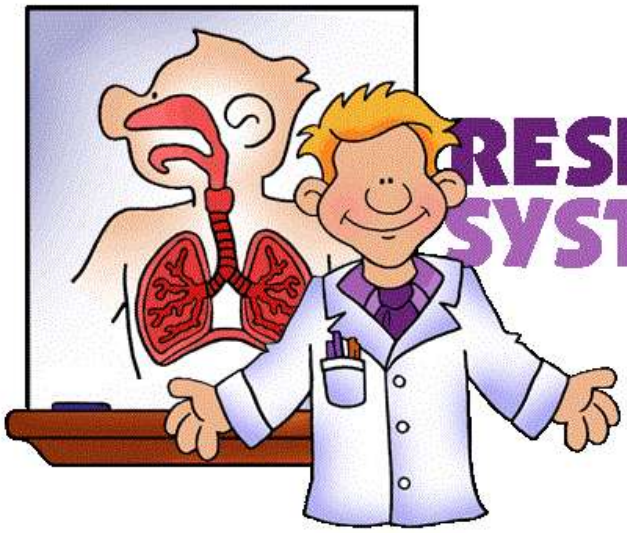
Physiology Team 432



1th & 2th Lectures

General Notes

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Lecture one

Notes:

-alfa1 anti-trypsin acts as an antagonist to photolytic enzymes released by the bacteria that's why it has an immune function

- the MAIN goal of respiration is to 1-get o_2 and remove co_2

-the conductive zone doesn't contribute to the gas exchange process

-acid base status = PH levels (H concentration)

-the internal and external gas process happens as follows :

You take up the o_2 from the alveoli to the pulmonary capillaries so that they will be carried from the pulmonary vein to the left atrium from there it goes into the left ventricle and into the aorta which carries the arterial blood (oxygenated blood) into the tissue (as aorta goes to the tissues it becomes smaller and smaller to give you systemic capillaries and there o_2 is taken by the tissue and they release co_2 . now the systemic capillaries will take the venous blood (deoxygenated blood) and start growing till its gives us the superior and inferior vena cava's which pour the blood to the right atrium . this blood goes to the right ventricle where it enters the pulmonary artery that will take the blood to the lung. There it becomes pulmonary capillaries , the gas exchange between the alveoli and the capillaries happens and cycle repeats itself.

From this notice that the pulmonary arty carries venous blood and the pulmonary vein carrier arterial blood, also know that

1-**external exchange** is between the PULMONARY CAPPILARIES and the ALVEOLI

2- **internal exchange** is between the SYSTEMIC CAPPILARIES and TISSUES

-The internal exchange is a metabolic process that's why we don't discuss in physiology

-external exchange is divided into three processes (go to the slides)

-The alveoli has 3 imp cells The type 1 alveolar , type 2 alveolar , dust cells (respiratory macrophages)

- the type 2 alveolar cells release the surfactant which is very imp in maintaining the alveoli and preventing their collapse cause it controls the surface tension which has a tendency to collapse the alveoli and the tension becomes stronger when there is air .

The respiratory membrane is made up of:

- Alveolar lining
- Interstitiaum
- Endothelial lining of capillary

Surfactant has three main functions:

1- Prevent alveolar collapse.
2- Decrease the airway resistance (meaning tthat passages aren't tight and the air can pass easily through).
3- Decrease the work of breathing (you don't have to perform a string effort to breathe).

- In case of infant RDS the treatment in via glucorticosteriod and cortisone which are known to have an effect on increasing the surfactant synthesis

Lecture two

Notes:

Ventilation is the process of inspiration and expiration (inward outward movement of air) between the lung and the atmosphere

-Inspiration is an active process (muscles are involved)

- In resting state you have diaphragm (mainly) and the external inter costal
- In the forced state you need more support coming from the scalene, pectoralis minor and sternocleidomastoid.

-Expiration is a passive process in resting state for no muscles are involved it is the simple action of the contracted muscles returning to their position, where are in forced it will be active for we have

- Internal intercostal (rib depressors)
- Anterior abdominal muscles (the one that help in pushing the diaphragm upward to its original position

- To explain the process of inspiration and expiration know that the air moves from where the pressure is high to where the pressure is low that's why:
    1- When you **inspire** it means you want air into your lungs , so pressure in your lungs has to be low and that's why its -1 (you get this value from the action of muscles contracting increasing the volume and lowering down the pressure which is the intra alveolar pressure)
    2- The opposite happens when you **expire**, you want the air out of your lung meaning the pressure in has to be higher. this is helped by the action of muscles relaxing and lowering the volume and increasing the pressure to +1 (more that the atmospheric) and pushing the air out

- In the phase between two breaths (atm pressure= intra alveolar pressure=0) and after inspiration intra alveolar pressure =0
- There is also another pressure when you ventilate which is the intra pleural .this one is the one that happens in the pleural cavity and like the intra alveolar when

you inspire it decreases to -7.5 (at resting state its -5) and when you expire it rises up a little.

- Trans pulmonary pressure can be called:
  - Distending
  - Expanding
  - Recoil – called this not because it recoils (on the contrary it prevents the recoils of the lung) but because you can calculate the force of lung recoil by understanding that there is the opposing transpulmonary pressure which has the same value as recoil force
- When the volume of lung increases the tendency to recoils increases (just like an air filled balloon the moment you let go and not close it ,it will recoil .
- Compliance 200 ml /cmH_20 when the lung is out of your body when in the body its 110 ml /cmH_20
- Emphysema is a chronic disease that happens with smokers it leads to destructions of the alveolar septum that has elastic fibers. these fibers contribute to recoiling of the alveoli and decreasing their volume and thus the lungs , but when they are destroyed they cant recoil and remain expanded and volume increases .

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✓ **These notes are up until the ventilation part and diffusion is not in.**

✓ **Lecture three and four are really clear on the slides and all see said is in the slides.**

Thank you Lama for sharing your Notes ..