MED437
KING SAUD UNIVERSITY

437
PHYSIOLOGY TEAM

Function & Organization of The Respiratory system

➢ Color index:

Red: important
Green: doctor's notes
Grey: extra information
Pink: found only in female's slides
Blue: found only in male's slides
Yellow: numbers

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Physiology 437 teamwork

Editing file

objectives:

By the end of the lecture you will be able to:

1-Describe the structures and respiratory zones functions of the conductive and of airways.

2-Understand the difference between internal and external respiration.

3-Understand the functions of the respiratory system, including non-respiratory functions, like clearance mechanism by mucus and cilia, production of surfactant and its physiological significance.

The Main Goal of Respiration:

Only in female's slides

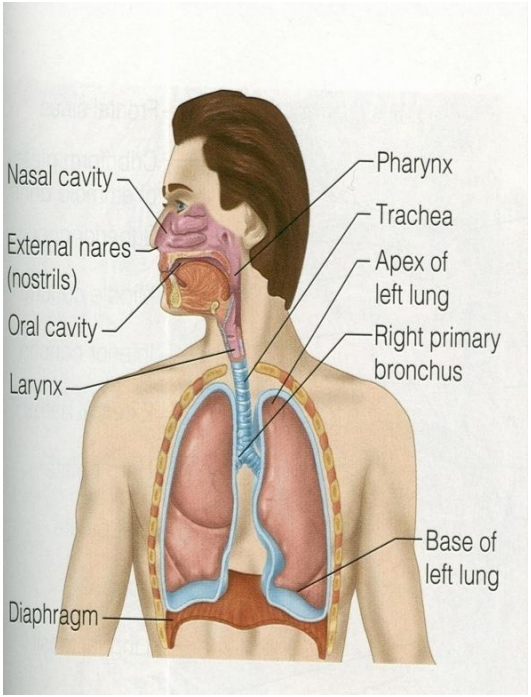
Overview "Aremando"

1- to provide oxygen to tissue.

2- remove CO_2.

Respiratory system consists of:

- Passages (airways)
- Muscles
- Centers



Functions of the respiratory system include:

Gas exchange: Respiratory function.

Non respiratory functions:

phonation: Is the production of sounds by the movement of air through the vocal cords.

Pulmonary defense:

- Immunoglobulin A (IgA).
- Alpha-1 antitrypsin.
- The pulmonary macrophages in the alveoli: engulf smaller particles which pass through the muco-cilliary barrier filter.

يعتبر الجهاز التنفسي هو خط الدفاع الثاني بعد الجلد فلابد أن يكون فيها أدوات دفاع ومن أمثلة هذا أن لما شخص يسافر ويتغير عليه الجو يصير له انفيكشن - بالعادة تسبب له الي الانفكشن كان يقضى عليها في البولمناري لكن هذي جديدة ما تعود عليها-

Immunoglobulin A (IgA)
A type of antibody that protects against infections of the mucous membranes lining the mouth, airways and digestive tract. It is the most common of the primary antibody deficiencies, IgA is the predominant Ig isotype in mucosal tissue and is believed to be involved in defense against viral and bacterial infections at these sites.
Alpha-1 antitrypsin
Is generally known as serum inhibitor, it protects tissue from enzymes of inflammatory cells.

- In respiratory tract infections bacteria produce trypsin a proteolytic enzyme (digests proteins) which will digest the structures of the respiratory system because they're made of proteins, so the body will produce Antitrypsin for protection against it.

Cont..non respiratory functions of lung

- Angiotensin I (in blood in inactive form) is converted to angiotensin II[1] with the help of angiotensin (peptide hormone regulates the blood pressure) converting enzymes formed by the lungs

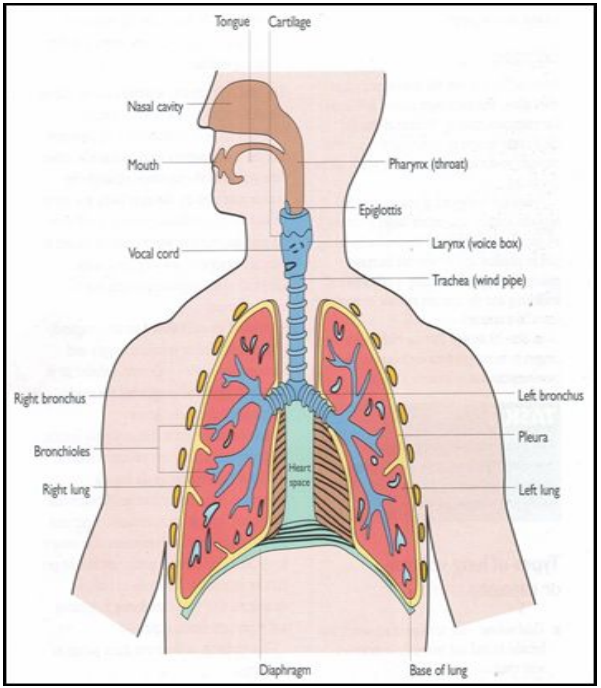
- Regulating the acid- base status of the body by washing out extra carbon dioxide[2] from the blood.

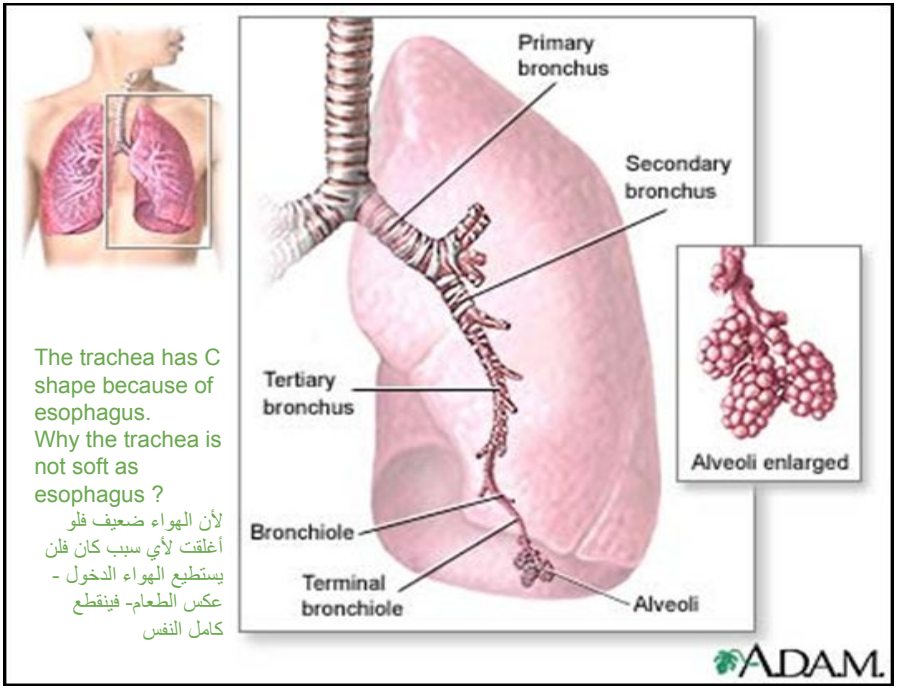
  High H+ > low PH+ > more Acidity (acidosis)
  Low H+ > high PH+ > more basal (Alkalosis)
  - We have one lecture about this concept

- Secretion of important substances like surfactant.

1: Angiotensin II plays a role in regulating the blood pressure, it's a vasoconstrictor which will lead to increasing the blood pressure.
2: Carbon dioxide is a volatile acid, removing it will decrease the acidity in blood. (pH regulation)

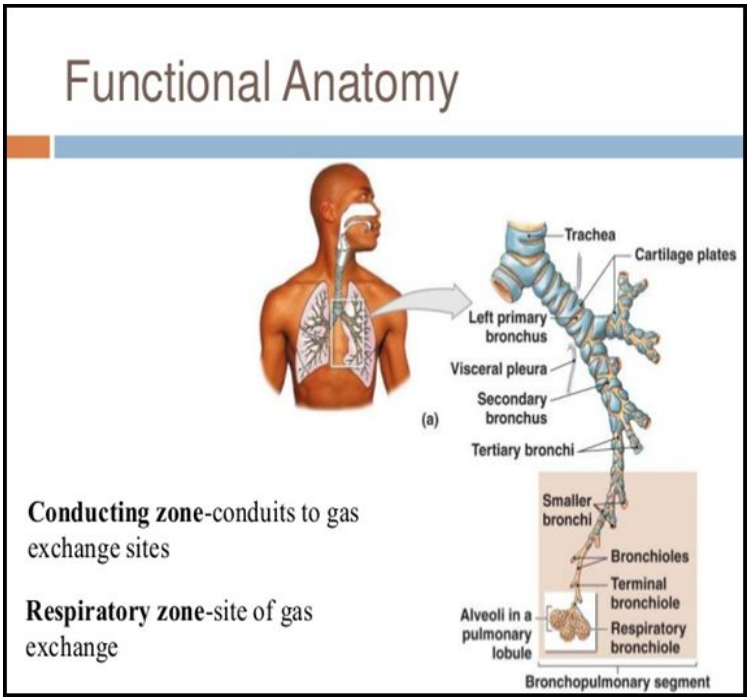
Respiratory passages (airways)

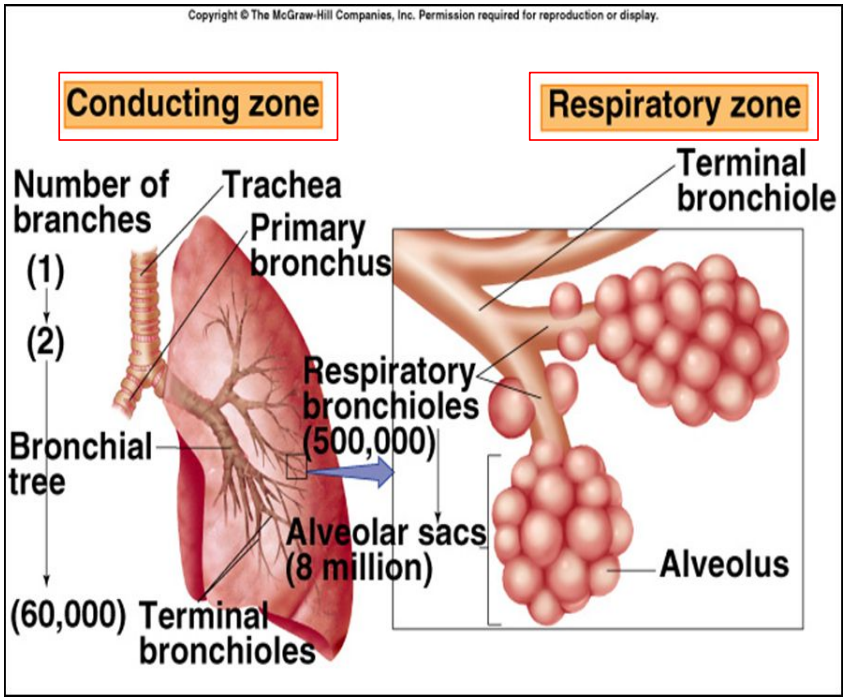




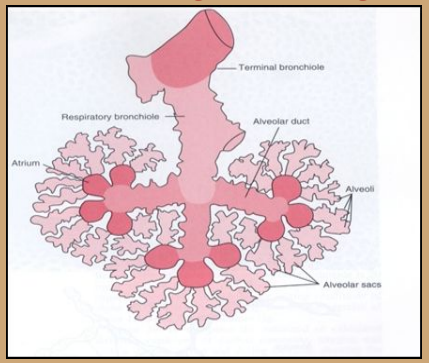
The trachea has C shape because of esophagus.
Why the trachea is not soft as esophagus ?
لأن الهواء ضعيف فلو أغلقت لأي سبب كان فلن يستطيع الهواء الدخول - عكس الطعام- فينقطع كامل النفس

Respiratory passages airways can be divided into





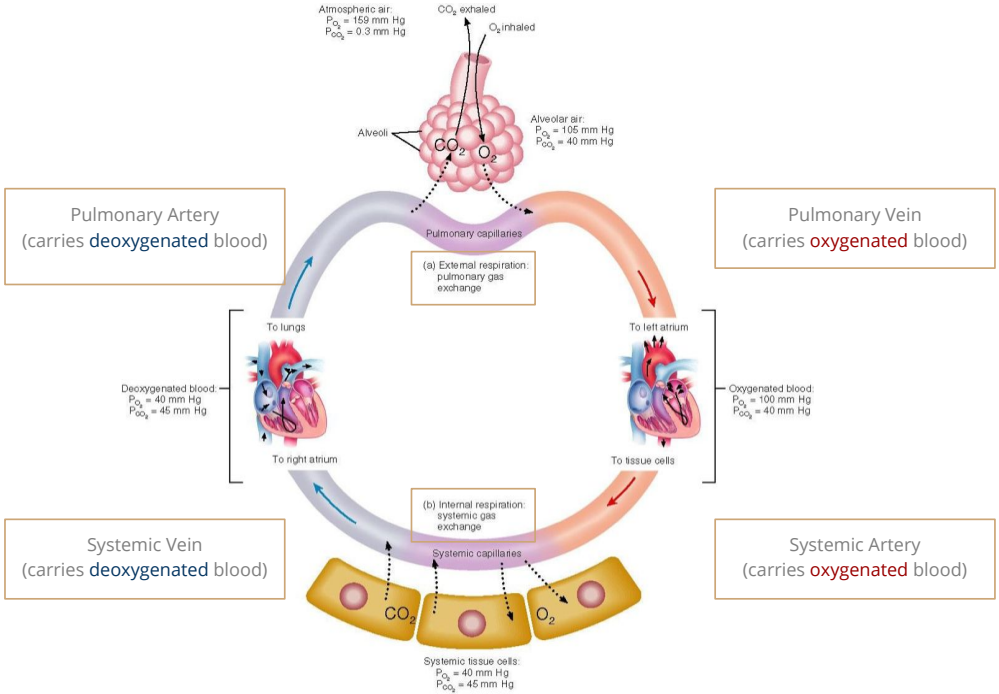
Respiratory passages (airways) can be divided into:

	1- Conductive Zone (مجرد ناقل not respiratory function)	2- Respiratory Zone (Respiratory unit):
Functions of each zone	○ *Help warming, humidification, filtration of inspired air.* ○ *Contains the olfactory receptors for smell sensation.* ○ *Conducts the sound during speech. (Phonation)* ○ *Protective function by cough and sneezing reflexes.*	○ Function in gas exchange. 
Structures in each zone	Starts from nose to the end of terminal bronchioles.	○ Respiratory bronchioles. ○ Alveolar ducts. ○ Alveolar sacs. ○ Alveoli.

*The rate at which new air reaches these areas is called **alveolar ventilation.** Or respiratory zone*

Internal & External Respiration

External Respiration (Gas exchange at lung level)	**Internal Respiration** (Gas exchange at tissue level)
Pulmonary gas exchange	Systemic gas exchange
Pulmonary artery carries deoxygenated blood	Systemic artery carries oxygenated blood
Pulmonary vein carries oxygenated blood	Systemic vein carries deoxygenated blood



Internal & External respiration

3 major functional events occurs during it:

•*1-**Pulmonary ventilation***: inward and outward movement of air between lung and atmosphere. (External respiration)

•2- ***Diffusion*** of oxygen and CO2 between the alveoli and the pulmonary capillary blood. (Internal respiration)

•3- ***Transport*** of O2 & Co2 in the blood and body fluids to and from the cells. (Internal respiration)

Respiration could be either :

Resting : normal breathing during resting conditions

Forced (maximal): during exercise, in patients with asthma, allergy,...etc.

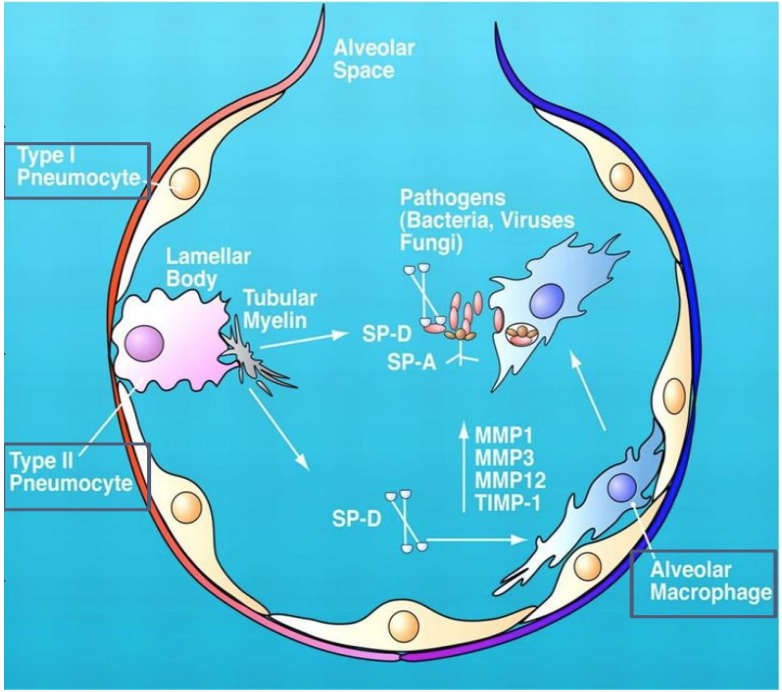
- 4th function is regulation (found in some books)

Respiratory Membrane: The area where gas exchange between air and blood occurs.
It is the fused alveolar and capillary walls (3 layers):

- Type 1 alveolar epithelium
- Fused basal laminae
- Squamous endothelial cells in pulmonary capillaries

Lining cells of the alveoli

Type I alveolar cells	- Also called Type I pneumocyte. - Participate in the respiratory membrane.
Type II alveolar cells	- Also called Type II pneumocyte. - Secretes surfactant.
Alveolar macrophages	Engulfs the foreign bodies that reach the alveoli.



يقل مع العمر Surfactant

Surface Tension (التوتر السطحي)

H2O molecules at the surface are attracted to other attractive forces that resist distension (inflation) called **surface tension**.

Surface tension is the attractive force between adjacent water droplets.

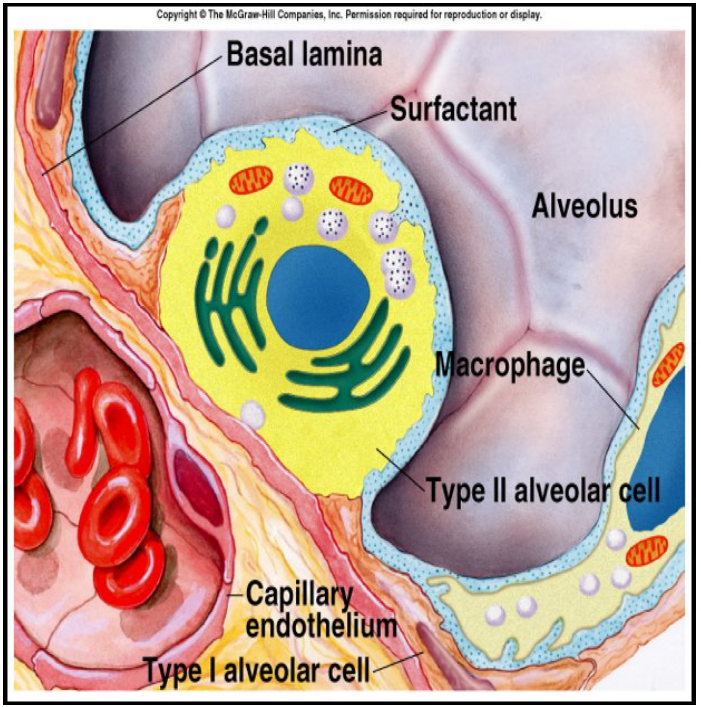
Air increases the surface tension.

Surface tension tends to oppose alveoli expansion

$$\text{pressure} = \frac{(2 \times \text{surface tension})}{\text{radius}}$$

Pulmonary surfactant reduces surface tension.

- Without surfactant the alveoli collapse.
- Surface tension tends to force air out of the alveoli through the bronchi and, in doing so, causes the alveoli to try to collaps



Surfactant

Will be discussed in biochemstry in details

- **Surfactant** is a complex substance containing phospholipids and a number of apoproteins.
- It lines the alveoli from the inside,which separates the air found in the lumen of the alveoli from water droplets on the cells
- **Secreted** by the Type II alveolar cells" Type II pneumocyte" { the earliest detection begins between 6-7th month much as the earliest detection but this could be delayed in others} at wk 35 of intrauterine life from fetal alveoli.
- **Surfactant's** functions are reduces surface tension throughout the lung, prevents alveolar collapse, decreases airway resistance and the work of breathing.
- Surfactant is a complex mixture of several phospholipids, proteins, and ions. The most important components are the phospholipid *dipalmitoylphosphatidylcholine, surfactant apoproteins*, and *calcium ions*. The dipalmitoylphosphatidylcholine and several less important phospholipids are responsible for reducing the surface tension.

- Deficiency of surfactant in premature babies cause respiratory distress syndrome of the newborn (RDS). (hyaline membrane disease)
- Smoking in adult, hypoxia(low oxygen concentration reaching the tissues) or hypoxemia (low oxygen in the arterial blood) or both, these can decrease surfactant secretion and cause adult respiratory distress syndrome.

How is surfactant related to the work of breathing? *(TEAM436)*

- More surfactant -> less resistance -> air flows in easily -> with one breath a good amount of air flows in (e.g. 0.5 ml) -> less energy -> less work.
- Less surfactant -> more resistance -> air **does not** flow in easily -> you need more breaths to be able to reach the amount of air taken in by one normal breath (e.g. 0.5 ml) -> therefore putting in more energy -> more work.

example of deficiency of surfactant.

- In **neonatal respiratory distress syndrome** surfactant is lacking. In the developing fetus Infants born before week 24 will *never* have surfactant. Without surfactant, small alveoli have increased surface tension and increased pressures, and will **collapse** (**atelectasis**). Collapsed alveoli are not ventilated and, therefore, cannot participate in gas exchange

- Distress Syndrome of the new born is a problem often seen in premature babies.The condition makes it hard for the baby to breathe.This disease is mainly caused by a lack of a oily substance in the lungs called **Surfactant**. This substance helps the lungs fill with air and keeps the air sacs from deflating. Surfactant is present when the lungs are fully developed.

Treatment:

- Ventilator
- Synthetic Surfactant

Glucocorticoids enhance the maturation of surfactant in the baby, so they're given to pregnant women expected to deliver prematurely.

Innervations of lungs and bronchi

- Innervated by autonomic system .
- Sympathetic stimulation causes dilatation of the bronchi.
- Parasympathetic stimulation causes constriction of the bronchi.
- Locally secreted factors :Histamine, slow reacting substances of anaphylaxis (SRSA) is formed by mast cells, due to allergy* (as in patients with asthma).Histamine often cause bronchiolar constriction and increase airway resistance.

*الحساسية قد تكون مكتسبة لما تتعرض لمحفز فترة طويلة فلما يجي مرة ثانية (ولو بكميات بسيطة) يقوم الجسم بافراز الهيستامين بكميات كبيرة كنوع من الحماية مع أنه ما فيه حاجة.

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Mechanics Of Pulmonary Ventilation

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Objectives:

By the end of the lecture you will be able to:

- List the muscles of respiration and describe their roles during inspiration and expiration.
- Understand the importance of the following pressures in respiration: atmospheric, alveolar, intrapleural, and transpulmonary.
- Explain why intrapleural pressure is always subatmospheric under normal conditions, and the significance of the thin layer of the intrapleural fluid surrounding the lung.
- Define lung compliance and list the determinants of compliance.

Muscles That Cause Lung Expansion and Contraction

Lungs can be expanded and contracted:

Expansion of the lungs (in inspiration)	**Contraction of the lungs (in expiration)**
Downward movement of the **diaphragm** will <u>lengthen</u> the chest cavity (vertically).	**Upward** movement of the **diaphragm** will <u>shorten</u> the chest cavity (vertically).
Elevation of the **ribs**, will <u>increase</u> anteroposterior diameter of chest cavity.	**Depression** of the **ribs** will <u>decrease</u> the anteroposterior diameter.

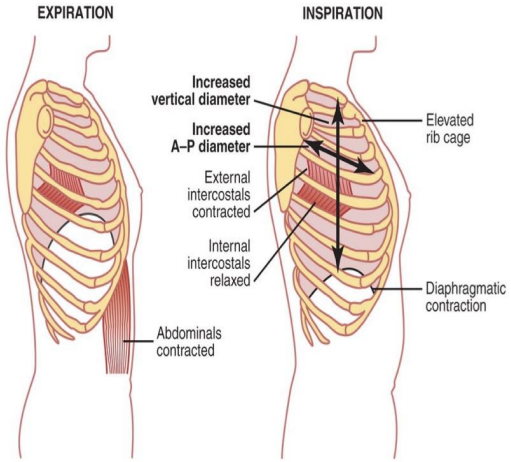


Figure 37-1 Contraction and expansion of the thoracic cage during expiration and inspiration...

Only in male's slides.

Respiratory Muscles

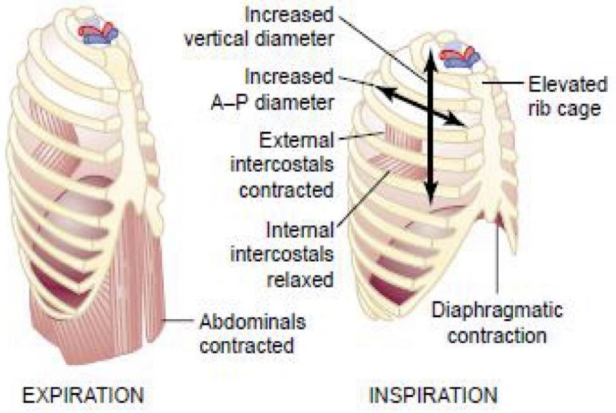
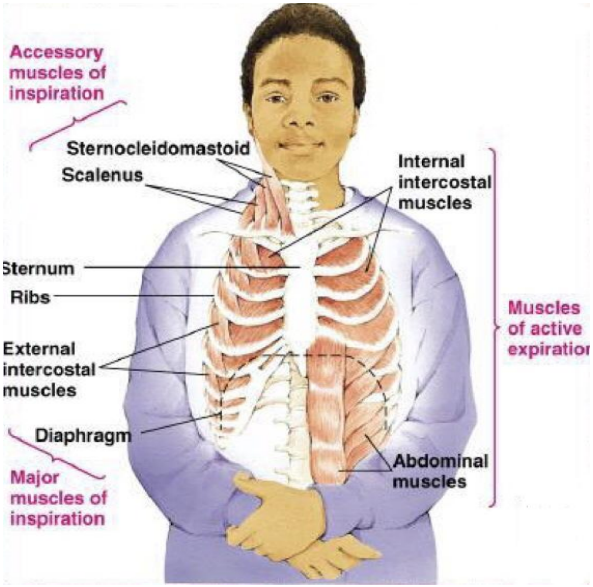


Figure 37–1

Contraction and expansion of the thoracic cage during expiration and inspiration, demonstrating diaphragmatic contraction, function of the intercostal muscles, and elevation and depression of the rib cage.



Only in female's slides.

Respiratory Muscles

Inspiratory muscles

During **resting** inspiration :
1. Diaphragm
2. External intercostals

During **forced** inspiration :
A- Accessory muscles of inspiration:
1. Sternomastoid
2. Anterior serratus
3. Scalene muscles contract

B- Muscles of resting inspiration

making the anteroposterior thickness of the chest about 20 percent greater during maximum inspiration

Expiratory muscles

Resting expiration is a **passive** process that depends on the **recoil tendency of the lung** and need **no** muscle contraction.

Forced expiration is **active** and need contraction of the
1. Abdominal muscles
2. Internal intercostal muscles

Deep Forceful Breathing

- **Deep Inspiration**

During deep forceful inhalation **accessory muscles** of inspiration participate to increase size of the thoracic cavity.

- **Sternocleidomastoid** elevate sternum
- **Scalene** elevate first two ribs
- **Pectoralis minor** elevate 3rd–5th ribs

- **Deep Expiration**

- Expiration during forceful breathing is active process.
- Muscles of exhalation increase pressure in abdomen and thorax.

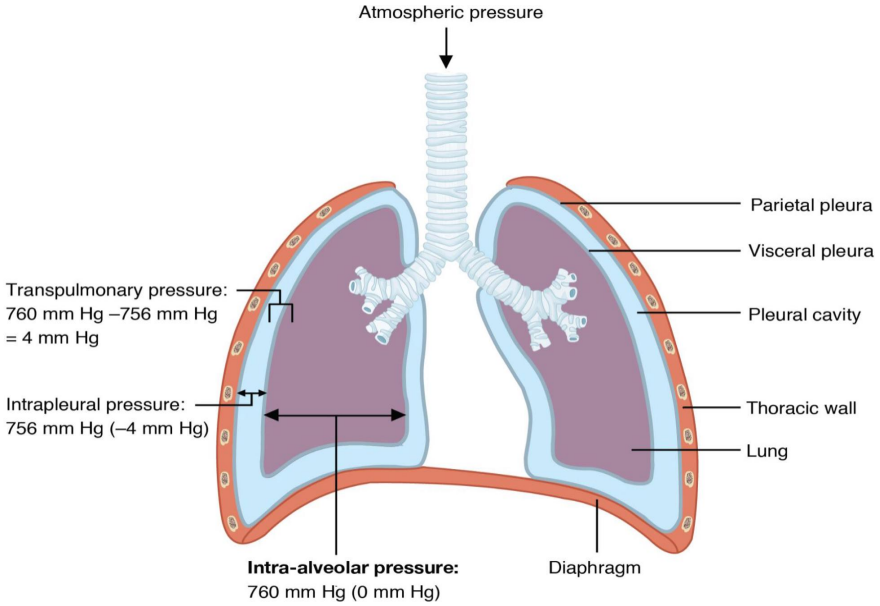
- **Abdominal muscles**
- **Internal intercostals**

Pressure changes in the lungs during breathing

Air will flow from a region of high pressure to one of low pressure-- the bigger the difference, the faster the flow.

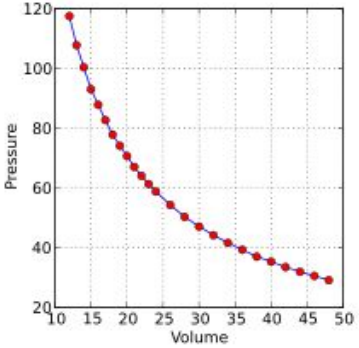
Boyle's law: the pressure and volume of a gas have an inverse relationship.

Mechanism of breathing:

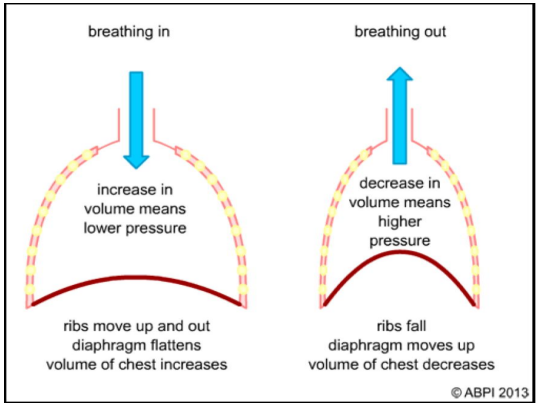


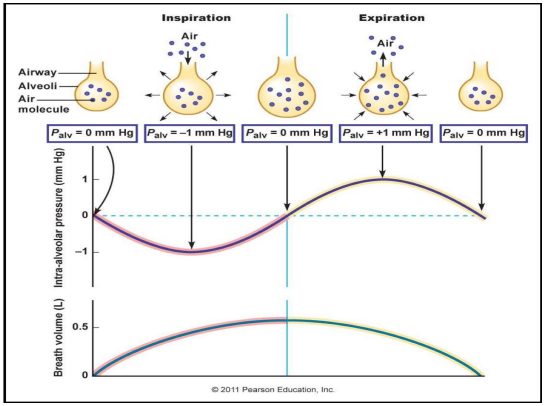
Extra explanation

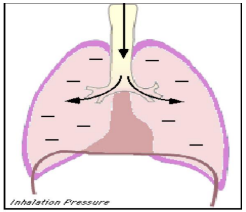
قانون بويل باختصار هو : أنه كلما زاد حجم الغاز كلما قل ضغطه .. في التنفس نستخدم العضلات -ذكرت في السلايدات السابقة- لزيادة الحجم > إذا زاد الحجم الثوراكس قل الضغط الهواء فيها > بما أن الغاز ينتقل من الضغط الأعلى(الهواء الجوي) للأقل(الرئة) فإن الهواء سيدخل الرئة وبكذا حصل الانسبيرايشن > العضلات يصير لها ريلاكس > يقل الحجم > يكون الضغط أعلى من الهواء الجوي > اخراج الهواء "اكسبيرايشن"

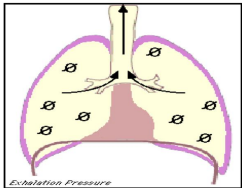


1-Intra-alveolar pressure (intrapulmonary pressure)



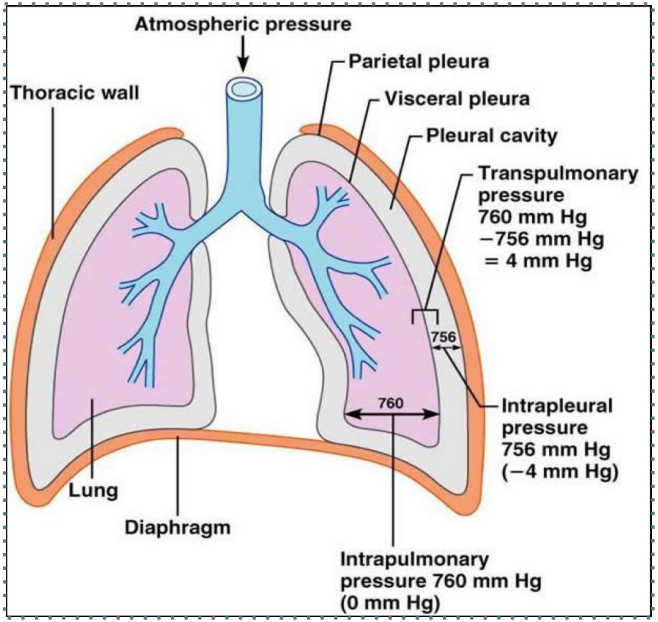


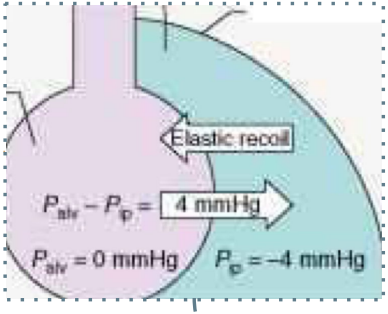




- **Between breaths** = zero pressure
- **During inspiration** = (-1 mmHg) and air (tidal volume) flows from outside to inside the lungs.
  Diaphragm contracts, pressure decreases and volume increases (Boyle's law), and air will flow from a region of high pressure (Outside) to lower pressure (Inside the lungs).
- **At the end of inspiration** = zero and air flow stops, because the pressure inside is equal to outside.
- **During expiration** = (+1 mmHg) and air flows out of the Lungs.
  Diaphragm relaxes, pressure increases and volume decreases (Boyle's law), and air will flow from a region of high pressure (Inside the lungs) to lower pressure (Outside).

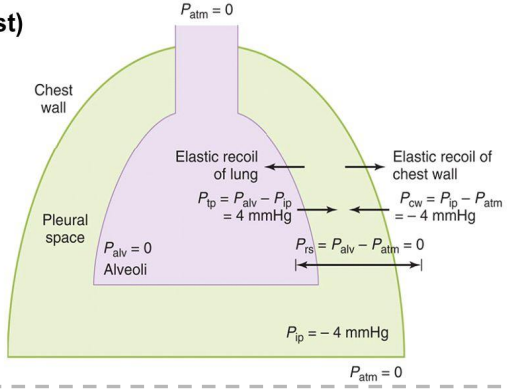
2-Intrapleural pressure





Extra for better understanding :

Elastic recoil: the tendency of an elastic structure to oppose stretching or distortion.



Only in female's slides.

2-Intrapleural pressure (IPP):

- Pressure in the pleural space is negative with respect to atmospheric pressure at the end of normal expiration (-5cmH2O).
- **Why negative?** (By negative we mean in comparison to atmospheric pressure.)

  1- The lung's elastic tissue causes it to recoil, while that of the chest wall causes it to expand. Because of these two opposing forces the pressure in the pleural cavity becomes negative.

  2- The pleural space is a potential space[1], (empty) due to continuous suction of fluids by lymphatic vessels.

  At all times: the chest is trying to inflate and the lung is trying to collapse.

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Malignancies, heart failure, obstruction of lymphatics or inflammation of pleura and production of more pleural fluid will cause accumulation of pleural fluid (pleural effusion) which is very dangerous and requires immediate suction of fluid because it erases the negativity. No negativity means no opposing force so the lung will collapse.

A layer of pleura (a very thin membrane) covers the lung (this layer is called visceral pleura) and another layer covers the inside of the ribs (parietal pleura), these two layers move together to make the movement of the lung non-painful. Between the two layers there is a very thin lubricant fluid film. The space between the two layers is called the pleural space (pleural cavity).

[1]: Potential space A region of the body in which two surface membranes adjoin, separated only by a small amount of fluid.

Values of Intrapleural Pressure (IPP):

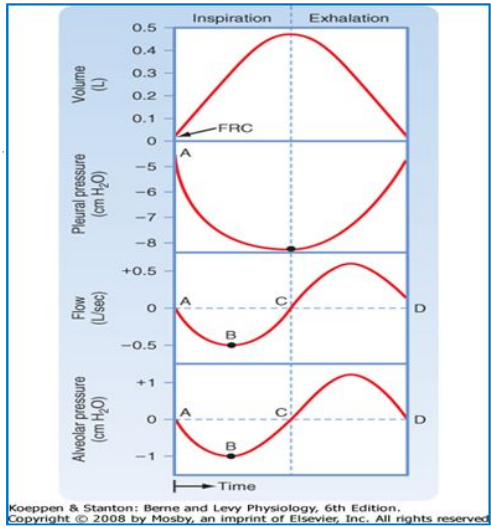
- **During resting position** between breathes it is = (-5) cm H2O.
- **During resting inspiration** it becomes more –ve (-7.5) cm H2O.

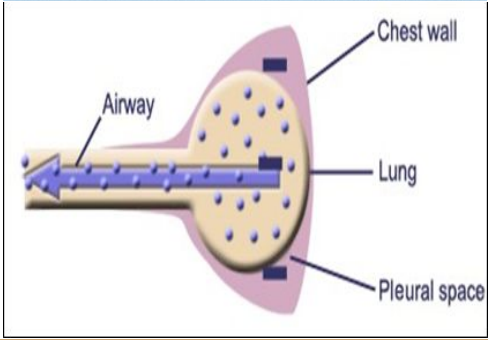
> *Dr. Aida: Focus only on values during resting breathing (-5, -7.5) cause they're the only ones written in guyton.*

- **During forced ventilation:**

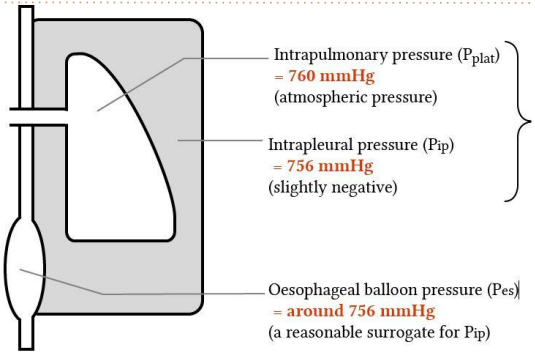
Insp. :-20 to - 40 cm H2O

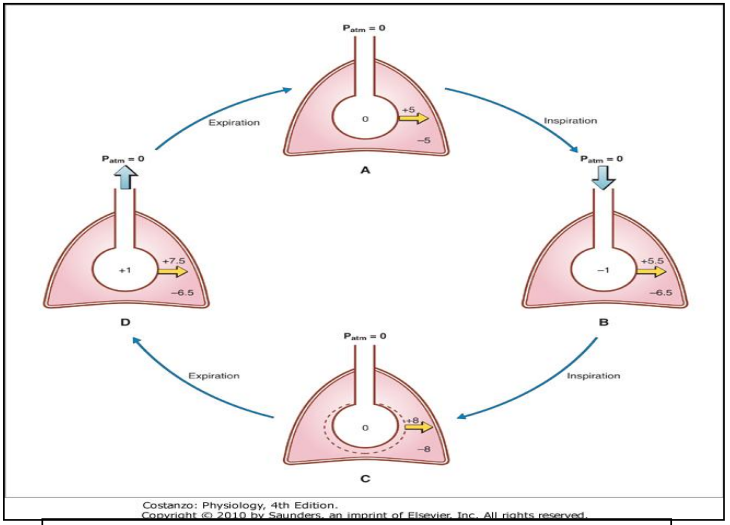
Exp. : + 30 cm H2O



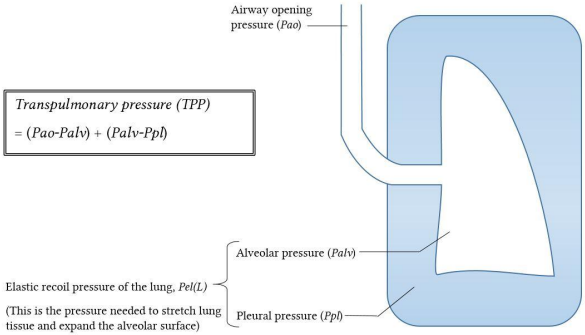


3-Transpulmonary Pressure (TPp) (Extending Pressure)





pressure and volume relationships in a single respiratory cycle.



3-Transpulmonary Pressure (TPp) (Extending Pressure)

- "Extending" pressure because it extends the lungs = prevents collapse of lungs.
- It is the difference between the alveolar pressure (*Palv*) and the pleural pressure (*Ppl*).
- *TPp = Palv-Ppl*
- It is a measure of the elastic forces in the lungs that tend to collapse the lungs (the recoil pressure).
- It prevents lung collapse.
- The bigger the volume of the lung; the higher will be its tendency to recoil.
- A change in volume always follows a change in pulmonary pressure.

Compliance of the Lung (CL)

- The extent to which the lungs will expand for each unit increase in transpulmonary pressure is called the lung compliance.
- CL = (Δ V)/(Δ P)

i.e the ratio of the change in the lung volume produced per unit change in the distending pressure.

- For both lungs in adult = 200 ml of air /cm H20. For lungs and thorax together = 110 ml/cm H20.

E.g. two rubber bands, thin and thick. The thin rubber band easily stretched, and is very distensible and compliant. The thick rubber band difficult to stretch and is less distensible and less compliant.

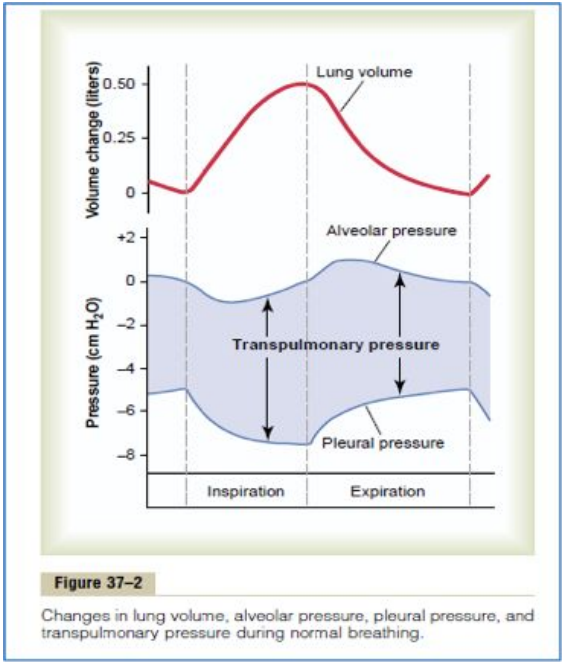


Figure 37–2

Changes in lung volume, alveolar pressure, pleural pressure, and transpulmonary pressure during normal breathing.

Compliance diagram of the lung

- The characteristics of the compliance diagram are determined by the elastic forces of the lungs. These can divided into:

(1) 1/3 is due to elastic forces of the lung tissue itself (elastin, collagen).

(2) 2/3 of the elastic forces caused by surface tension of the fluid that lines the inside walls of the alveoli and other lung air spaces.

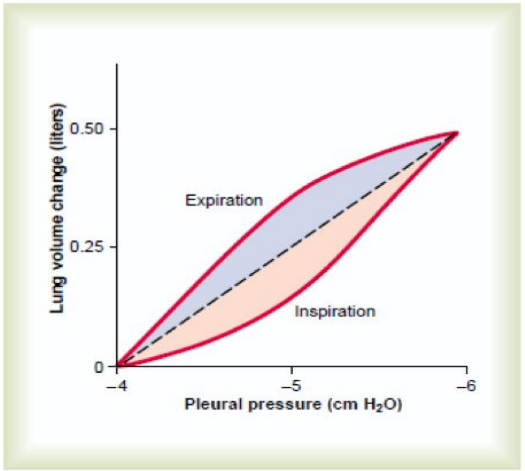


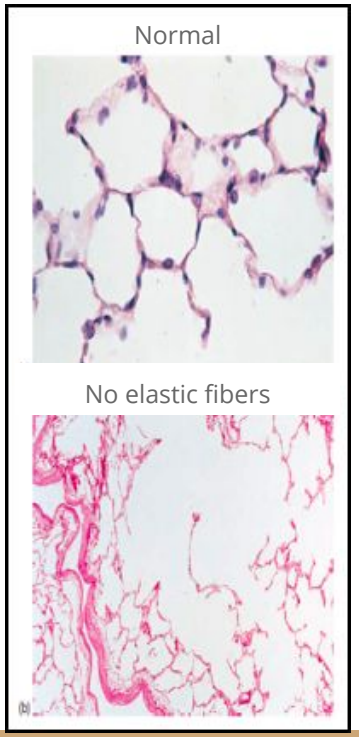
Figure 37–3

Compliance diagram in a healthy person. This diagram shows compliance of the lungs alone.

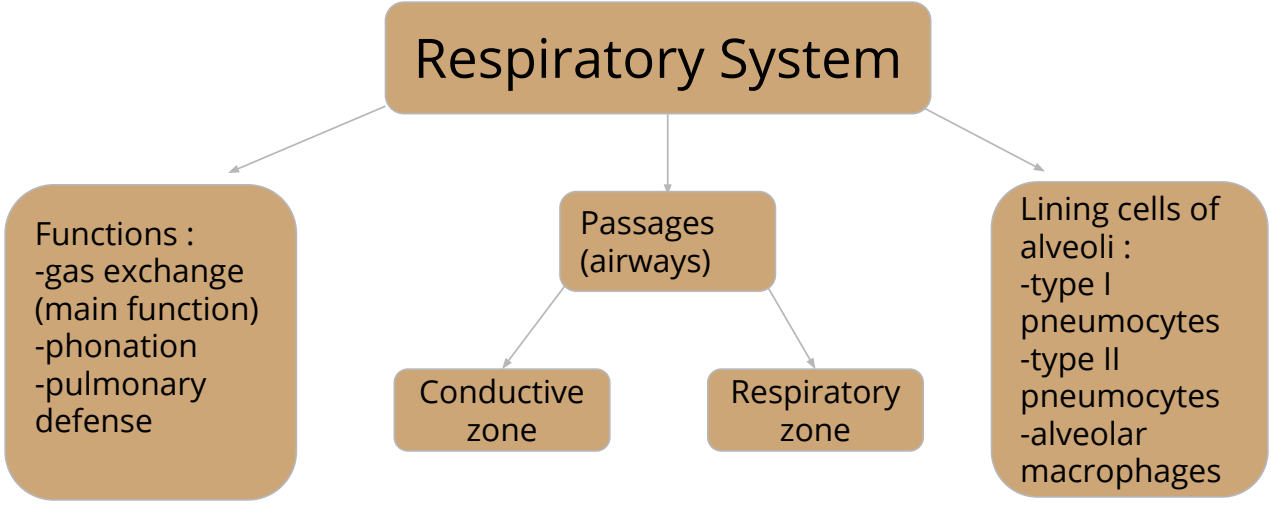
Only in Female's slides

Diseases that affect compliance of lung

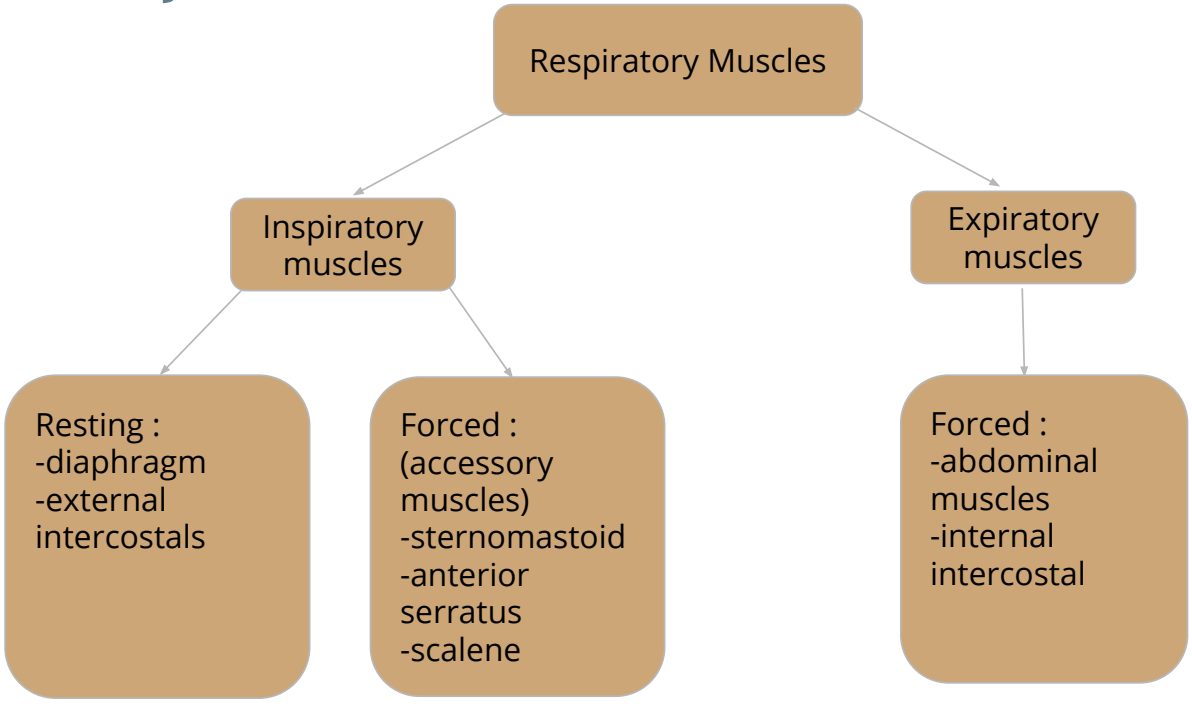
- Higher compliance is better because the main function of the lung is to increase the volume to accommodate more air. So anything that increases the compliance is a good thing, and anything that decreases the compliance is a disease.
- All respiratory diseases limit expansion, so there will be a decrease in compliance, ***except*** for Emphysema (increase compliance)**!**
- Lung compliance is reduced in pulmonary fibrosis , pulmonary edema, diseases of the chest wall (kyphosis, scoliosis).
- Emphysema increases the compliance of the lungs because it destroys the alveolar septal tissue rich with elastic fibers that normally opposes lung expansion.



Summary :


Respiratory System
Functions :
-gas exchange (main function)
-phonation
-pulmonary defense
Passages (airways)
Conductive zone
Respiratory zone
Lining cells of alveoli :
-type I pneumocytes
-type II pneumocytes
-alveolar macrophages

Summary :


Respiratory Muscles
Inspiratory muscles
Expiratory muscles
Resting :
-diaphragm
-external intercostals
Forced :
(accessory muscles)
-sternomastoid
-anterior serratus
-scalene
Forced :
-abdominal muscles
-internal intercostal

Quiz

MCQ:

(1) The Surfactant is produced by which of the following cells ?

A) Alveolar macrophages
B) Type l alveolar cells
C) Type ll alveolar cells
D) Red blood cells

(2) Intrapleural pressure is negative due to?

(A) Opposing forces of recoil and expansion force of the chest wall
(B) The suction of pleural fluid by lymphatics
(C) Both

SAQ:

(3) List three functions of Conductive Zone:

(4) List the function and the structures of Respiratory Zone:

Answers:
(1): C
(2): C
(3):
- Help *warming,humidification, filtration* of inspired air.
- *Conducts the sound during speech.*
- Protective function by cough and sneezing reflexes.
- Contains the *olfactory receptors* for smell sensation.

(4):
Function :
- gas exchange.

Structures :
- Respiratory bronchioles.
- Alveolar ducts.
- Alveolar sacs.
- Alveoli.

Female's team:

Leader: Alanoud Salman Alotaiby

Members:

Dimah Alaraifi

Rahaf AlShammari

Hadeel Awartani

Maha Alnahdi

Rinad Alghoraiby

Majd AlBarrak

Maha Barakah

Male's team:

Leader: Abdulhakim AlOnaiq

Members:

Naif Almutairi

Abdullah Alzaid

Saad Alfawzan

Saud Alatawi

Khali Alogaily

Saif Almeshari

Fahad Alhussain

Abduljabbar Alyemani