

Respiratory system physiology



sheet



handout



slides

Number

1

Doctor

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- ✓ Respiratory system works with the CVS and the bone marrow to reach the main target of providing an oxygen concentration in the systemic arterial blood of 20 mL O₂ /dL of blood (20 mL O₂ / 100 mL blood or 20%)
- ✓ The extraction ratio of oxygen by cells (what cells are going to utilize) is normally 25% (5mL/ 20mL *100% = 25%)
Leaving 15 mL (75%) of oxygen in the venous blood to go back to the right atrium , which means that the venous blood contains significant amount of oxygen to start with
- ✓ Hypoxia is decreased utilization of oxygen by cells and it is multifactorial
- ✓ To reach the final target of 20 mL O₂ we need:
 1. Normal hemoglobin that can bind oxygen :[Hb] = 15 g/dL
1g Hb can bind reversibly 1.34 mL of oxygen
(15g/dL * 1.34mL/1g =20 mL/dL)
Hb must be fully saturated with oxygen (100% saturation)
 2. Oxygen availability in plasma to bind Hb and form HbO₂(oxyhemoglobin)
Because oxygen is a gas , it is studied by its partial pressure
pO₂ in plasma must be 100 mmHg in order for O₂ sat to be 100%
Note: If pO₂ is more than 100mmHg , then Hb is already saturated
If it is less than 100mmHg , then here it is another scenario
 3. Hb saturation with oxygen of 100%
100% saturation doesn't guarantee 100mmHg partial pressure.
AND 100mmHg doesn't guarantee 100% saturation
- ❖ If the O₂SAT is 100% and pO₂ is 100mmHg but we don't reach the 20mL/dL this means there is hemoglobinopathies (abnormal Hb : no binding or no releasing)
- ❖ Hb needed is the one that loves O₂ and hates O₂: Hb in the lungs should bind with high affinity while Hb in tissues should bind with low affinity to release O₂

REM: The normal Hb needed is HbA which is the adult type ($\alpha_2\beta_2$)

❖ If someone had Hb of 7.5 , 100% O₂ SAT & pO₂ of 100mmHg then the arterial oxygen concentration is 10 mL/dL (7.5 * 1.34 = 10) not 20mL/dL

This proves that all the three factors together are essential to reach the target of 20mL/dL

❖ The only function of the lungs is to make the PaO₂100mmHg

❖ In case of hypoxia:

- It is not always due to problems in the lungs
- If the PaO₂ is 100mmHg and arterial blood gases (ABG) are normal then the problem isn't in the lungs
- If PaO₂ is less than 100 mmHg then look for the lungs

➤ We need the oxygen inside the mitochondria in the electron transport chain to generate ATP (concentration of oxygen is proportional to amount of ATP produced)

➤ Sometimes oxygen is enough but the respiratory enzymes in the mitochondria are unable to utilize oxygen so we end up with cellular hypoxia (due to presence of cyanide , toxins, g-ve septicemia)

○ We can imagine the lungs as a tube ending with a balloon (airways and alveoli)

○ Alveoli are surrounded by capillaries that come from the pulmonary artery (right ventricle) and return back through the pulmonary vein to the left atrium

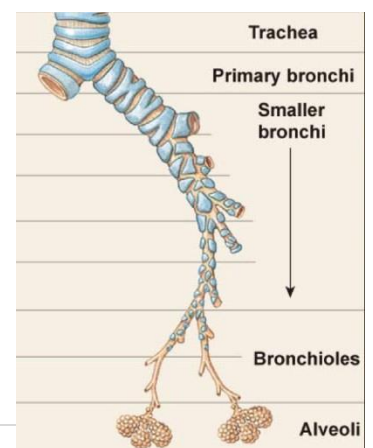
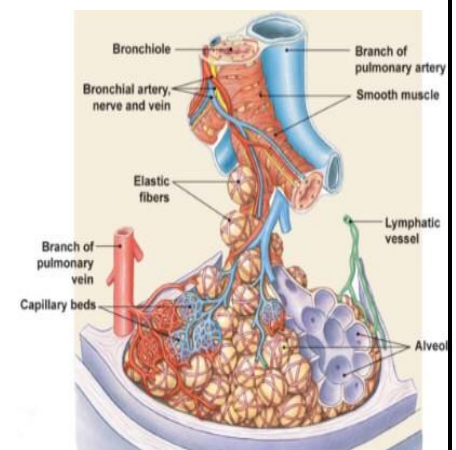
○ Normally we have 300-600 million alveoli

○ Each alveolus is surrounded by a huge network of capillaries that function to exchange gases: O₂ & CO₂

○ Airways start from nasal & oral openings , pharynx, larynx, trachea, right & left main bronchi

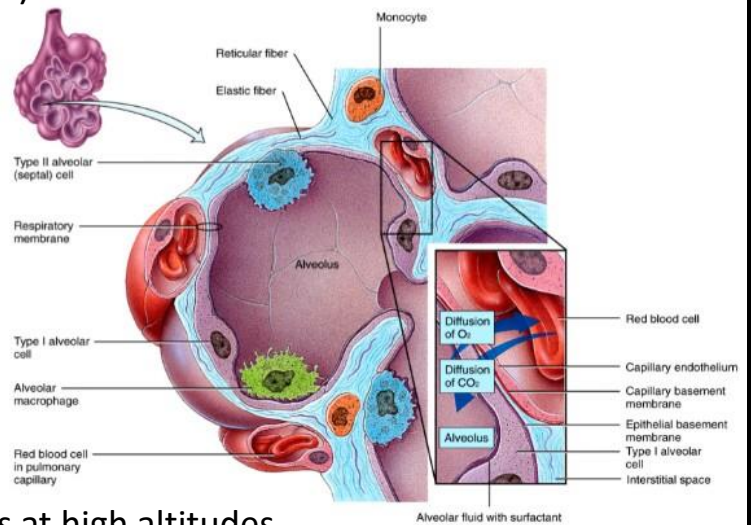
○ Trachea will divide into right & left main bronchi , secondary bronchi, tertiary bronchi ... until we reach 23 divisions (number 23 is the bulb like alveoli , number 0 is the trachea)

This dividing structure is called *Bronchial Tree*



- Oxygen is going to cross the alveolar wall to the capillaries surrounding the alveolus to bind Hb
- In order for oxygen to diffuse , it has to cross the 6 layers of the *Respiratory Membrane* (0.2-0.6 μ m) from inside to the outside:

1. Surfactant (surface acting agent)
2. Alveolar epithelium
3. Basement membrane of alveoli
4. Interstitium
5. Basement membrane of capillary
6. Endothelium



Hypoxia (Potential Causes)

- 1) Oxygen isn't available in the air such as at high altitudes
At the summit of Mount Everest people will die within 2 hours
- 2) Increased airways' resistance (ex: COPD, emphysema, bronchitis)
Airways may be not patent and become narrower and as we know resistance is inversely proportional to the fourth power of the radius
- 3) Rigid and stiff lungs
Inflatable balloon (collapsed) such as in ARDS
In the early stages of a lung disease oxygen is affected
With advanced distractions of the lungs, there will be low O₂ and high CO₂
- 4) Problems in the respiratory membrane
Oxygen can cross any biological membrane as if the membrane doesn't exist
Oxygen availability to the body **isn't diffusion limited** but it is **perfusion limited** (depends on the cardiac output)
CO₂ crosses the membranes 20 times easier than oxygen because it's 20 times more soluble than oxygen and it is also not diffusion limited
The diffusion becomes limited if the membrane is thickened as in pulmonary edema, pneumonia, TB , fibrosis, infiltration of the interstitium
- 5) Anemia, bleeding, heart failure , no mitochondria, cyanide poisoning, septicemia , abnormal Hb , muscular problems (myasthenia gravis)
To inhale, we need to inflate the lungs and we need the muscles like diaphragm which needs a motor nerve to send impulses
- 6) Control problems involving the motor nerves (polio) or suppression of respiratory centers by anesthesia or drug overdose
- 7) Apnea

Breathing In

- Inspiration and expiration are flows and flow is controlled by Ohms law
- Flow is directly proportional to the driving force and inversely proportional to the resistance
- The driving force in the flow of blood and air is pressure gradient
- This force is opposed by airways resistance as if there is an infinite R with whatever was the force there will be no flow
- The more the resistance, the more the driving force needed to maintain the same flow
- The pressure outside the airways is the atmospheric pressure (zero =760) and it's the same as in the airways, so the driving force is zero and even with zero resistance the flow is zero
- Two options to generate a driving force:
 - a. Atmospheric pressure > alveolar pressure
 - b. Alveolar pressure < atmospheric pressure

We can't manipulate the atmospheric pressure but the alveolar pressure can become sub atmospheric (-1)

Our normal pattern of breathing is -ve pressure breathing and this needs muscles contractions

If muscles can't contract, we do an intubation in the trachea and the tube is connected to a respirator (ventilator) where the pressure is made +ve to push air in or -ve to drive air out

This artificial type of breathing is +ve pressure breathing

- Gas particles in the alveoli continuously hit the walls generating a force
- For the air to enter , first lungs need to inflate then the pressure drops

So the inflated lungs drive the air in

When enough air enters the pressure goes back to zero

- **During inspiration the intra-alveolar pressure is sub atmospheric and at the end of inspiration the pressure is zero**
- The driving force is small and the resistance in the airways is small and negligible