PASSION ACADEMIC TEAM yu - MEDICINE

Sheet# 2

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Lec. Title : Pulmonary volumes & capacities

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

RESPIRATORY SYSTEM

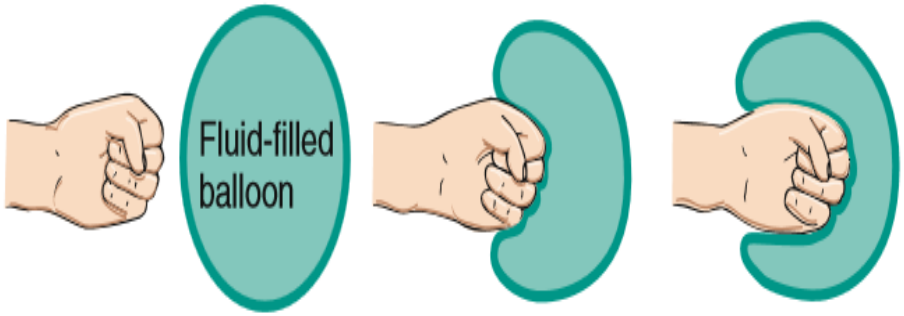
LUNG

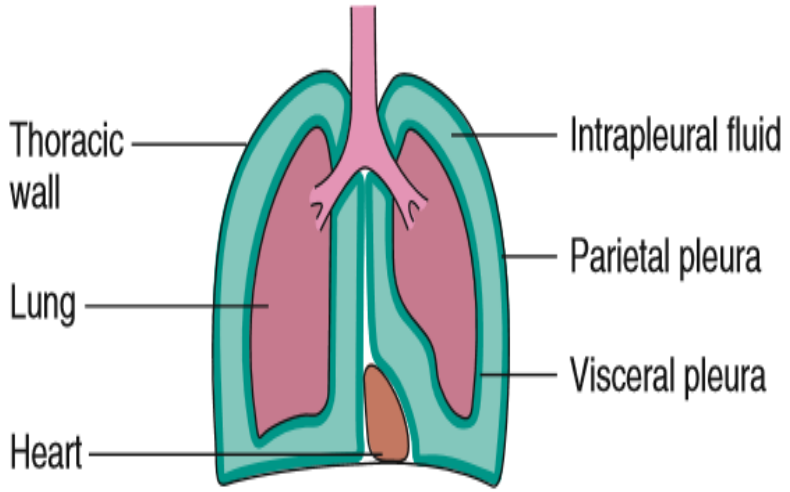
The lungs are covered by pleura.
There are two layers:

- The parietal pleura
- The visceral pleura

- Pleural fluid, which is the ultrafiltrate of plasma. Normally, about 10–20 ml (about 10 µm thick) of this fluid is present in the pleural cavity.

- Pleural effusion??





SHEET 1

We have 2 layers that surround the lung :

- **parietal layer** : lines the chest wall (the outer one)
- **Visceral layer** : covers the lung
- In between there is tiny space , known as pleural space or pleural cavity and there is small volume of serous fluid , known as serous pleural fluid , the source of this fluid is blood plasma.

- Blood vessels are present in **the parietal layer** , the plasma is filtrated from these blood vessels into the pleural cavity , producing pleural fluid **(ultra-filtrate of blood plasma**).
- **In visceral layer** there is lymphatic vessels , they **remove** continuously the excessive amount of pleural fluid

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- We have BALANCE between **filtration** of pleural fluid by capillaries and **removing** of pleural fluid by lymphatic
- If lymphatic drainage is blocked OR the filtration become more than removing of pleural fluid it will cause **pleural effusion** (fluid accumulation in the lung)

•**pleural effusion** has bad consequences on respiratory system and the fluid will cause compression pressure and it will compress (contract)the lung (we can say it collapses but not totally)

The pleural effusion also affect the Diffusion , by affecting the distance of the membrane

• respiratory membrane is a tiny memb. **0.1 – 0.5 micron** in thickness , if the thickness increase because of fluid accumulation it will cause obstruction of the diffusion in the lungs

•NOTE: the space of intrapleural pressure has nearly always negative pressure =sub-atmospheric pressure ,rarely positive pressure during expiration

Another importance of the pleural fluid :

- It helps the lungs to move easily by working as a lubricant .
- Protect the lungs from external damage . HOW?

-*The non expansive and the adhesion nature of pleural fluid* keeps the pleural and visceral layer together (stuck to each other , non separated) like 2 microscopic slides contain a drop of water in between can't be removed from each other , but move with each other , this help the lungs to adhere to chest wall , so if there is expansion in the chest wall there will be expansion in the lungs too

-The lungs don't contain any muscles , but it can expand when the chest wall muscles expand ,also when there is compression in chest wall there will be lung compression

-SO as a consequence, any change of the chest wall movement will cause change in the lung's volume and pressures

- Changing pressures will cause pressure gradient which is **the requirement of ventilation**

-So the pleural keeps the lungs **glue** with the internal surface of the chest wall , but it also help the lungs to slide freely and easily on the chest wall.

SHEET 4

- The pressure gradient is the difference between atmospheric and lung pressure

- The atmospheric p. is constant 760mmHg (also known as 0) which can't be changed , so we change the pressure of the lungs by changing the volume (the pressure decrease when the volume increase according to boyle's law) , so how it can be changed ?
- The lungs are stuck along with chest wall and move with it , they expand and compress together , and so on it causes the change in lung's volume and pressure .

- If a puncture , trauma or any damage in the chest(thoracic) cavity occur ,it will cause lung collapse.

Mechanics of Breathing

OUTCOMES

- **On completion of study of this chapter, the student MUST be able to:**

١. **List the muscles of inspiration and expiration.**

٢. **Describe the mechanics of pulmonary ventilation**

٣. **Explain pleural pressure, alveolar pressure and transpulmonary pressure.**

٤. **Define lung volumes and capacities and give their normal values.**

٥. **Define lung compliance and explain the factors affecting it.**

٦. **Understand the relaxation pressure-volume curve of lungs.**

SHEET 5

- Mechanics of Breathing are forces that determine respiration process , these forces are 2 types :
- **Recoiling force (elastic recoil force)**
- **Transmural force**

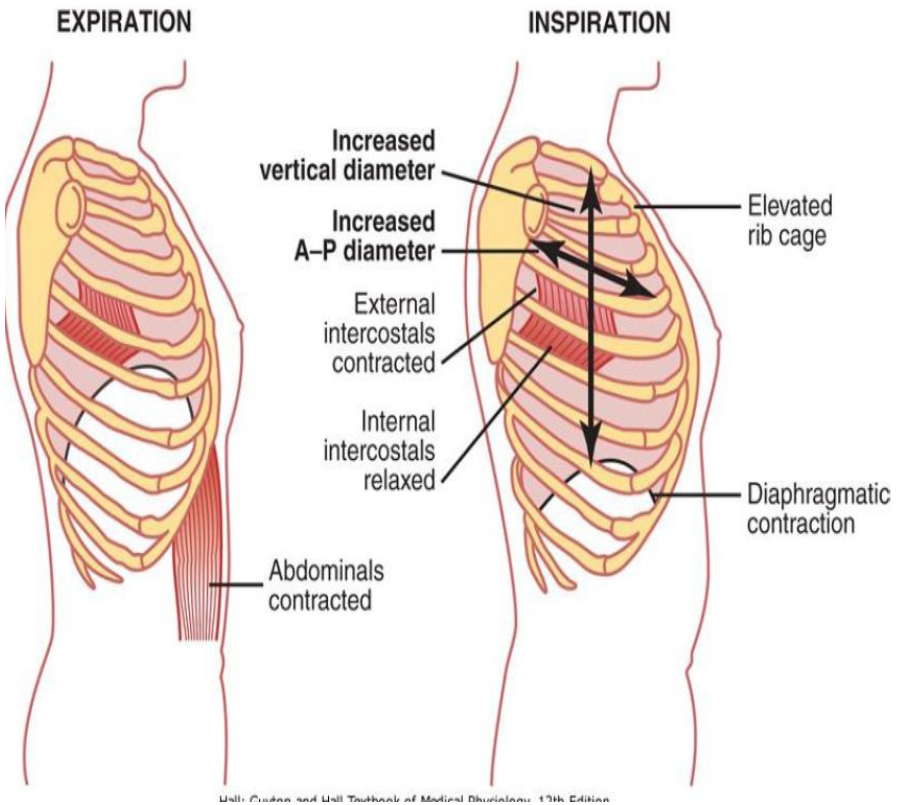
- if Transmural force is higher than recoiling force of the lungs , there will be an **expansion**
- If the recoiling is higher than Transmural , the lung will **contract and compress**

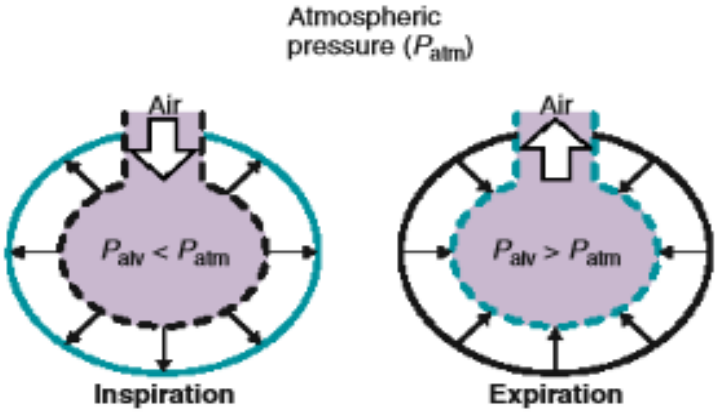
- Ventilation need pressure gradient between atmosphere and the lungs . We can induce it by changing the volume of the thoracic cavity :

- Expansion of thoracic cavity → expansion of lung (increase in volume)→ decrease in alveolar pressure →**inhalation**

- Compression in thoracic cavity→ compression in volume of the lungs (volume decrease) →increase in alveolar pressure (or pulmonary pressure) → becomes higher than atmospheric pressure →(bulk flow) **expiration**

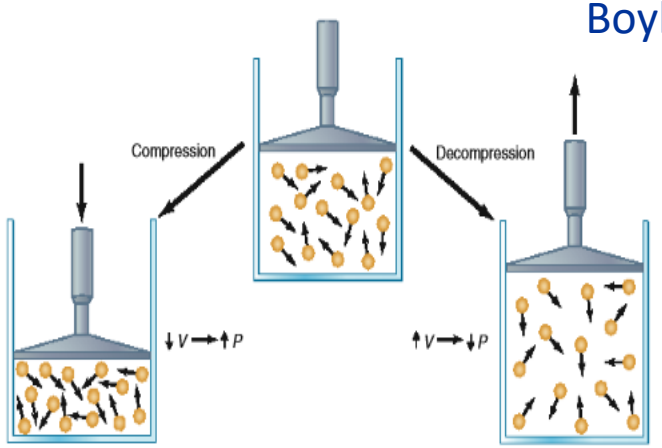
Mechanics of Ventilation



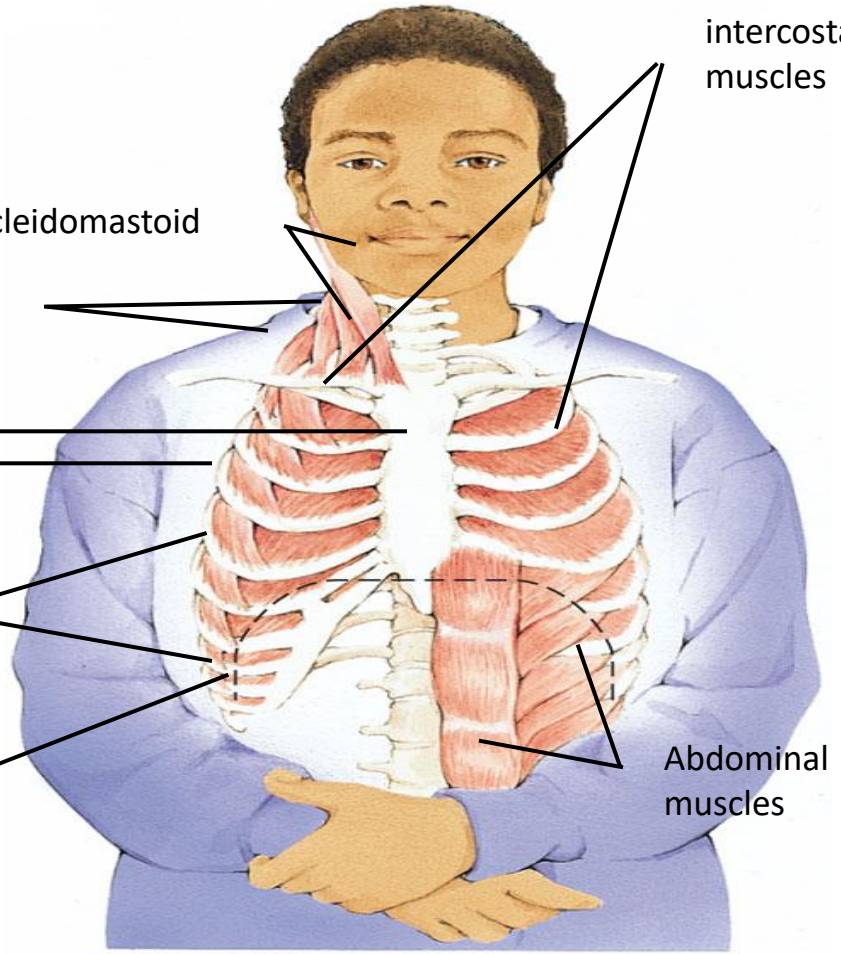


$$F = \frac{P_{alv} - P_{atm}}{R}$$

Boyle's Law



Inspiratory Muscles



SHEET 6

•To cause a change in lung's volume , we need to change size of thorax (change in volume in all dimensions , interiorly, posterior, superiorly, inferiorly and laterally
•The respiratory muscles are responsible for increasing the size of thoracic cavity.

•In **quiet breathing** (normal breathing) the inspiration is an **active process** which means it needs muscle contraction
• quite expiration is **passive** (spontaneous process)—without muscle contraction.
• the major muscle in quite inspiration is the diaphragm (work alone without external intercostal M.)

WHILE

•**Forced breathing** (both inspiration and exhalation) become **active processes** , both need muscle contraction
•All muscles are needed in forced breathing , diaphragm , External Intercostal muscle, Sternocleidomastoid , and others … , why ?
•To cause a higher change in thoracic cavity **size**= more increase in lung volume = more decrease in the pressure = which ultimately cause more air to get in the lungs.

Pressures involved in ventilation

✓Atmospheric pressure (P_{atm}) 760mm Hg (cm H20)

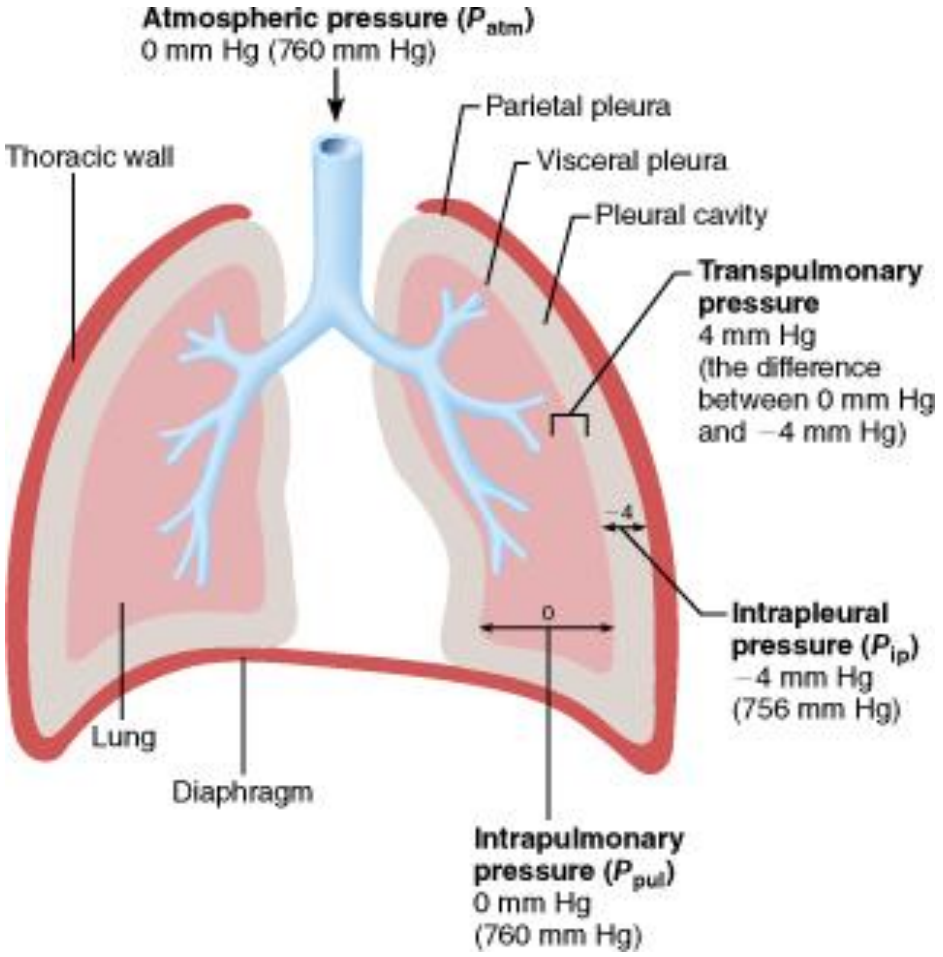
- Conventionally, atmospheric pressure is set to zero and all other pressures are set relative to this.

✓ Intra-Alveolar pressure (P_{alv})

- Negative during inspiration
- Positive during expiration

✓ Intrapleural pressure (P_{ip}): Negative and subatmospheric

✓ Transmural pressures



SHEET 7

•When contraction of the muscles occur , it changes the pulmonary pressure (alternate to Lesser than and greater than atmospheric which lead to inhalation and exhalation respectively)
•BUT In addition to changing the volume of lungs , we have other pressures also change during muscle contraction, expansion and compression of the thoracic cavity .

These pressures are :
•Intrapleural pressure
•Transmural pressure
•Intrapulmonary pressure (alveolar pressure inside lungs)

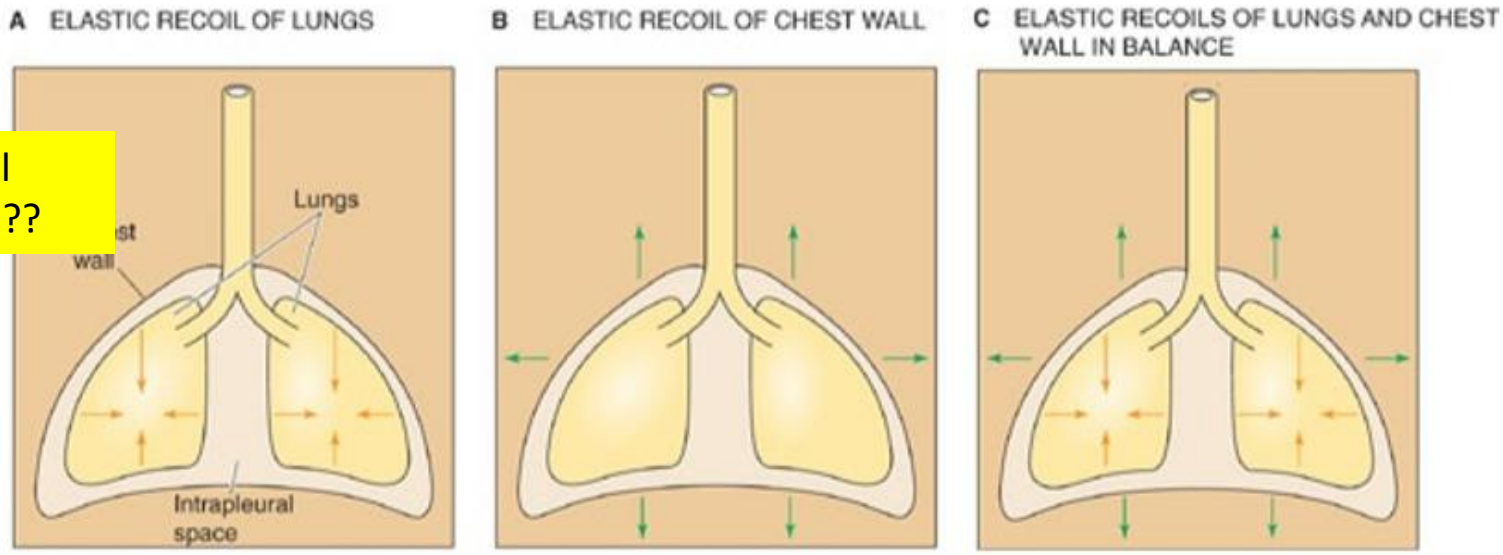
•**Intrapleural pressure** always sub-atmospheric (less than zero/760 mmHg) , in rest its negative value between **-4 to -5** (in average) in the apex it will be more negative for example -10 because the space is larger , while in base it will be less negative for EX. (-2)because the space is smaller (larger space=less pressure) so its not constant , can be changed.

•In case of **forced expiration** the value of intrapleural p. could be positive.

Intrapleural Pressure

❖ How is the Intrapleural Pressure created and Why is It Negative?

Physiological Significance ??



SHEET 8

- How is the intra pleural pressure created and why its negative ?

- By Ultra-filtrate of plasma (pleural fluid) from capillaries in parietal layer , but then continual suction (drainage) occur which create a negative pressure
- Intrapleural is negative because its between 2 forces ,equal in magnitude but opposite to each other , lung recoil force and chest wall recoil force

- **Alveolar P. (pulmonary P.)** alternate between positive & negative
- Positive during exhalation , negative during inspiration
- in rest it equal zero (at the end of expiration and the beginning of inspiration there is no air flow so alveolar p. = atmospheric p. ,no gradient (**atmospheric p. =alveolar p. = 0)**

- **The clinical significance of the negative intrapleural p. is :**

1. Increase venous return from tissues to right ventricles in the heart (because negative p. inside the pleural also reflex the pressure in the space around the heart , the pressure in the interstitial fluid , large blood vessels , esophagus and most spaces in thoracic cage all are negative pressures)

2. to prevent lung collapse (the lung has tendency to collapse due to elastic recoil and surface tension) . the negativity of intrapleural P. keep the lungs open, when interrupted (when air fills the space outside of the **lung**, or by fluid accumulation ,..) there will be only the affect of elastic recoil force therefore collapse occur

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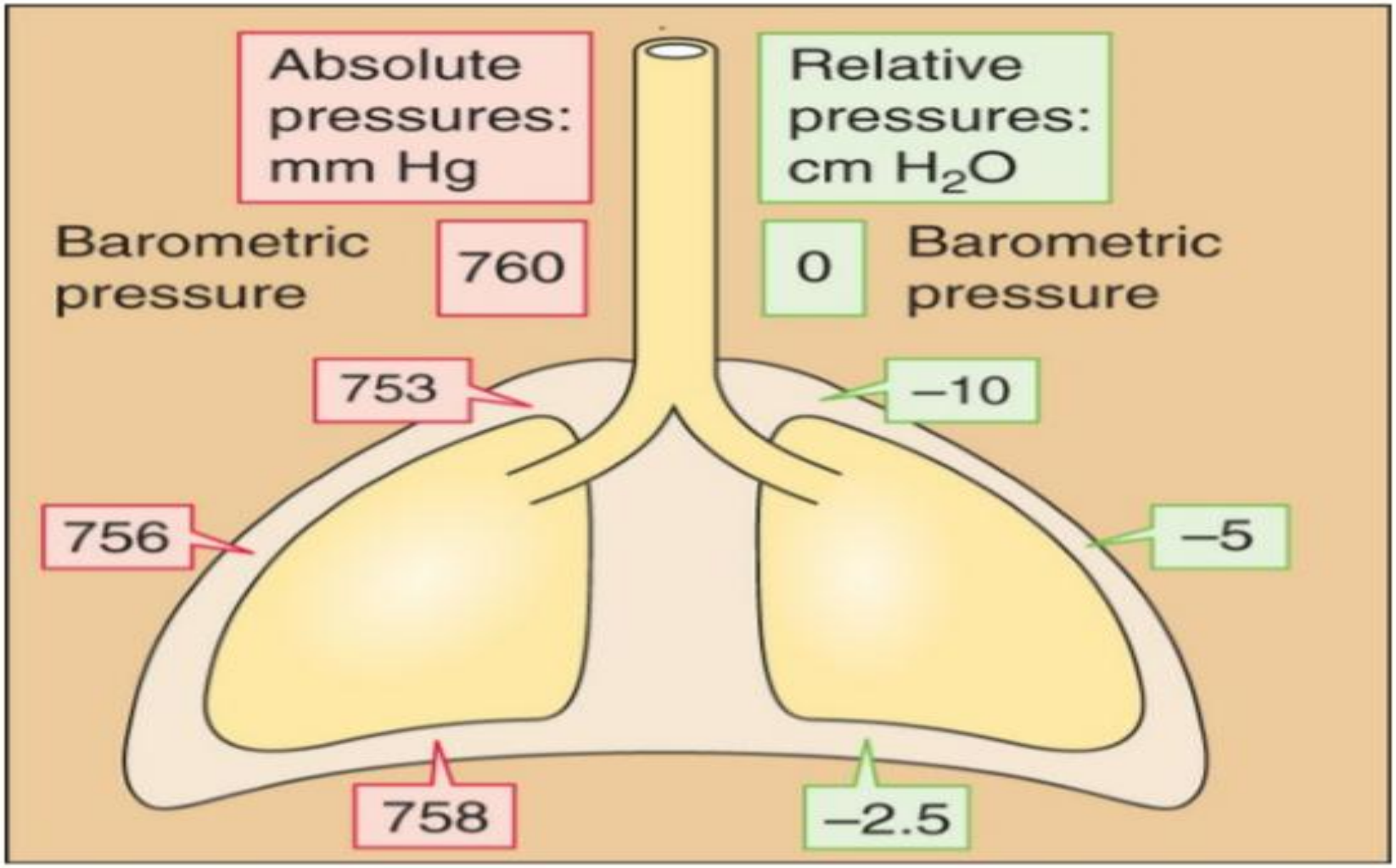
- Elastic recoil force in lung is **inward force** , tendency to collapse .While elastic recoil in chest is **outward force** , has the tendency to spring out
- So they create **a negative intrapleural pressure** (having distance in between)
- Any 2 surfaces come close to each other , they create a positive pressure . But if they stay away from each other they create a negative pressure

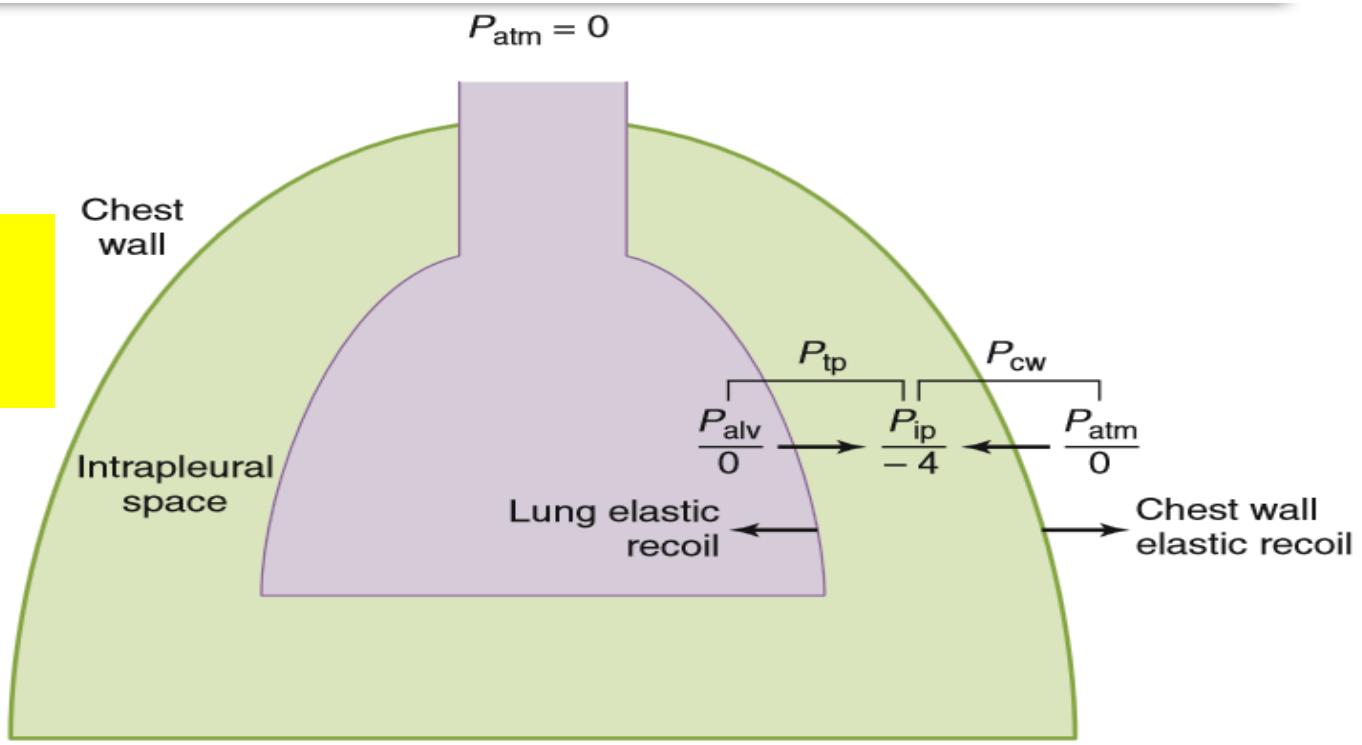
Effects of Posture and Gravity

intrapleural pressure



Transmural pressure

Transmural pressure (Ptm) is the pressure difference across a wall.



Transmural Pressure	$P_{in} - P_{out}$*	Value at Rest	Explanatory Notes
Transpulmonary (P_{tp})	$P_{alv} - P_{ip}$	0 − [−4] = 4 mmHg	Pressure difference holding lungs open (opposes inward elastic recoil of the lung)
Chest wall (P_{cw})	$P_{ip} - P_{atm}$	−4 − 0 = −4 mmHg	Pressure difference holding chest wall in (opposes outward elastic recoil of the chest wall)

SHEET 10

•At rest (at the end of expiration) when intrapleural p. equal -4 and intrapulmonary P. Equal 0 , there will be a resting volume of a lung which is called **functional residual capacity**
•In resting state : Transmural P. will be equal to elastic recoiling force of the lung also equal to elastic recoiling force of the chest wall
•Transmural p. is the pressure difference of a wall (pressure inside the wall – the pressure outside the wall)
•Transmural p. of the lung = p. inside the container(wall) – p. outside the wall

= alveolar pressure - pleural pressure

•At rest (at functional residual capacity):
• the alveolar pressure inside the wall = 0
•While pleural pressure = -4
•Transmural pressure = 0 – (-4) = 4
•This 4 Transmural p. at rest is equal to elastic recoiling force of the lung but in opposite direction (positive cause inflation (positive means inside higher than outside)) & (if Transmural p. is negative compression occur)

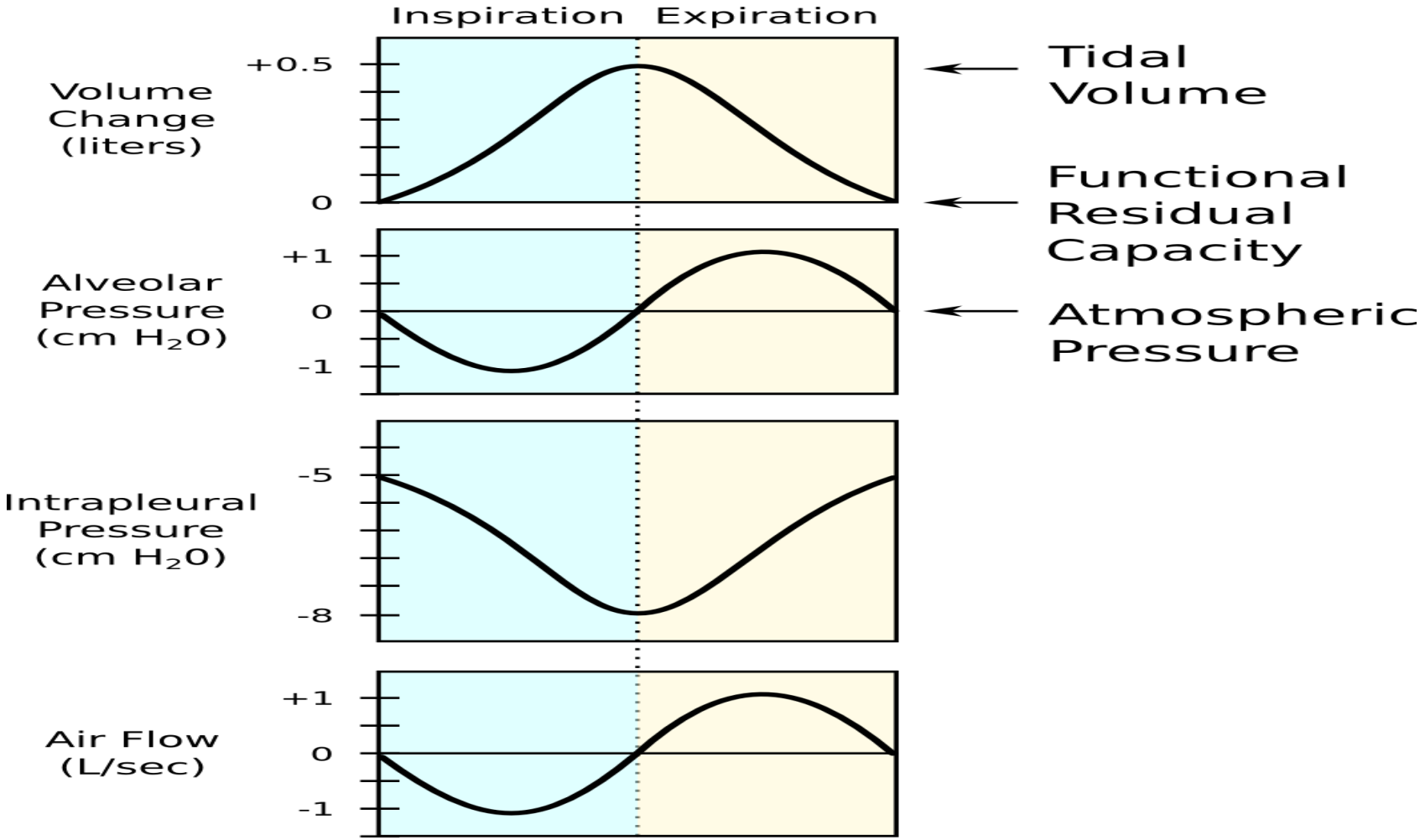
•Although it cause inflation but the volume of the lung will stay constant because it reflect the lung collapse by this inflation (recoiling force) →no change in volume →no change in pressure → no air flow (0) → which means the lungs is at the end of expiration &beginning of inspiration
.

- This pressure difference hold the lungs open , stay inflated so it won't collapse because the outside pressure is **negative** which means the Transmural p. positive , ultimately the lung will stay open .
- This pressure oppose the inward elastic recoil of the lung, so lung stay inflated
- Transmural pressure in the chest wall equal intrapleural p.(inside p.) – atmospheric p. (outside p.)

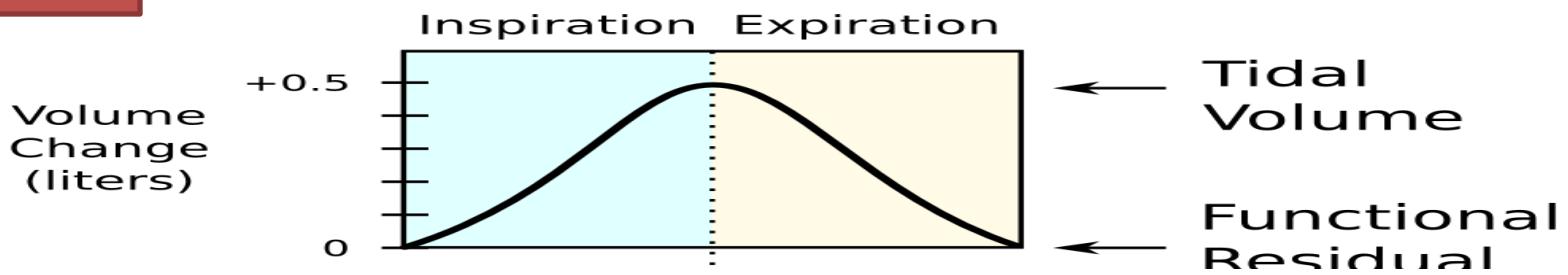
  = -4 – 0 = **-4**
- **Negative** means compression (contraction) , but also it opposes the outward elastic recoil of the chest wall to inward (this pressure difference hold the chest wall in)
- So the chest wall remain fixed in position , for ex. If injury happen it will cause a shrinkage of the chest and it change in position to out , but the pressure difference in normal condition is what hold the chest in (oppose the elastic recoil of the chest)

- I**nhalation** cause increase in Transmural p. more than elastic recoiling of the lung →causes **expansion** of the lung because Transmural p. is higher than elastic recoiling of the lung → then it get back to its normal condition because recoiling p. of lung become **equal** to Transmural p. . HOW?
- changing in volume(increasing), cause changing in pressure which cause **expansion** , BUT as the volume of the lung increase to a specific point, the elastic recoiling p. will try to get the volume back to its normal which causes the elastic recoiling p. and the Transmural p. to become equal (balance)

General concepts of ventilation mechanics



SHEET 12



- **Zero** here means the volume is at resting volume of the lung →functional residual capacity (the air that stay in the lung continuously even after normal expiration)

- Even when deep expiration occur , and decreasing in functional residual capacity occur , there will still little air we call it **residual volume .**

- Even when lung collapse this doesn't mean the volume is 0 , residual volume decrease but it still present , and this little remaining air called minimal air volume (see next page**) .**

- The importance of the minimal volume :

- **We can know if the new born baby , was born died or died after the delivery . HOW ?**
- If the newborn was breathing for little time then the collapse occurred , in this case there will still **minimal air volume in the lungs** (by taking a section of the lung and see if there is a minimal volume)
- If the newborn was born died there will be no minimal air volume (never breathed after delivery)

- They can put the section of the lung in water , if it float up, it means it has air inside , if it Sank (fall down) mean it has no air inside and he never breathed with his lungs .
- we have this minimal volume air even when collapse occur. HOW ? Because proximal passage airways collapses before the distal airways and they trap a minimal amount of air inside .

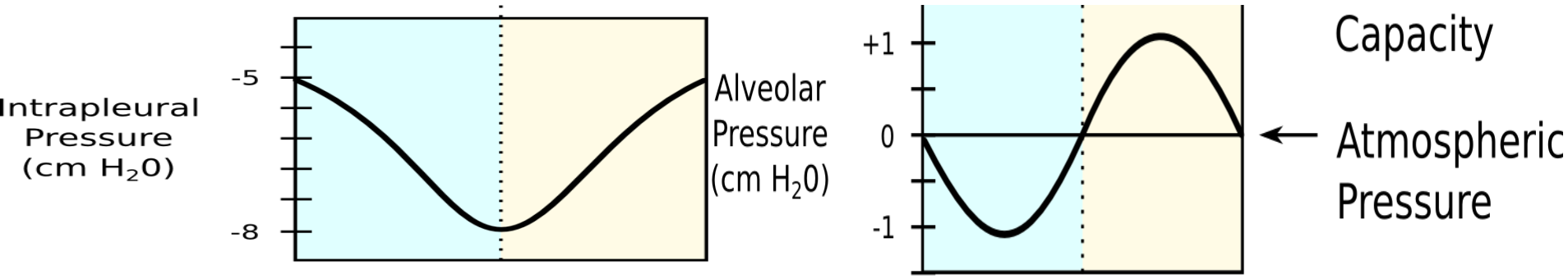
SHEET 14

- **The importance of functional residual volume :**
- First we know that Respiration is **cyclic process** (inspiration & expiration, air present then no air) while blood flow is **continuous process** .
- In Inspiration gas exchange between the blood and the lung occur
- in expiration IF the lung had no air →it means the blood will go to the alveoli , but it won't be able to exchange gases → which reflect badly on Pco2 & PO2 (partial pressure of CO2 & O2 in arterial blood vessels)→ which causes hypoxia, hyperkapnie , and others

- this functional residual volume act as a **BUFFER** , so we can have continuous gas diffusion (continuous exchange between blood and alveoli)
- Also this is how we can hold our breath.

- If a lung collapse , when you need to make inspiration next time you will need tremendous breathing effort to re-inflate the lungs..
- Also collapse after expiration will cause collapse in pulmonary vasculatures → which lead to increase in resistance and cause high load on right ventricles (blood flow through high pressure vessels because of its resistance) .
- normally pressure in pulmonary vessels are low even with exercise and increasing cardiac output, which is very imp to prevent edema and others..

SHEET 15



- At the end of expiration , at rest , the pulmonary (alveolar) p. =0 (no air flow)
- Intrapleural p. = -5 or -4 cm H2O
- 1 mmHg = 1.36 cm water

- See the next slide about the figures ..

SHEET 16

- Increasing in thoracic cavity as consequence of diaphragm contraction in all dimensions , leads to pull the parietal layer (that lines the chest wall and move along with it)→ so alveolar space increase but only a little ,because there still a fluid which is in-expansive and adhesive fluid (they stay adhered to each other but there will be a little increase in the space) →leads to **more negative pressure** from -5 to -6 to -7 decrease gradually due to pleural fluid which allow the lung to freely slide against the chest wall

  (expansion in both , compression in both at the same time)→ expansion in thoracic cage= expansion in lungs , so the pressure became -1 instead of 0 (we create a pressure gradient between outside which is atmospheric and =0 , and the inside which was zero and became -1)
- Transmural p. **at rest** = 0 – (-5)= 5 (inflation)

- Transmural p. **in previous case** = -1 – (-7) = 6 (more inflation in lungs and decrease in pressure , so air move from outside to inside , inhalation (air flow inside) so as air enters the lungs the alveolar pressure start to increase(because of air entering) and continue to increase until it reach 0 which means the end of inspiration (Transmural p. = elastic recoiling p. of the lung but in opposite direction)

- Then relaxation of muscles occur, which lead to decrease in intrapleural p. (reach -8 look at the figure) then re-increase , which means Transmural p. became less than elastic recoiling p. = increase in alveolar pressure to become 1 (which is higher than atmospheric pressure) and so on **expiration occur** .

THE END

“ it does not matter how slowly you go as long as you don’t stop”