zero(ex. P.alveolar >P.capillary), the ratio V/Qc= ∞

We have 3 types of capillaries in our body:

a) the most common type ,Filtration at arterial end(20l), reabsorption at the venous end(17l by venous drainage,3l by lymphatic)
b) Only filtration across the entire length of the capillary, no reabsorption, exist in glumerular capillary of the kidney
c) Only reabsorption, no filtration, in the intestinal capillary

BP in different capillary (artery,capillary,vein in order)

a) Most capillaries :40mmHg ➡ 30mmHg ➡ 20mmHg
b) Glumerular capillary:60mmHg ➡ no difference ➡ 59mmHg
c) Pulmonary capillary: between (10-7)mmHg

- Pc= 10 , is an filtration force that pushing the plasma out from capillary
- Πc=28 colloid blood pressure, reabsorption force, due to albumin ( because it has the higher molecular weight, not because it's most abundant)
- If we have 1g of Albumin & 1g of globulin, the no. of molecules of Albumin is higher.

- Pi (hydrostatic pressure)=-5, filtration force
- Πi=14, this is a high no. Which means that proteins CO. Is high & the capillary allows proteins to pass, (in skeletal muscle 2g, in liver 6g because the capillaries are open)

14+ 5+10=29

Πc= 28

Net pressure:29-28= +1 mmHg, it's favoring filtration force; this is to keep the lungs dry because the blood coming from those capillaries is for gases exchange only, but small amount of solution can pass , if this amount increases, a PULMONARY EDEMA will develop ( from interstitium edema to alveolar edema)

Very important note:

- if the surfactant isn't exist ➡️ + surface tension ➡️ + collapsing force➡️ + negative plural pressure to over come the high collapsing force
- In infant, very high surface tension , low r, so more -ve pressure is needed➡️ + fluids sucking into interstitium ➡️ pulmonary edema ( this indicates the importance of surfactant): P=2T/r

If:

1)Pi = - 15 instead of -5 (for ex.) ➡️ filtration pressure = 39 instead of 29( very high)➡️ Net pressure =+11mmHg instead of +1 mmHg

2)Pc= 28 instead of 10(for ex.)➡️ Net pressure =+19 instead of +1

**In the two previous cases, the filtration increases, but no pulmonary edema is occurring; because the lymphatics take care of this (pulmonary edema safety factor)

**Exiting Clinical case ❓** 🚨 (not included)

If a patient had a MI & failure of the left ventricle so the heart can't pump blood➡️+ pressure in left atrium & left ventricle ➡️+pressure in pulmonary veins ➡️+pressure in pulmonary capillaries.

-first thing you should be afraid of is pump failure ➡️ electrical abnormalities(ventricular fibrillation),the patient will die in 2 minutes ☹️. This is due to ectopic pacemakers, you should call an ambulance & give pain killer until he/she reaches the hospital.

-in hospital we Use DC shock ,which do what is called ( over drive suppression) to stop all electrical effects on the heart ➡️ the ECG will show

the heart stop ➡️Na start leaking again ➡️ SA node work again

(The doctor explained until this part in lecture 8)

We have finished our talk about lungs and membranes. The rest of this lecture was a review to blood and hemoglobin.

## Hemoglobin

These numbers are a must to know, the professor said.

- Blood volume equals 7% the of total body weight, 5-L in a 70-Kg individual.
- 5-L = 5,000,000 $\mu$ liters, in each $\mu$ liter there is 5,000,000 RBC's.
- Each RBC possesses 280,000,000 Hb molecules, each capable of carrying 4 Oxygen molecules.
- ***General facts about Hb, review:***

Hb concentration is (14-16) g/dL, each 1 gram of Hb can carry up to 1.34 mLof O2.

**1- Each dL of blood, containing 15 g/dL Hb on average, can carry up to 20 mL O2.**

2- Oxygen solubility in plasma = O2 partial pressure (PPO2) * solubility of O2 (SO2)

***This is called Henry's law.*** = 100 * 0.003

= 0.3 mL /dL blood, so 0.3 mL is dissolved in blood whilst 20 mL is bound to Hb, so the total Oxygen concentration equals to *20.3 mL/dL.*

*3-* **Fe++ (ferrous), binds O2 reversibly. Yet Fe+++ (ferric) binds O2 irreversibly.**

**5-** Hb is a tetrameric protein, 2$\alpha$ & 2$\beta$.

***6-*** *HbA is* $\alpha 2, \beta 2$.

**7- Hemoglobin behaves as an allosteric protein; each binding facilitates the next.**

***8-*** Hb undergoes to different states, *R: high affinity to bind, T: low affinity to bind.*

**9- Bohr effect: If CO2 or H+ binds hemoglobin, O2 will be released immediately, this is the case in the tissues.**

**10-** *Reversed Bohr effect " Haldane effect"* : If $O_2$ binds hemoglobin, $CO_2$ and $H^+$ will be released immediately, this is the case in the lungs.