PASSION ACADEMIC TEAM YU - MEDICINE

Sheet#1 Physiology

Lec. Date :

Lec. Title : Introduction to respiratory system

Written By : Mustafa Tawaha

If you come by any mistake , please kindly report it to shaghafbatch@gmail.com

RESPIRATORY SYSTEM

Respiratory System

❖ RESPIRATORY FUNCTION

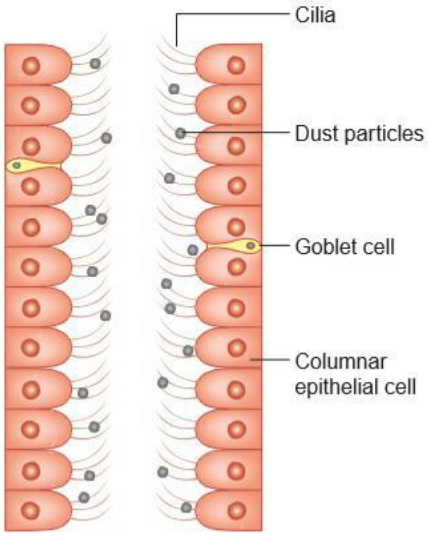
This is the main function of the R.S, exchange of gases (O_2 and CO_2) between the external environment and the body tissues (for respiration).
*although both important, but removing CO2 is more important

❖ NON-RESPIRATORY FUNCTION

1. Defense mechanisms:

✦ The R.S tissues are able to produce **IgA which has a defense mechanism** against the respiratory infection.

✦ **Ciliary escalator action**: How?



If the inhaled particles is:
1-bigger than 10 Mm→ trapped in the nostril by nasal hair and mucous
2-10-2 Mm→ trapped in mucous and eliminated by **Ciliary escalator :** most of the component of the R.S are lined with ciliated epithelium (MOST of them not ALL). for example, trachea and bronchioles have ciliated epithelium, these cilia are able to get rid of the unwanted inhaled particles (toxic substances, pathogens) which are firstly entrapping by the mucus membrane, then these particles will be removed by these cilia by pushing them upward to the pharynx, where they will be either swallowed or expectorated, it will not reach the respiratory zone which is a critical zone and most of its components are not ciliated.
3- less than 2 Mm→ pulmonary alveolar macrophages can defend the body against the inhaled particles. Once these macrophage become fully occupied with these inhaled particles they migrate upward to trachea

2. **Reservoir for the left ventricle**: pulmonary vessels are highly compliance, they can store about 0.5 Liter pumped from right ventricle.

This mean when they store this amount of blood, the pressure in them still the same.(it is useful because it doesn't allow for edema make the distance between alveoli and vessels longer→which affect diffusion process)
Also, to avoid increasing in pressure in pulmonary vessels: there are capillaries called **recruitments** that are closed at rest ,but they open when the cardiac output increase(like in exercise)→ pressure remain the same.

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when the left ventricle output is more than venous return, this shortage in venous return will be compensated by sending more blood from the pulmonary vessels.

Shortage in venous return there's no complete filling in ventricles
This is lead to shortage in cardiac output.
But this does not happen because of the blood reservoir in the pulmonary vessels which will compensate this shortage.
3. **Pulmonary circulation acts as a filter that's able to filtrate any waste product in venous return**. Blood returns back to the lung from the tissues crossing the right side of the heart, this blood filled with air bubbles, clots and detached cancer cells, all these will be filtered in pulmonary capillaries before reaching the left ventricle; because for example, if an air bubble reaches the left ventricle it will be ejected through the systemic blood, where it could occlude certain vessels, which can lead to infraction.
Q:why these clots are not harmful for pulmonary circulation unlike systemic circulation?
In systemic, it will cause hypoxia. Unlike in pulmonary which have **direct contact with O2 in <u>respiratory zone</u>+ bronchial circulation** that innervate **<u>conducting zone</u>** .So, it doesn't need O2 from the pulmonary circulation.
يعني حتى لو سكرت الاوعية في عندنا شيئين احتياط بعوضن حاجة الانسجة للأوكسجين

<u>Other explanation</u> :Air bubbles will be taken by alveolus and exist as air, clots will be lysed by fibrinolytic system in the epithelium cells of

pulmonary capillaries. Small clots are not harmful to alveolus because it does not need O_2, BUT if it's a large clot many problems will happen in pulmonary circulation and it can lead to edema.)

4. **Removal of fluid from alveoli**

5. **Role in absorption of drugs:** like anesthetic gases which are absorbed by pulmonary tissues and bronchodilator drugs.

6. **Conversion of angiotensin I to II** by producing angiotensin converting enzyme (ACE) which is mainly produced by epithelium tissue of pulmonary capillaries.

7. **Surfactant** production: it's very important in inspiration, it decrease the surface tension, **thus preventing alveolar collapse**.

8**. Inactivation partly or completely of many vasoactive substances present in the blood** like bradykinin, acetylcholine, norepinephrine, serotonin, prostaglandin and others

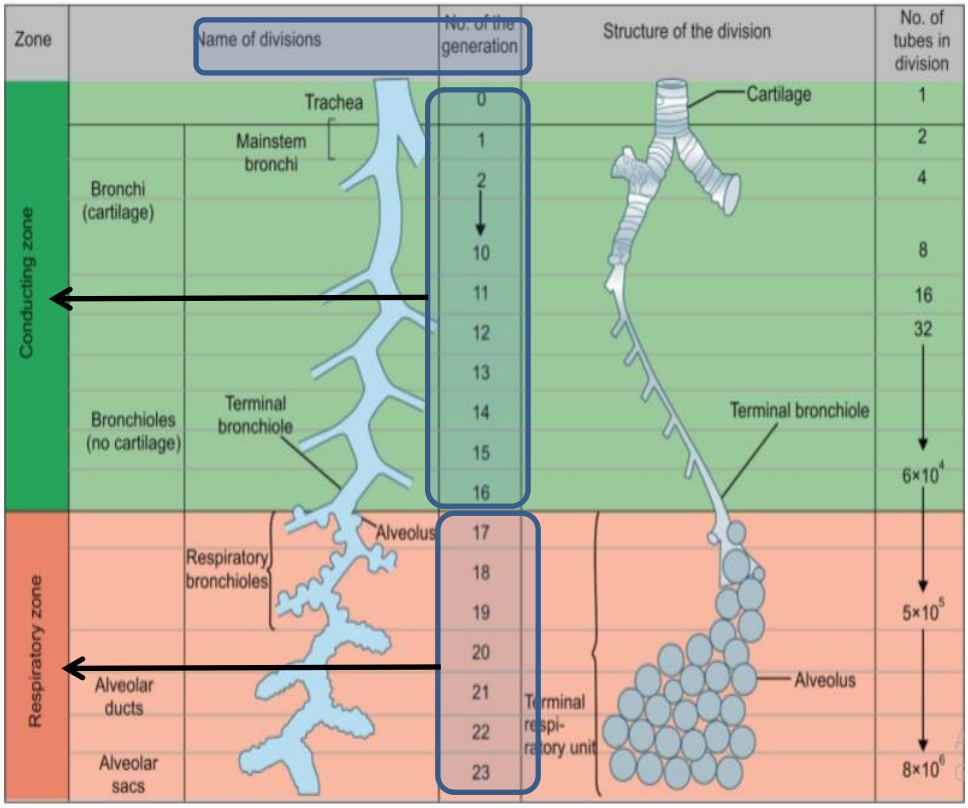
STRUCTURE OF THE RESPIRATORY SYSTEM

-respiratory system divides into 2 Anatomical parts of respiratory system:

1-upper respiratory tract

2-lower respiratory tract

Division=generation



2- Physiological parts of respiratory system:

☯**Conducting Zone**: warm ,filter , moisture and conduct air

***we need to warm the air** because As blood temperature decreases ,the solubility of gases in blood increase . When the amount of a dissolved gas exceeds the limit of its blood **solubility**, the gas molecules join in aggregates which form **bubbles** in the water this forms air bubbles that form air emboli which damage the tissues.So warming air in conclusion is to protect tissues

-These components have ciliated epithelium cells -importance in defense- and have smooth muscles which receive sympathetic and parasympathetic innervations.

✓**Sympathetic** innervations act on **β2** receptors and induce bronchodilator. β_2 receptor mostly affected by epinephrine which is a hormone secreted by adrenal gland.

✓**Parasympathetic** innervations act on **muscarinic** receptors which induce bronchoconstriction.

*We have another neurotransmitter :**VIP**(non-cholinergic and non-adrenergic) cause bronchodilation like sympathetic neurotransmitter.
The tone of these muscles is maximum in the morning, while in evening it's not, so in morning there's bronchoconstriction and in evening bronchodilation.
Side note: bronchial circulation drain the deoxygenated blood into the pulmonary vein
So pulmonary vein doesn't have 100% full oxygenated blood

☯**Respiratory Zone**: the cilia start to disappear here "not ciliated" and they get rid of inhaled particles (pathogens) by alveolar macrophages. Once macrophages are completely full with these

particles, they will migrate upward to pharynx where they will be swallowed or expectorated. Also they have no smooth muscles

✓ **Dead space**

a. Anatomic dead space

-is the volume of the conducting airways.

-normally approximately 150 mL.

b. Physiologic dead space

-is a functional measurement.

- defined as the volume of the lungs that does not participate in gas exchange.

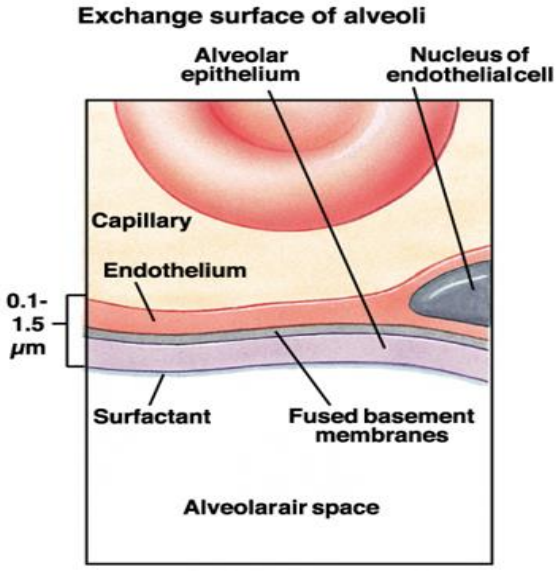
-is approximately equal to the anatomic dead space in normal lungs.

-may be greater than the anatomic dead space in lung diseases in which there are ventilation/perfusion (V/Q) defects

◆Wasted air=Dead space

◆Wasted blood= shunt= blood that reach alveoli but don't take O2 and come back deoxygnated

The alveoli



The alveolus consist of three main types of cells:

1. **Pneumocytes I** (alveolar type I cells).
2. **Pneumocytes II** (alveolar type II cells).
3. **Alveolar macrophages**.

✞ Pneumocytes I & II form the wall of the alveolus and their numbers are the same(ratio 1:1). But 94% - 98% of the surface area of the alveolus covered by pneumocyte I, while 2% - 6% covered by pneumocyte II. This is because :

Type II is found as clusters due their function: synthesis of Surfactant ,while type I is found as separate cells due their function in diffusion of gases

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✓**function** of pneumocyte I: it's the cell through which the gases exchange occur, because of this function the cytoplasm is very thin and that reduce the diffusion distance, while if it has a thick cytoplasm the diffusion distance for gases exchange will be long; so there's no efficient gas exchange.

Also the basement membrane for pneumocyte I and the epithelium of pulmonary capillaries is very thin.

These basomembrans, also known as **respiratory membrane**, are very thin about 0.5µ in thickness, this makes gas diffusion highly efficient. sometime, the basement membrane of type I cell and the epithelium of pulmonary capillaries fuse together, these have no interstitial fluid at all, so the fuse together, as it’s clear in the figure.

✓ pneumocyte II cells have two major **function** :

1. Surfactant secretion which is a mixture of phospholipids and lipoproteins.

2. Differentiate into type I cells during early stage of gestation. Only type II cells present in early stage in the alveolus because the lungs are not functioning, there's no gas exchange, but in the late gestation period type II cells differentiate into type I cells.