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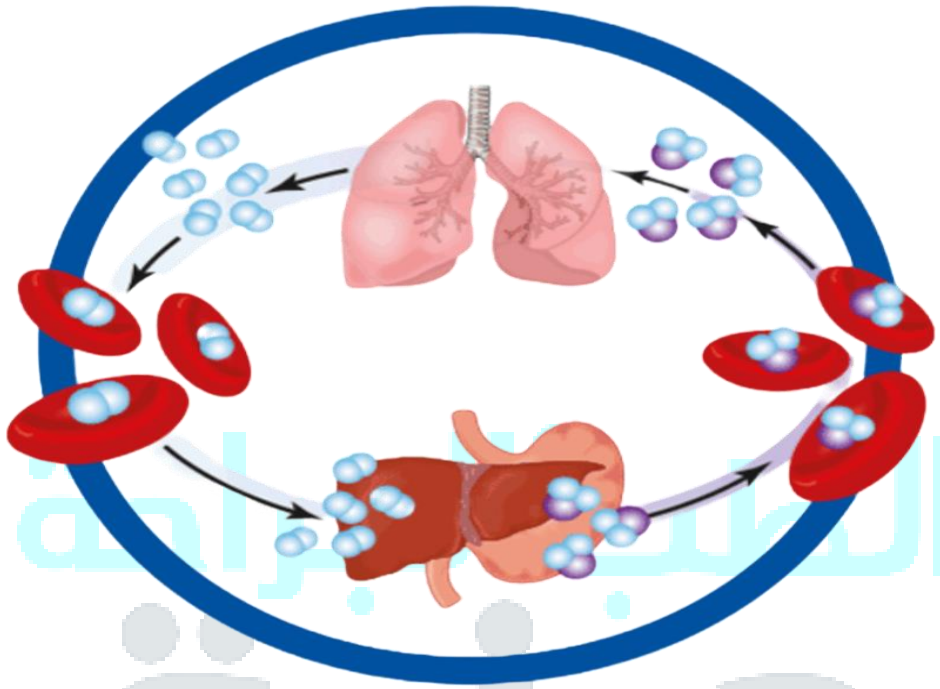
Doctor 2020 - wateen - medicine - MU



physiology sheet

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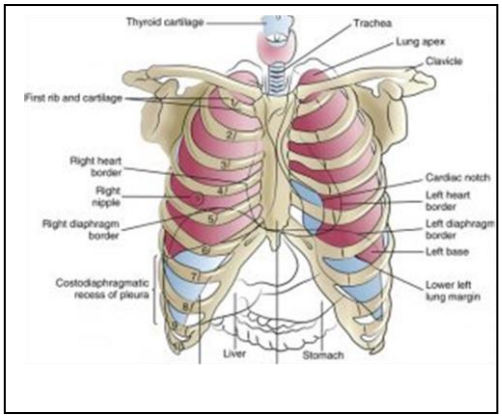
Pulmonary volume and capacities



**** anatomical introduction :**

Thoracic cavity (cage) **consist** of : ribs ,

vertebral cord , Clavicle , sternum …

_ land marks :

1- **clavicle (collarbone)** attached to sternum (very Hard bone) & medial border of Scapula.
2- **sternum :** can sense the manubrium
3- **Anula of Luis :** indication that I reach second rib .

What is inside the cage ? lung , heart , muscles , diaphragm , pleura which cover the thoracic cage opens & have two layers :

1- Visceral : covers the lung
2- Partial : next to the chest
And they are both separated by pleural fluid which makes pressure called intra-pleural pressure .

- Different in pressures makes the movement of air .

Quit normal breathing

Inspiration (negative pressure) → the air inside the human body

WHY NEGATIVE ? compared to atmospheric pressure , it's lower than atmospheric pressure (pooling the air)

Expiration (positive pressure) → outside the human body (atmospheric pressure)

- The air moves from (+) to (-) , from high to low

Intrapleural or intrathoracic pressure: is always ***negative*** due to dynamic harmonious antagonism between the chest wall and the lung

To prevent pooling and direct adhesion .

Elasticity of the lung → assuming the smaller size (inflating)not deflating

Mechanics of breathing

There are three types of pressure;

1. Intrapulmonary a.K.a, intra-alveolar. [can be positive or negative)

2. Intrapleural a.k,a. intrathoracic. (always negative)

Controls all the pressures in the chest wall

WHY ORGANS INSIDE THE CHEST WALL HAS THE SAME PRESSURE OF INTRAPLEURAL ?

If the pressure increase inside the esophagus

Comparing to the lung → the esophagus size will increase

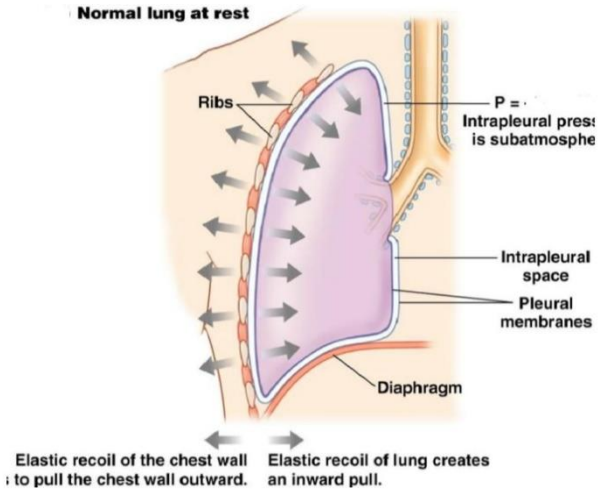
So that that's wrong and doesn't occure in normal cases , and then the Intrapleural pressure = intrathoracic pressure = pressure in esophagus so that its shape doesn't change

3. Transthpulmonary a.k.a, transmural: the difference between 1 and 2 [always positive)

** atmospheric pressure caused by gases in the air (O2 , CO2 & N2)

Intra plural pressure

- -5 to -7 normal
- Less negative emphysema
- Zero at birth and stab wound without valve



- Positive stab wound tension pneumothorax with valve and Valsalva maneuver

- **What conditions will lead to zero value in intrapleural (intrathoracic) pressure ?**
  1- in pneumothorax without a valve also called non valve open pneumothorax (occurs in stab wound by sharp tool)
  2-in the lung of birth (لأنه الطفل بكون لسا ما استخدمها)
- **What conditions will lead to make intrapleural pressure more positive than atmospheric?**
  In valve pneumothorax (When the pleural cavity is damaged or ruptured)

Emphysema (chronic obstructive disease) : narrowing in lumen , so when inspiration the air inter to the lung , but when we expired the air the lumen will be narrower and the size of lung will increase and decrease the volume of pleural cavity , so tow layers of pleura will close to each other and then according to boyel's low → increase interpleural pressure → decrease the negativity

- **obstructive disease : occurred in the airway ; like emphysema**
- **Restrictive disease : caused by the elasticity of the lung**

in both types if we take the air in and couldn't get it out <- وجه الشبه بينهم

The greater resistance is found in the **bronchi.**

• That's why asthma can be dangerous → bronchial hyperreactivity →

constriction of bronchi (which already have more resistance than bronchioles) → decreased radius → increased resistance → air cannot exit→ obstructive lung disease.

Which control the resistance of arteries as a first line ?

Radius ✔

Which part of conducting system has the highest resistance ?

Bronchi ✔

So that , in asthma (obstruction in the airways) happens in the bronchi and all drugs we take are dilate the bronchi , not bronchiole dilator ☺

- Question: is the problem worse during inspiration or expiration?

• Answer: -expiration, because the airways are narrower → less radius → more resistance.

- so, during expiration, I cannot get the air out (Obstructive lung disease)

	inspiration	***expiration***
Nature	active	passive
Duration	longer	shorter
Dimensions	expansion in 3 dimensions	decrease in 3 dimensions (lung recoils)
	increased volume —> decreased pressure (Boyle's law) • *I mean the intrapulmonary (intra-alvelolar pressure)* pressure decreasd to **-1 cmH2O** *assuming that the atmospheric pressure is zero.*	decreased volume —> increased pressure (Boyle's law) • *I mean the intrapulmonary (intra-alvelolar pressure)* pressure increasd to **+1 cmH2O** *assuming that the atmospheric pressure is zero.*
Muscles	• Diaphragm: decends. • external intercostals: ◦ —> elevate ribs —> increase transverse diameter. ◦ —> evert ribs —> increases AP diameter	— passive— -diaphragm ascends, lungs shrink by their elastic recoil.
Accessory muscles for forced...	Forced inspiration: • Sternocleidomastoid. • serratous anterior • scalene muscles	Forced expiration (voluntary "musical instuments", obstruvtive [COPD], restrictive [fibrosis]): • internal intercostals • abdominal muscles "abdominal recti" [abdominal breathing]
Effect on	Inspiration accentuates the right-sided murmurs ❤️	Expiration accentuates the murmurs of left side of the heart

توضيح ابرز النقاط بالجدول :

During inspiration: intrapulmonary pressure and intrapleural pressure very very negative

At the end of inspiration: just intrapulmonary pressure approximately equal atmospheric pressure .., intrapleural pressure is negative and will increase slowly to be zero

WHAT IS THE CHARACTERISTIC OF THE LUNG WHICH PRIMARILY RESPONSIBLE FOR INTRAPULMONARY PRESSURE ?

Elasticity ✔

Surfactant: ↓ surface tension

Elastic: ↑ volume

recoil ⟶ collapse

- **In the COPD:** (not normally)

(Elastic will be affected) ⟶ decrease

(volume of the lung) ⟶ increase

The volume greater than pressure So, the lung has a lot of air, that can't exhalation

Intrapulmonary pressure ↓

The compliance ↑

In the opposite side:

- **Fibrosis**

The problem is in the inhale

When the pressure increase (inside the lung) ⟶ ↑ the recoil

But the volume will be decreased

And if the trans molar increase, the recoil Will be increased

• Intra-pleural pressure which is the responsible for a recoil

So, ↑ volume ⟶ ↓ pressure ⟶ ↓ Intrapulmonary pressure.

- Trans molar pressure, that is between Intrapulmonary pressure and Intrapleural pressure.

- Trans molar pressure = Intrapulmonary pressure - Intrapleural pressure

- The volume of the molar pressure approximately equal to Intrapleural pressure (neglecting the reference) , but molar pressure (positive) & Intrapleural pressure (negative)

- Boyle's law: when the chest wall increase, the volume and oxygen volume will be increased. so, the pressure Will be decreased

Transmural pressure getting the lung to smallest size , but intrapleural pressure pulls up the lung out

In obstructive lung disease the transmural pressure decrease and the recoiling decrease

Lung volume and capacities

Volume: one thing

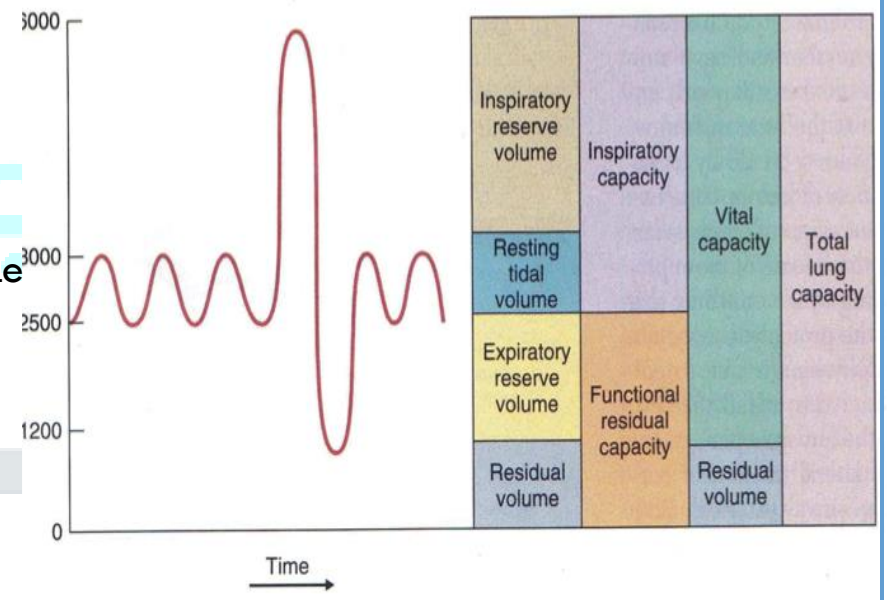
Capacity: more than one volume

Spirometer cannot measure

RV

FRV

TLC



توضيح الشكل :

The subject is asked to take full inspiration as much as he can, go up as far as he can , the volume of the lung then is called the total lung capacity (TLC) , Then the subject is asked to breath out (full expiration) as far as he can and he breaths out all the way down to what called residual volume (RV)

The volume at the end of a normal expiration is larger than the residual volume and called functional residual capacity (FRC)

Vital capacity (VC) = full inspiration + full expiration

** Total lung capacity : vital capacity + FRC

= full inspiration + full expiration + functional residual capacity ☺

- When I want to measure the volume of lung - patient should be in upsetting position , to make sure that their is no effect in diaphragm and the muscles of the neck and the spine are intact

Residual volume

RV: the air that remains in the lung after maximal forced expiration

Physiological significance

Maintain aeration of blood

Prevent sudden flotation of blood gases

Clinical significance

Obstructive lung disease (emphysema)

RV/TLC>30% emphysema normal =21%

Forensic significance

Autopsy

Stillborn lung will sink in water

Child homicide lung float on water

VC= IRV+TV+ERV=4600mL

Male=2.5L/m2

Female = 2L/m2

Note

FVC (Timed) 3600mL and VC (not timed) 4600mL

the lung residual - to keep the lung inflate and prevent collapse

Low vital capacity

استرااحة ☺ بنعرف انكم تعبتم و المكتوب طويل لكن القادم ان شاء يكون أسهل ☺❀

Physiological

- Pregnancy
- Recumbent position

Pathological

- Chest wall
- Deformities in the bone (kyphosis, lordosis…..)
- Neuromuscular (muscular dystrophy, myasthenia gravis……)
- Lung Obstructive (emphysema)and restrictive (pulmonary fibrosis)
- Heart (congestive heart failure) pulmonary edema
- Abdomen Hepatomegaly splenomegaly and ascities

- Getting O2 to alveolar sac and then to capillaries (the most close blood vessel to alveoli) called (perfusion)

Pressure of capillary = Pressure in alveoli

Because they're very close (direct effect)

- Aeration (diffusion) of the blood : (blood gas exchange) in membranes between alveoli and blood vessels

FEV1/FVC ratio

FEV1/FVC ratio: 4L/5L=80%

FEV1: the amount of air quickly and forcibly exhale in 1 second

FVC: the amount of air quickly and forcibly exhale after maximum inhalation (timed)

- low FEV1/FVC ratio -> obstructive lung disease
- low TLC -> restrictive lung disease.
- high FEV1/FVC ratio -> restrictive lung disease
- low FEV1/FVC ratio and low TLC -> mixed lung disease (obstructive + restrictive)

• In order to make the transfer of gases as easy as possible :

1- gases transfer by simple diffusion (no need to ATP) , not by active way

2- large area as possible

- according to fick's law , which increase the diffusion of gas ?

1- surface Area

2- Pressure gradient (P1-P2)

- which decrease the diffusion of gas ?

1- Thickness

2- molecular weight (MW)

Normal range of breath (12 - 18 breaths per minute)

- The sources of O2 in blood are from : ventilation , perfusion and diffusion .

Alveolar ventilation

External respiration

Pulmonary Ventilation

Alveolar ventilation

Perfusion

Diffusion

Internal respiration

Mitochondria

FIO2 PAO2 PaO2 SaO2 Carbmino-Hgb PVO2 CO2 breathout

IF WE DO PNEUMOECTOMY TO ONE LOBE FROM THE LUNG , WILL BREATHING BE AFFECTED ?

No , because , each lobe has its own blood circulation and JUST the surfactant will be affected , but this doesn't affect the O2 & CO2

Alveolar capillary membrane diffusion (blood gas barrier)

Very thin Fibrosis = 1 micron

Huge surface area Pneumonectomy " Restrictive"

6 layers

Fluid lining the alveoli Pneumonia (consolidation)

Alveolar epithelium Emphysema

Epithelial basement membrane fibrosis

Fluid in interstitial space capillary endothelium pulmonary edema

Endothelium basement membrane

Simple diffusion no ATP

CO2 out

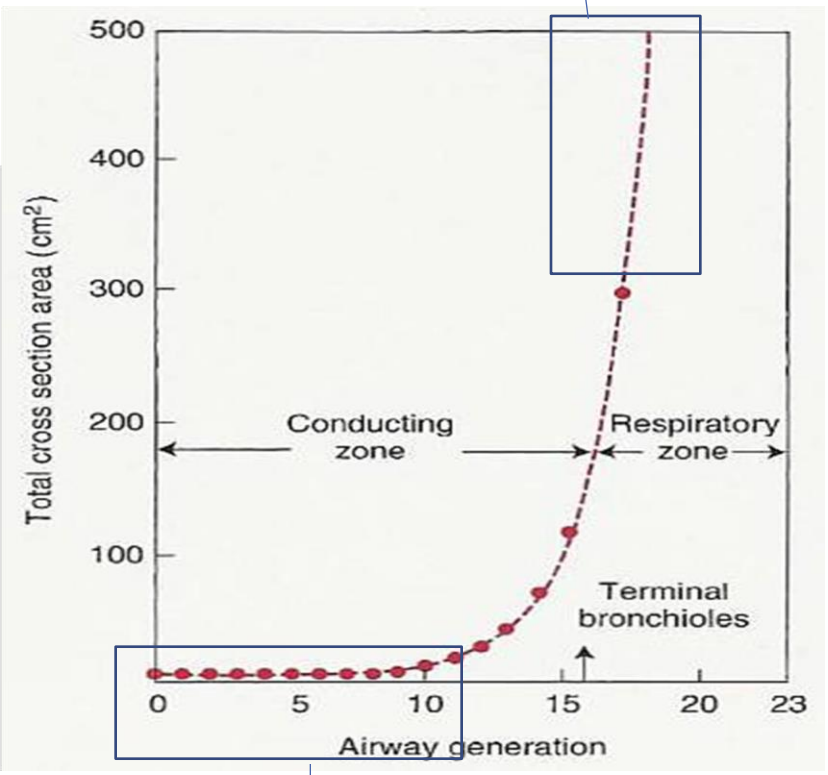
Narrow range of PH 7- 7.7

Cross sectional area of the airways

The extremelv rapid increase in total cross-sectional area of the airways in the lower respiratory zone

It is look like trumpet بوق

When we blow in it , it will start from very very small area and then increasing enormous area



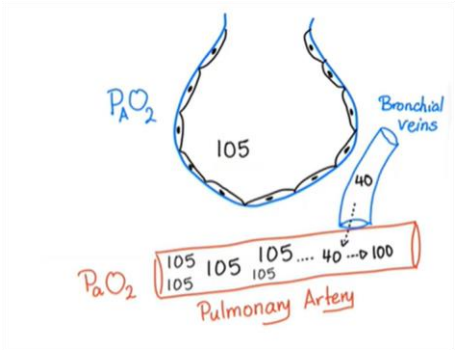
In first few generation , the total cross sectional area doesn't change , It will start very very small tell reach 10 generation , and then it will increase till reach 16 generation —> flaring out —> very very increase in cross sectional area

In people who smoke, the dust stuck in the terminal bronchioles

BUT , WHY IT DOESNOT REACH THE RESPIRATORY ZONE ?

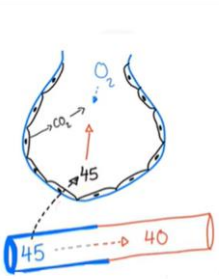
THE MAIN FUNCTION OF RESPIRATORY IS ?

Getting CO2 out of the body , in order not to affect the PH —> intact proteins



$$P_AO_2 = [(P_b - P_{H_2O}) \times F_iO_2] - [\frac{P_aCO_2}{0.8}]$$

P_iO_2 P_ACO_2



A-a gradient normal extrinsic restrictive lung disease
A-a gradient abnormal intrinsic restrictive lung disease

Not all O2 in alveoli will reach to capillaries !

Little bit of O2 will thaw

In pulmonary circulation : part of blood pumping by aorta to the lung and the another part of blood from bronchial veins to the pulmonary veins (dual circulation or collateral circulation)

Patient A "normal"

RR= 12/min, TV = 500 mL

• Pulmonary ventilation = respiratory rate x tidal volume = 12x 500 = 6 liters.

Alveolar ventilation = respiratory rate x (tidal volume - dead space) = 12x (500-150) = 12x350 = 4.2 liters.

- فيه شغلة بسميها ال respiratory minute volume و اللي هي وانا قاعد حاليًا كم بتنفس هواء ؟؟ و هو تقريبا 6000 يعني 6 لتر, كيف طلعناها ؟
هسا احنا بنتنفس 12 مرة و كل مرة باخذ tidal volume >> نص لتر (500 ملي لتر هواء)
12 * 500 = 6000 ملي لتر = 6 لتر
لكن مش كل هذا الغاز يصل إلى منطقة الرئة فيه , فكم بشيل منه ؟
بحكي ال 6000 بشيل منهم (12 عدد مرات التنفس * 150 يلي هو ما بيوصل عندي للرئة عند كل مرة بتنفس فيها) = 6000 – 1800 = 4200 ملي لتر

Patient B "increased respiratory rate"

• RR = 30/min, TV = 200 mL

• Pulmonary ventilation = respiratory rate x tidal volume = 30 x 200 = 6 liters

• Alveolar ventilation = respiratory rate x (tidal volume - dead space) = 30 x (200 - 150) = 1,5 liters.

Here we increase the respiratory rate to 30

Patient C "increased tidal volume"

• RR = 6/min. TV=1,000 mL

• Pulmonary ventilation = respiratory rate x tidal volume = 6x 1000 = 6 liters.

• Alveolar ventilation = respiratory rate x (tidal volume - dead space) = 6x (1000-150)= 5.1 liters.

Therefore, increasing the tidal volume is a better way to achieve more alveolar ventilation than increasing the respiratory rate.

But, everything is good within limits.

• if you increase the tidal volume too much -> the alveoli will expand tremendously (inspiration) and then

collapse (expiration)…. This big difference, repeated over and over again, can lead to INFLAMMATION!

Here we increase the tidal volume to 1000

In conclusion we notice that if we want to increase the respiratory rate we should increase the tidal volume

Valsalva maneuver : occurres when getting birth and in constipation

—> then the intrapleural pressure will be positive (less negative)

ذاكر وانت بتعيط

مفيش وقت