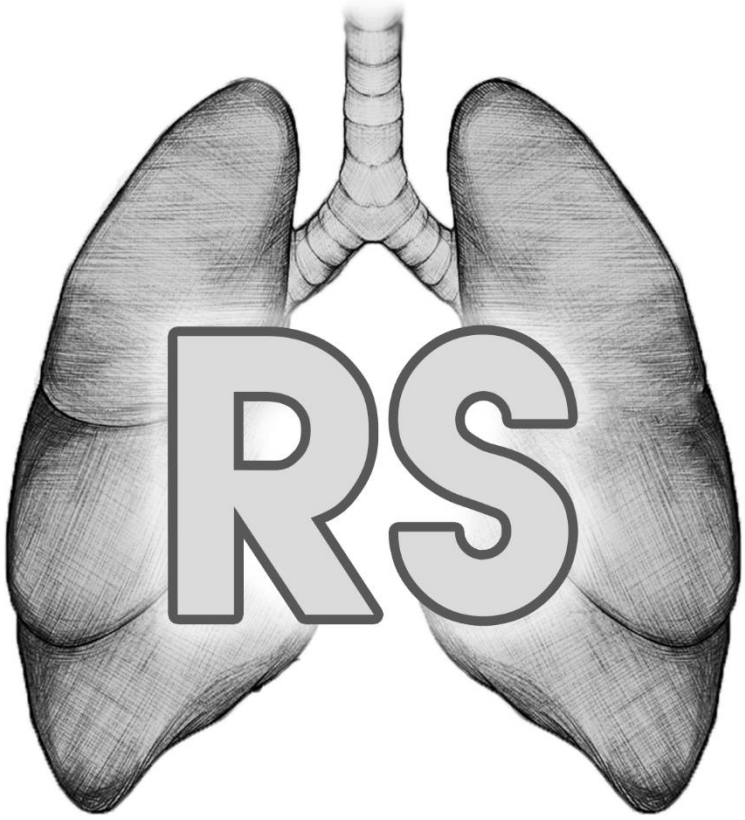

PHYSIOLOGY

☑ Sheet ○ Slide ○ Handout

Number

3

Subject

Airway Resistance and Emphysema

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بسم الله الرحمن الرحيم

This sheet was written according to the recording from section 1&2.

Review:

- The lungs are elastic, like a balloon, and to inflate them you need to apply force. However, no force is required for the relaxation of the lungs so they recoil back to their resting state just by the removal of the force used to inflate them.
- The lungs have a functional residual capacity (FRC) of 2.2 liters.
- The lungs can collapse because they are, again, like a balloon. To prevent them from collapsing, an opposing force is needed. The force is manifested in the negative intra-abdominal pressure. This force keeps the lungs inflated despite the fact that the airways are still open to the outside air.
- If this negativity is removed by a stab wound or a gunshot in the chest, the air will enter the pleural cavity causing **pneumothorax**.
- In pneumothorax, the pressure becomes 0 or positive. Therefore, the lungs will collapse to their resting volume. Resting volume is the volume of the lung at which the lung has no collapsing tendency. It is also called minimal volume. It is usually 150 mL.
- Minimal volume concept is mainly applied in forensic medicine. If a newborn dies after taking the first breath, pieces of the lung obtained during autopsy will float. However, if a newborn dies before taking the first breath (stillbirth), pieces of the lung obtained during autopsy will not float.

Airway Resistance:

Respiratory Minute Ventilation is similar to Cardiac Output in CVS

- Flow is the volume of air entering or leaving the lungs per minute.
- **Respiratory minute ventilation** = Tidal volume × Respiratory Rate
  = 0.5 × 12 = **6 L/min**
- The respiratory minute ventilation is very close to the cardiac output (5 L/min).
- The respiratory minute ventilation is indirectly proportional to airway resistance, while cardiac output is indirectly proportional to total peripheral resistance.
- *Comparisons between the cardiovascular and the respiratory systems are very important because they are interconnected and hence a disease in one system will cause a problem in the other. For example, left heart failure results in pulmonary edema. Chronic obstructive pulmonary disease (COPD) results in right heart failure.*
- Ohm's law states that flow is directly proportional to the driving force (ΔP; linear difference between atmospheric and intrapleural pressure) and indirectly proportional to resistance (airway resistance).
- When there is great airway resistance we need too much ΔP to keep flow constant.
- The more airway resistance the more the driving force and not the opposite.
- The resistance is very difficult to measure in a biological specimen (like the human body).
- How is resistance measured in humans?
  Indirectly by knowing how much ΔP is needed. If airway resistance is high, ΔP would be high too.
- The pressure difference between the outside air (atmospheric) and the intrapleural pressure (in the lungs) is just **1 mmHg.**
- In the peripheral circulation, total peripheral resistance is the sum of resistance in arteries, arterioles, capillaries, venules, and veins. They are connected in series. Resistance is not calculated by adding the resistance of each part of the circulation. Just like in the respiratory system, resistance in the cardiovascular system is calculated after figuring out the pressure difference (in the cardiovascular system it is **100**).
- Vascular resistance is 100 times more than the airway resistance. However, there is a difference in the fluid (in the vascular system the fluid is blood which is mainly water and in the respiratory system the fluid is air).

*The lung and the heart are connected, which means lung diseases affects the heart and heart diseases affects the heart

1st take home message: Airway resistance is small and negligible because we need very small driving force to overcome it

- Does the airway resistance manifest in the large airways or the small airways?..

First let's understand these notes:

1. Resistance is inversely proportional to the fourth power of **radius** ($\mathbf{r^4}$).
2. The area of the circle (**A**) = πr^2.
3. The more the **cross-sectional area** the less is the resistance. In the vascular system, the cross-sectional area of the all capillaries is much more than the cross-sectional area of the arterioles and that's why arterioles are the major resistance vessels. To determine the cross-sectional area, use velocity.
   Velocity = flow/area
   The smaller the area is, the greater the velocity becomes.
   For example, the velocity of blood flow in the capillary is the slowest because of the huge cross-sectional area.

2nd take home message:

Because of the huge cross sectional area of the 15th generration and beyond, the airway resistance in them is almost zero and air velocity is low

4. Fractions of airway resistance under normal conditions:
   a. 40% resistance above the larynx. (nose mouth pharynx)
   b. 40% resistance in the first 4-5 generations of the respiratory tree (refer to table below).
   c. 20% resistance in the rest of the respiratory tree (but from the 15th generation and beyond there is zero resistance).

	Name	Division	Diameter (mm)	How many?	Cross-sectional area (cm^2)
Conducting system	Trachea	0	15-22	1	2.5
	Primary bronchi	1	10-15	2	↓
	Smaller bronchi	2	1-10	4	
		3		↓	
		4			
		5			
		6-11		1×10^4	↓
Exchange surface	Bronchioles	12-23	0.5-1	2×10^4 ↓ 8×10^7	100 ↓ 5×10^3
	Alveoli	24	0.3	$3\text{-}6 \times 10^8$	$>1 \times 10^6$

5. Therefore, **under physiological conditions most of the airway resistance resides in the large airways**:

- If we face more airway resistance, the negative pressure inside the alveoli will become more negative to drive air in (increase pressure gradient).

- This increase comes from small airways because the large airways are supported by **cartilage** which prevents them from collapsing. The cartilage is present in up to **10th** generations in the respiratory tree. The bronchioles after the 10th generation are not covered with cartilage.
- Small bronchioles(generation 10th-11th and above) are not supported by cartilage "collapsable" but they have smooth muscles surrounding them. Contraction of the smooth muscles "by irritants like pollens" will cause bronchoconstriction, which closes them
- Bronchioles(*which are 1mm of diameter)* are so small that any Mucus plugs (water and protein) can close them. Mucus Plug will not obstruct the entire lumen of the trachea.

3rd take home message:

In pathological condition airway reistance increases, this increase comes from the small airways because the large airways are supprorted by cartilage which makes them non collapsable in addition because it's large any mucus plug will not block the lumen unlike the small bronchiloes

- Small airways contribute to increased airway resistance because they have:
  1. Small **lumen**
  2. Small **diameter**
  3. **Smooth muscle cells**
  4. No **cartilage**
  5. **Goblet cells** which secrete mucus. If these cells are irritated (by smoking), more mucus will be secreted which will cause an obstruction.
  6. **Thinner epithelium** so inflammation in the submucosal layer will cause obstruction too.

Mucus is protein + water

Water is reabsorbed which leaves the solid dry protein as a plug that cannot be removed easily by coughing

- Which is more difficult after an increase in airway resistance, inspiration or expiration?
  1. The pleural cavity not only surrounds the alveoli, it also forms an atmosphere for the bronchioles.
  2. During **inspiration**, intrapleural pressure becomes more negative thus causes the opening of the bronchioles.
  3. During **expiration**, intrapleural pressure becomes less negative "or positive if increased to much due to obstruction" thus permits the closure of the bronchioles.
  4. In partially obstructed bronchioles, more effort is needed for expiration. The patient will produce wheezing sounds due to turbulence in the obstructed bronchioles. *Note: wheezes are bilateral because the obstruction is on both sides*

4th take home message:

Which is more difficult to inhale or exhale, When bronchiloes are Partially obstructed?

- patient faces more difficulty during expiration rather than

TESTS FOR AIRWAY RESISTANCE:

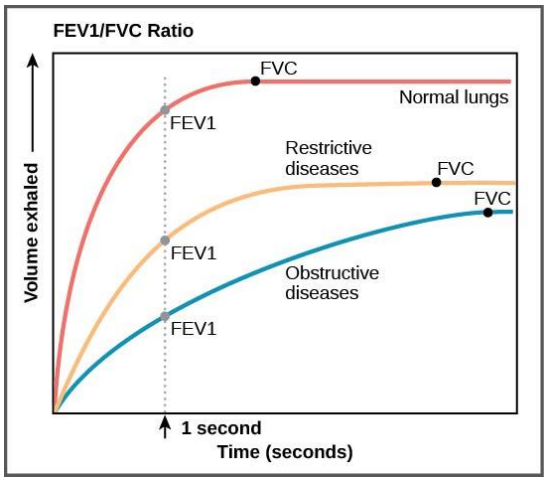
Most of the tests are done during the expiratory maneuvers because the problem resides in expiration! *"patient is able to fill the lungs but face a difficulty in exhaling the air (barrel chest) which results in high TLC ~ 7-7.5L"*

PFT:

- Pulmonary function tests (**PFT**) are done to determine the degree of airway resistance (or to identify whether there is increased airway resistance or not).
- We ask the patient to fill his lung to full lung capacity then to exhale through the device as much as he could to measure the **F**orced **V**ital **C**apacity **FVC**…

- **FEV_1/FVC** Test:
  1. The patient is asked to fill his/her lungs to the maximum (6 L; total lung capacity).
  2. Then, the patient is asked to empty his/her lungs forcefully as quickly as possible.
  3. Suppose the patient expired 5L of air. The forced vital capacity (FVC) is 5L.
  4. The FVC is usually produced in less than 5 seconds. However, if FVC is produced in 5.5 seconds, it is not a big deal.

- A normal person can exhale 80% of the vital capacity within 1 second. This is called the forced expiratory volume (FEV). Forced expiratory volume in the first second (FEV_1) is 4 L (assuming that the vital capacity is 5 L), While abnormal person may exhale 3L (60%) *curve is shifted downward to the right.*
- This is the observed FEV_1 and FVC, The predicted FEV_1 , FVC is determined using the patient's history (age/gender/weight/etc…).



- The observed FEV_1 is divided by the predicted FVC and multiplied by 100 to give the ratio of FEV_1 /FVC "generally FEV_1/FVC * 100"

- This will differ from one patient to another due to differences between individuals.

- Results far below 100% or significantly exceeding 100% are alarming:

1. Above 80% is normal.
2. Between 60%-79% indicates mild COPD.
3. Between 40%-59% indicates moderate COPD.
4. Below 40% indicates severe COPD.

FEV1/FVC % is used to determine the severity of COPD

- FEV1/FVC % does not only indicate obstruction, it also indicates the stage (severity) of the obstruction (just like in heart failure).
- Vital capacity for patients with obstruction is greater than that of normal individuals because expiration of air is difficult.
- Vital capacity can be divided into 3 parts:
  1. First 25% is <u>easy</u> for the patient with obstruction and the normal individual to exhale.
  2. The last 25% is <u>difficult</u> for both the patient with obstruction and the normal individual to exhale.
  3. The middle part which is the remaining 50% (25-75%)

❖ If we took the middle part **"mean expiratory flow rate"** (to remove dilution effect of the first 25% of vital capacity, and to remove the last "hard to exhale" 25%), and took the FEV1/FVC ratio from it the test will be more **sensitive**, for example initial result was 83% and after using the "mean expiratory flow rate" it became 70%! which is not that big of a difference between the two readings yet still more specific,

❖ So we cut the first and last quarters because the first quarter is "سهل" and the last quarter is "صعب" and we end up with the middle more sensitive part (25-75% (2.5L) of vital capacity)

❖ **Closing volume** is more sensitive than the mean expiratory flow rate (and this will be discussed in the coming lectures). *After the ventilation perfusion relationship*

So the "take home" messages again are:

1st: airway resistance normally and physiologically is small amd negligible

2nd: this airway resistance resides in the **large airways**

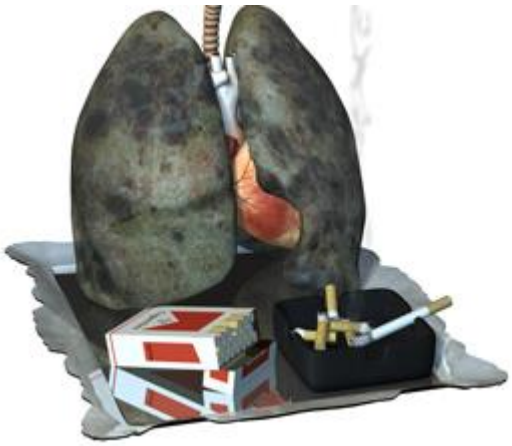
3rd: in pathophysiology the increased airway resistance comes from the **small airways** not the large airways because of the 6 factors previously mentioned

4th: patient with increased airway resistance face more difficulty during **expiration** rather than inspiration

COPD diseases that we care about in physiology are 2:

a)**Emphysema** b)**Chronic Bronchitis**

Emphysema:



- It occurs mainly due to smoking.
- Smoking inhibits anti-trypsin, anti-elastase, and anti-protease. Therefore, the walls of some parts of the lungs are destroyed resulting in bulging or hyper-inflated areas in the lungs.
- Walls of alveoli are destructed so surface area for diffusion will decrease.

- Elastic fibers in the bronchioles are tense to keep the airways open. In emphysema, elastic fibers are destroyed so the airways close. This is an example of obstruction from the **outside**. In chronic bronchitis , due to smoking, the goblet cells are stimulated to produce more mucus and the movement of the cilia is impaired so mucus stays in the lung causing chronic infections. If the patient has been consistently coughing (productive cough) for successive 2 years then that indicates chronic bronchitis.

Productive cough= cough with sputum

- Any damage due to emphysema and chronic bronchitis is irreversible.
- Destruction of alveoli is following by the destruction of the pulmonary capillaries, which will result in decreased capillary bed
- The pressure in the pulmonary artery ranges from 8-25 mmHg. The mean pulmonary arterial pressure is 15 mmHg. This is considered to be the after-load to the right ventricle. When 50% of the capillary veins are destroyed, the area is decreased so the resistance will increase. There will be increased pulmonary vascular resistance due to smoking. This is referred to as pulmonary hypertension.
- Pulmonary hypertension will result in dilatation of the right ventricle and eventually right heart failure due to increased load on the right ventricle.

- First stage of emphysema includes dilatation of the right ventricle. Therefore, emphysema is associated with right heart failure, this is called **Cor Pulmonale**
- so cor pulmonale is right ventricular dilatation +/- right ventricular failure due to lung disease --> dilatation today = failure tomorrow
- In addition to increased airway resistance, emphysema will cause disruption in blood gases:
  1. pO_2 will decrease due to decreased surface area of gas exchange in the alveoli.
  2. pCO_2 will increase later on because solubility of CO_2 is 20 times more than that of O_2.
- Emphysema patients will struggle to finish a meal without gasping for air.
- Emphysema is very complicated disease, it's not pure obstructive disease, it involves obstruction, decreased surface area, increased pulmonary vascular resistance & Cor Pulmonale

Cor pulmonale is defined as an alteration in the structure and function of the right ventricle (RV) of the heart caused by a primary disorder of the respiratory system. Pulmonary hypertension is often the common link between lung dysfunction and the heart in **cor pulmonale**

Cor تعني "قلب"

Pulmonale تعني "رئوي"

Cor pulmonale تعني مرض القلب الرئوي

#بالعربي

O. Saffar

Good luck! ☺

Le Fin.

"you can do anything you set your mind to" -Marshall Mathers III

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