

# RESPIRATORY SYSTEM

PHYSIOLOGY



**Title:** Sheet # 3 – Mechanics of Breathing

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In this lecture we are going to talk about **mechanics of breathing**.

✚ First of all, let's discuss some terms and formulas.

Boyle's law:

$P_1V_1 = P_2V_2$ , which means that in a certain container (thoracic cavity) the pressure \* volume is constant (they are **inversely** proportional).

An increase in volume would result in a decrease in pressure and vice versa.

Flow (gas) = Pressure gradient / Resistance.

As we know, gas moves from higher pressure to lower pressure (if we have no pressure gradient we won't have gas flow).

Notice, increasing the pressure gradient to have the same amount of gas flow is mainly because of an increase in the resistance (R) (airway resistance).

These formulas illustrate how we breathe, how?

We are dealing with mainly 2 pressure values: **Alveolar Pressure ( $P_{alv}$ )**, and the **Atmospheric Pressure ( $P_{atm}$ )**. In case of inspiration (inhalation) we need the atmospheric pressure to be higher than alveolar, since gas flow from higher pressure to the lower. And in case of expiration, we need a higher alveolar pressure than the atmospheric.

The change in the alveolar pressure is controlled by changing the volume (**inversely** proportional).

In case of ventilator/respirator/resuscitator (artificial breathing) we generate pressure gradient to get gas flow in the direction & amount needed. (**it's called the positive pressure breathing**). If you insert very high pressure gradient by the device, this could harm the patient by: 1- Damaging the alveoli themselves. 2- Obstructing the capillaries by this high Pressure (high gas flow).

Normal physiological breathing is called **Negative pressure breathing**.

Our lungs are surrounded by pleural cavity (potential cavity between the parietal and visceral pleura).

$(P_{alv})$  usually =  $(P_{atm}) = 760$  mmHg (can be written as **0 mmHg**), and the pressure in the pleural cavity is usually **756 mmHg (-4 mmHg)** (also called the intra pressure).

**HYPOVENTILATION** : is when alveolar ventilation is LESS than  
CO<sub>2</sub> production → increase in  $P_{ACO_2}$ .

**HYPERVENTILATION** : is when alveolar ventilation is more than  
CO<sub>2</sub> production → decrease in  $P_{ACO_2}$ .

It is different from **increased ventilation**. (for example: if someone is exercising, his CO<sub>2</sub> production is increased, but the washout of CO<sub>2</sub> is also increased by **increasing the ventilation** leading to fixation of  $p_{ACO_2}$ . In this case we don't say that he is hyperventilated because his  $p_{CO_2}$  didn't change.)

-Note:  $P_{ACO_2}$  is **proportional** to CO<sub>2</sub> production, but **inversely** proportional to alveolar ventilation.

✚ Now let's talk about how inspiration and expiration take place in details.

## 1. Inspiration

Since we want the gas flow to be from the atmosphere to alveoli, **the pressure in the alveoli must be lower than 0 mmHg (760 mmHg)**.

This is done by 3 stages: **increasing the volume of the thoracic cavity**, which will cause the **intrapleural pressure to decrease** (becomes almost -6 mmHg), which will inflate the lungs (increase in volume), and according to Boyle's law this increase **will lower the alveolar pressure** (becomes almost -1 or -2 dependent on how much the lungs inflated).

The increase in the thoracic cavity is done mainly by the **diaphragm** (most important muscle for inspiration), when this muscle contracts, it pushes the abdominal contents downwards and thus **increasing the thoracic cavity's volume**, followed by –as we said– **decrease in the intrapleural pressure**, and finally **inflation of the lungs** (decrease in  $(P_{alv})$ ) which will make the **inspiration** happens. (in addition to diaphragm, external intercostal muscles' contraction also increases the volume of thoracic cavity, but it's less important and the doctor couldn't care less about them).

✓ Let's clarify 2 things about this process :

1. The inspiration is an **active process** since it is done by the contraction of the **diaphragm**.
2. We needed much less pressure gradient (only 1 mmHg was enough to inhale 6L/min on average) in comparison to blood flow in the heart which needed much more pressure gradient (almost 100 mmHg to pump 5L/min), and that's because the resistance (which is **inversely** proportional to the flow) is higher in the blood flow (TPR). **"blood resistance is almost 100 times airways resistance"**

**Remember:** lung is an elastic tissue which means :

- 1- It is compliant (doesn't need much effort to inflate it)
- 2- It has recoil tendency (after stretching, it will get back to its normal volume passively)

(you can differentiate between compliance and elasticity by simply saying that **compliance is the tendency to inflate**, while **elasticity is the resistance** to inflation).

The inspiration will come to an end when the ( $P_{alv}$ ) = 0 mmHg (equals the atmospheric pressure). As long as the ( $P_{alv}$ ) is **sub atmospheric** (less than 0), the inspiration won't stop. Of course at the end of inspiration the intrapleural pressure also go back to -4 mmHg.

## 2. Expiration

After the inspiration ended, the **diaphragm** is going to relax, abdominal contents will go upwards again, lowering the lungs' volume, and thus (according to Boyle) **increasing the  $P_{alv}$**  (becomes almost +1 mmHg) which is the driving force needed for **expiration**.

Again, during expiration,  $P_{alv}$  is above 0mmHg. At the end of it,  $P_{alv} = 0$  mmHg.

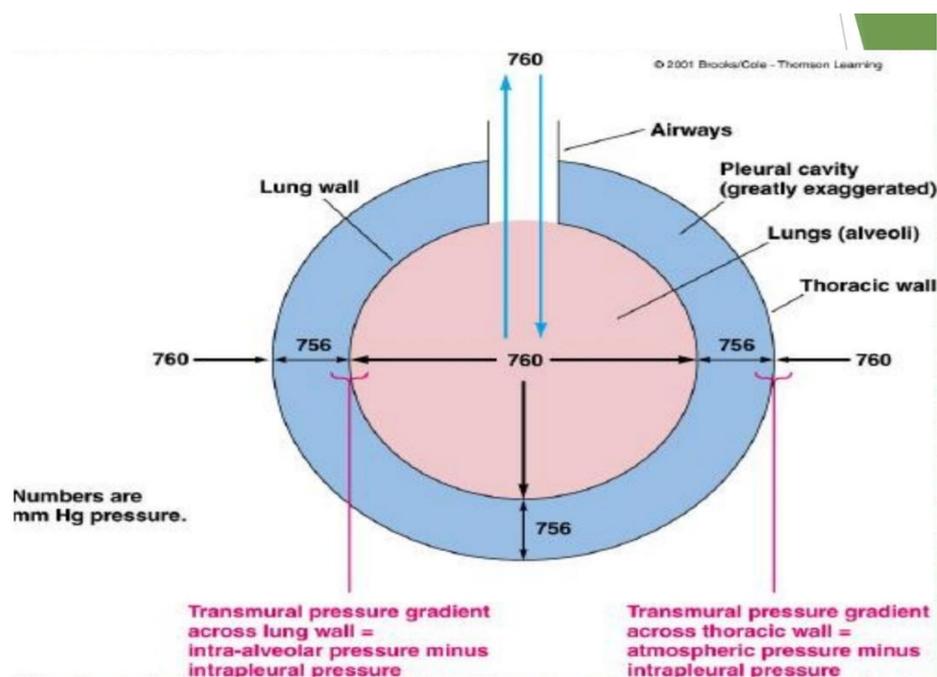
Notice that the **expiration** process is normally a passive process, done only by the relaxation of the **diaphragm**, and doesn't require any oxygen consumption like inhalation. (contraction of the muscle needs  $O_2$ ).

To demonstrate the idea, think of the lung as a balloon, you need force to inflate it, but no force is needed to deflate it (same idea for a rubber band).

Consumption of the inspiration muscle (mainly **diaphragm**) is almost 2-3% of the total ATP produced in the body.

Expiration is normally **passive**, but can be **active** (needs ATP) in some cases (**exercising**), in this case we need **expiration muscles** (like **abdominal and internal intercostal muscles**) to contract resulting in a lower volume in the lungs, higher pressure and a greater driving force. But we can't tolerate this all the time, because muscles used for breathing can consume up to 80% of ATP produced.

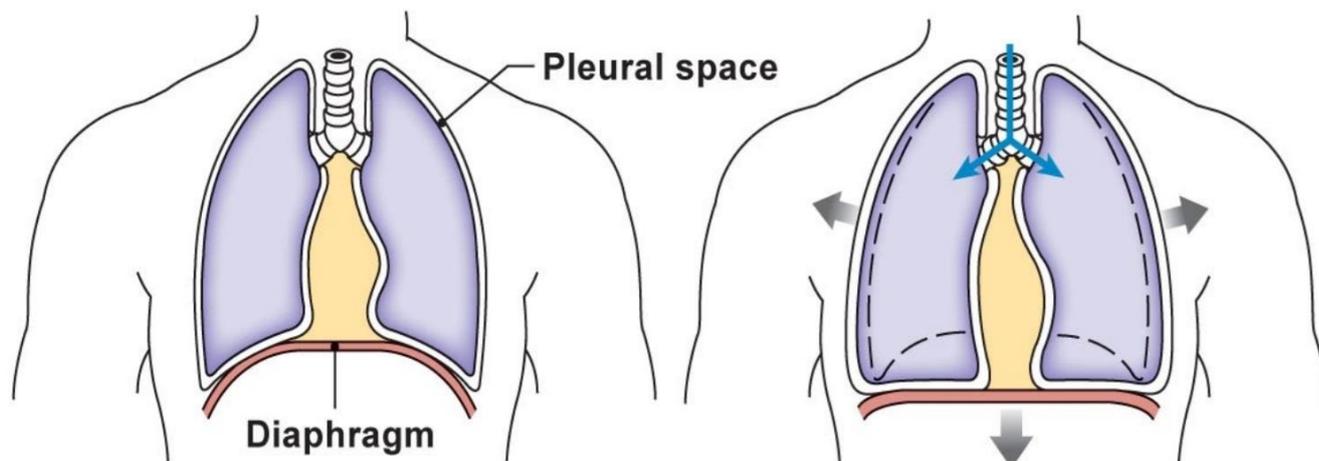
Inspiration process takes 2 seconds, while expiration takes 3, the cycle takes a total of 5 seconds. Normal respiratory rate (number of breaths per minute) =  $60/5 = 12$  breaths. (10-20 is normal) (tachypnea more than 20 breaths per minute) (bradypnea less than 10).



This picture summarizes what we talked about, the **pink** (not sure it's pink) colour resembles the lung. The **blue** (pretty sure this time) colour shows the pleural cavity which covers the lungs from outside, and lines the thorax from inside.

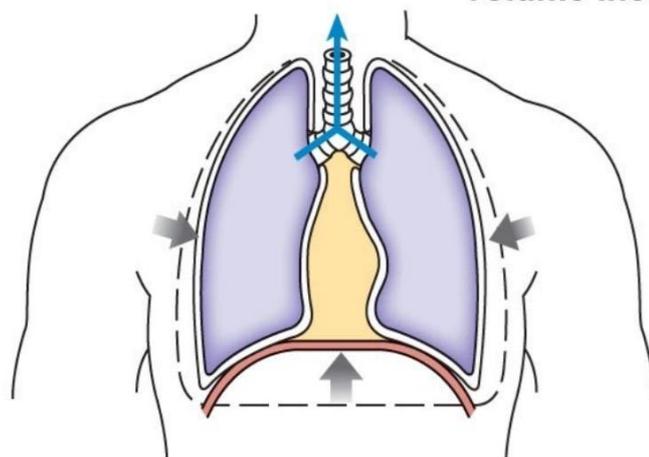
**Pneumothorax** is a pathological condition in which air inflow into the pleural cavity and will cause an increase in its pressure, causing the lungs to collapse. It is pretty

serious and should be immediately treated by surgical intervention by connecting the cavity with a tube having ( -ve) pressure so we get gases out of the cavity (**hemothorax** is a condition where blood accumulate in the cavity).



**(a) At rest, diaphragm is relaxed.**

**(b) Diaphragm contracts, thoracic volume increases.**

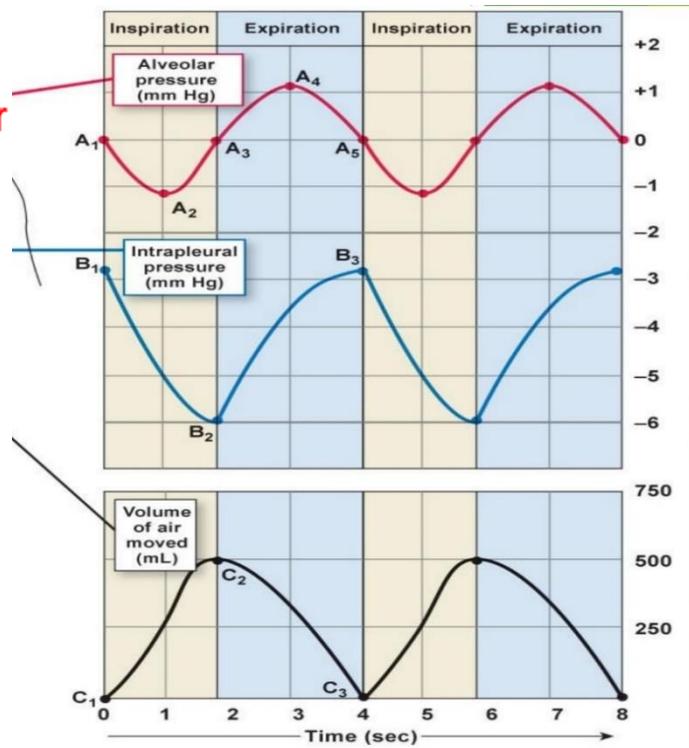


**(c) Diaphragm relaxes, thoracic volume decreases.**

This picture shows the role of the **diaphragm** in the inspiration and expiration processes. (most important inspiration muscle). Should all make sense, if not, check page 2 again.

In newborn respiratory distress syndrome (NRDS), the baby's lungs are not fully developed, so he/she uses all inspiratory muscles (diaphragm, external intercostals, some muscles of the neck) trying to inflate the lungs. (also very briefly mentioned).

This picture shows the relationship between **alveolar pressure** and **intrapleural pressure**, keep in mind that the decrease in the intrapleural pressure will cause inflation of the lungs, and thus decreases its pressure (**not the opposite**). And the decrease in alveolar pressure will cause the gas inflow.



Important concept: Respiratory Minute ventilation RMV (respiratory rate\*tidal volume “12\*0.5=6L/min”).

RMV= anatomical dead space ventilation + alveolar ventilation “Total amount of air moved into and out of respiratory system per minute”

Now let’s talk about some **Gas Laws**:

- **Ideal gas law:  $PV = nRT$**  (the pressure and volume (**PV**) of a container is directly related to the temperature (**T**) of the gas, and the number of molecules (**n**) in that container. (**R** –universal gas constant- = 8.3145 j/K.mol).

This law helps us converting Pressure of a gas (CO<sub>2</sub> for example) from **mmHg** into [CO<sub>2</sub>] **mmol/L** which is important in renal physiology (we will use it later on).

- **Henry's law**

At a constant temperature, the amount of a given gas dissolved in a given type and volume of liquid is directly proportional to the partial pressure of that gas multiplied by its solubility.

\* Solubility has a constant which is different for each gas.

Using this law, you can predict how much O<sub>2</sub> and CO<sub>2</sub> are available in dissolved form.

Solubility hardly change, but PO<sub>2</sub> and PCO<sub>2</sub> can both change.

When a liquid or gas (blood and alveolar air) are at equilibrium, the amount of gas dissolved in fluid reaches a maximum value (Henry's Law). This depends on the solubility of gas in the fluid, temperature of the fluid and partial pressure of the gas.

**concentration = P \* solubility**

[O<sub>2</sub>] = paO<sub>2</sub> \* solubility = 100 \* .003 = 0.3ml/dl.

Note: This is the concentration of dissolved O<sub>2</sub> ONLY, O<sub>2</sub> bounded to Hb isn't taken in consideration.

[CO<sub>2</sub>] = 40 \* .06 = 2.4ml/dl.

Here are some info from the slides that the doctor didn't explain, but he said that they are required.

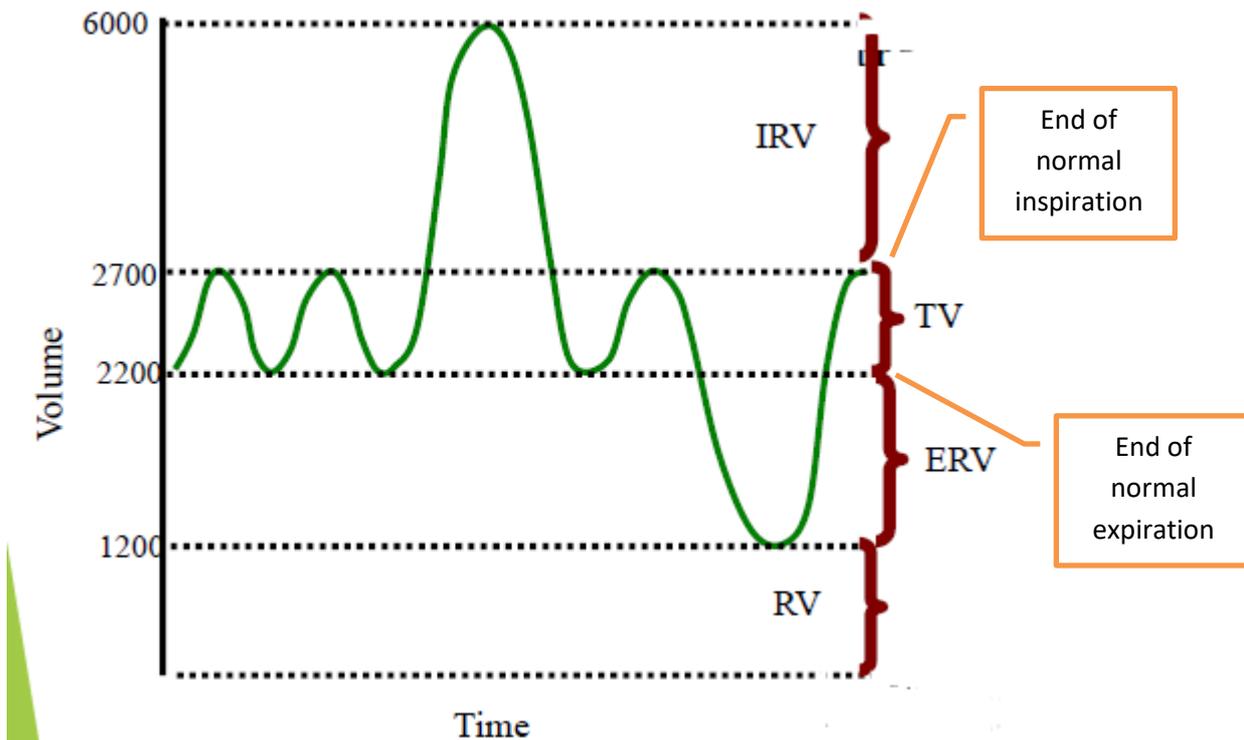
Note: **Functionally,**

1- alveoli at **apex** are **underperfused** but **overventilated**

2- alveoli at the **base** are **underventilated** but **overperfused**

This is mainly due to gravity effects on alveoli.

## Pulmonary Function Tests (PFT): lung volume and capacities



TV: Tidal Volume

IRV: Inspiratory Reserve Volume

ERV: Expiratory Reserve Volume

RV: Residual Volume

- **Inspiratory capacity (IC):** the maximum amount of air that can be inspired following a normal expiration.

$$IC = TV + IRV$$

- **Vital capacity (VC):** the maximum amount of air that can be expired following a maximal inspiration.

$$VC = IRV + TV + ERV$$

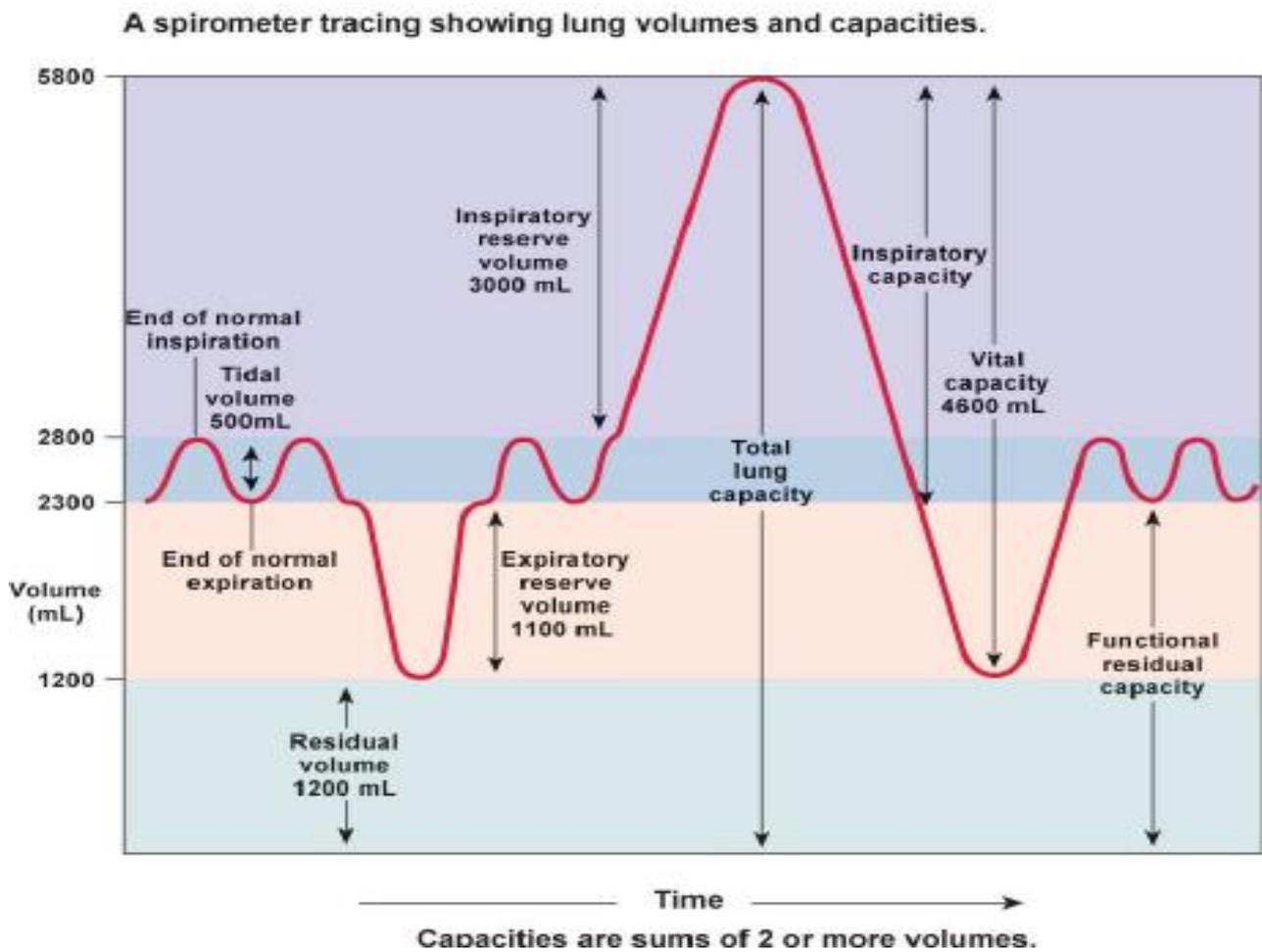
- **Function residual capacity (FRC):** the amount of air remaining in the lungs following a normal expiration.

$$FRC = ERV + RV$$

- **Total lung capacity (TLC):** the amount of air in the lungs at the end of a maximal inspiration.

$$TLC = IRV + TV + ERV + RV$$

Here is a diagram to summarize this:



*Good Luck*