




I

# Functional organization of the respiratory system

-  Very important
-  Extra information
-  Terms

Forget past mistakes forget failures.  
Forget everything except what you're  
going to do now and DO IT!

# Objectives

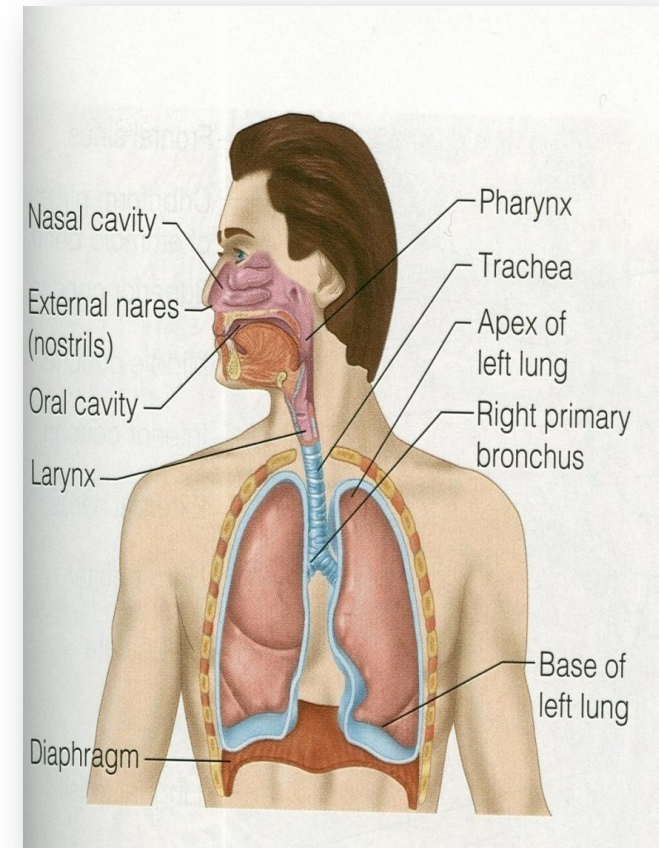
- 1-Describe the structures and respiratory zones functions of the conductive and of airways.**
- 2-Understand the difference between internal and external respiration.**
- 3-Understand the functions of the respiratory system, including non-respiratory functions, like clearance mechanism by mucus and cilia, production of surfactant and its physiological significance.**

# The main goal of respiration

- 1- Provide oxygen to tissues.
- 2- Remove CO<sub>2</sub>.

This process is done by the **Respiratory system**.  
And respiratory system consists of:

- **Passages (airways)**
- **Muscles (The power that allow air to enter in and out)**
- **Centers (To regulation the concentration)**



## Functions of the respiratory system

1- Gas exchange.

2- Phonation.

3- Pulmonary defense.

4- Metabolism.

5- Regulation.

6- Secretion.

# Functions of the respiratory system

## 1- Gas exchange

It's the main function of respiratory system. "Respiratory function."

## 2- Phonation

-**The production of sounds** by the movement of air through the vocal cords.  
-The main part of phonation is the **Larynx**.

## 3- Pulmonary defense

**By two ways :**

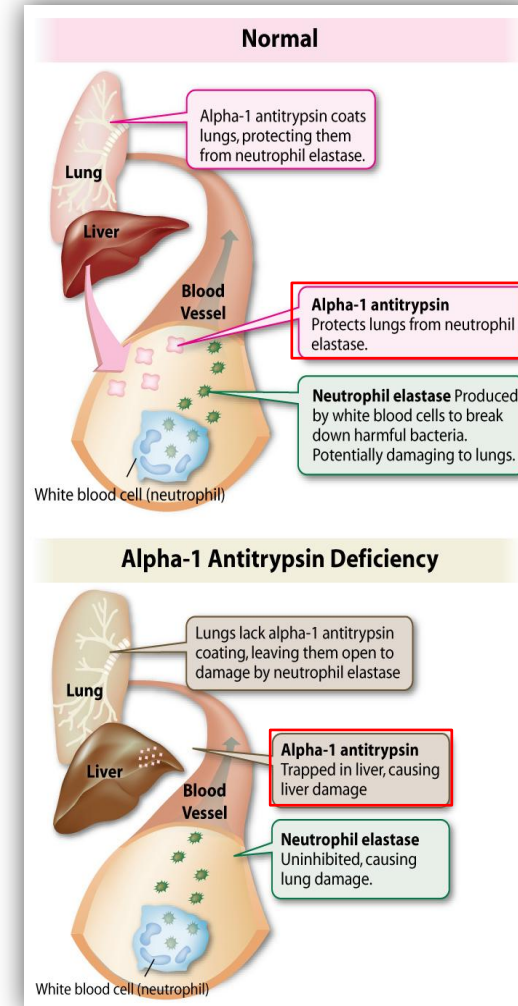
1) by secreting:  
-Immunoglobulin A (IgA)  
-Alpha-1 antitrypsin

2) By The last line defense (**pulmonary macrophages**) in the alveoli: they engulf smaller particles which pass through the muco-cilliary barrier filter.

•We know that the air which enter our body it's not only contain oxygen ,there are other substances which might be dangerous. So that way we need pulmonary defense.

مصدر التريبسين : عند دخول البكتيريا مع الهواء لمجرى التنفس تبدأ تفرز هذا الإنزيم وهو التريبسين و وظيفة هذا الإنزيم تحطيم البروتين و جدار المجرى التنفسي من المعروف أنه مكون من بروتين بالتالي يتحطم وخصوصاً ال alveoli لأنها حساسة جداً.

\* **Trypsin** is an enzyme that breaks protein so it lysis the respiratory wall, and that's why we have **antitrypsin** to prevent wall lysis. Generally known as "serum inhibitor".



EXTRA

# Functions of the respiratory system

## 4- Metabolism

**Angiotensin** is a peptide hormone that regulates blood pressure. When angiotensin goes from blood to the respiratory system, it becomes activated: The lungs help in the formation of an enzyme: **angiotensin converting enzyme** Which converts **Angiotensin I** to **Angiotensin II**.

## 5- Regulation

Regulating the acid-base balance (pH) of blood by washing out extra  $CO_2$ .  
**Blood PH:** it's the concentration of "Hydrogen ions" in the blood.  
 More  $H^+$  in blood > **Acidosis**.  
 Less  $H^+$  in blood > **Alkalosis**.

## 6- Secretion

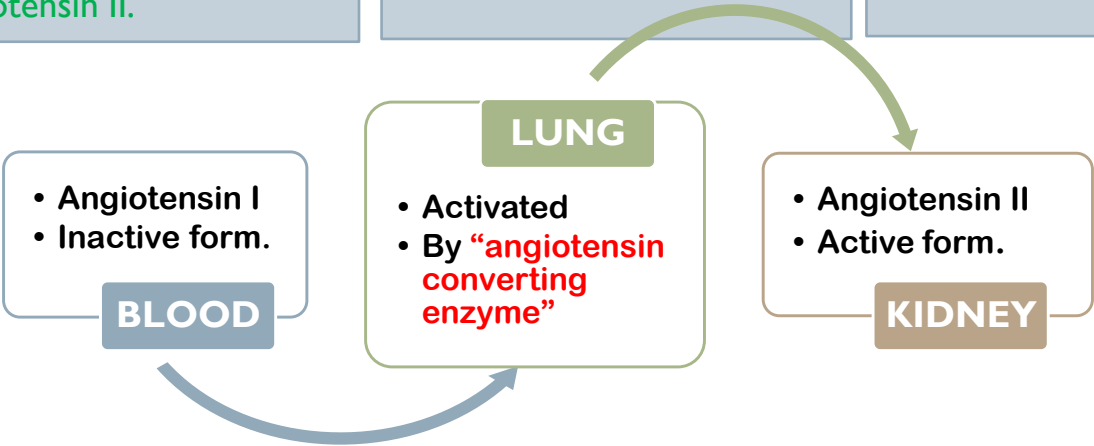
Secretion of important substances like **surfactant** "**in the alveoli**"

## More explanation

إذا زادت الحامضية في الدم معناته عندنا أيونات هيدروجين كثيرة من وين جت؟ عن طريق ثاني أكسيد الكربون بطريقة غير مباشرة.  
 ثاني أكسيد الكربون الموجود في جسمنا يتحد مع الماء الموجود في الدم وينتج مركب اسمه "كربونيك أسيد" يعني حمض!  
 وهذا الحمض يتحلل وينتج مركبين هم "داي أوكسايد + أيون الهيدروجين" وهذا معناته أن أحد مصادر أيون الهيدروجين هو ثاني أكسيد الكربون.  
 فلو تخصلنا من ثاني أكسيد الكربون بالتالي ينتخلص من أيون الهيدروجين ورح تقل الحامضية الزائدة. يعني نحتاج نزيد معدل التنفس عشان يطلع ثاني أكسيد الكربون أكثر.  
 لكن، إذا زارت القاعدية في الدم معناته عندنا أيونات هيدروجين قليلة! بهذه الحالة لازم أزيد من ثاني أكسيد الكربون وأقل من خروجه بحيث إني أبطئ من معدل التنفس وأحاول أوازن ثاني أكسيد الكربون في الجسم.

**Metabolic alkalosis :** in metabolic alkalosis (a condition resulting from excess bicarbonate retention) the rate and depth of ventilation decrease so that  $CO_2$  can be retained ,this increase carbonic acid level.

**Metabolic acidosis :** in metabolic acidosis (a condition resulting from excess acid retention or excess bicarbonate loss) the lung increase the rate and depth of ventilation to eliminate excess  $CO_2$  ,this decrease carbonic acid levels.



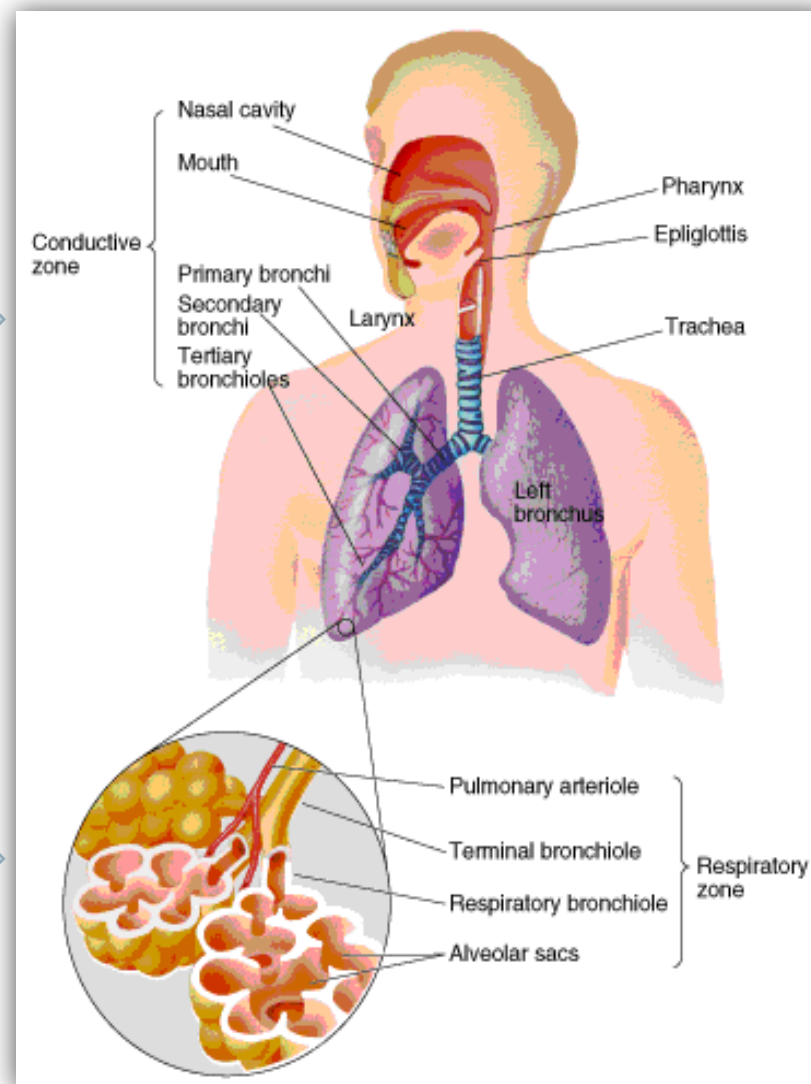
# Respiratory passages (airways)

## Conductive Zone

- **Main function is : conducts air from atmosphere to the next zone “respiratory zone”**
- 1- Starts from nose to the end of terminal bronchioles.
- 2- Help in **warming, humidification and filtration** of inspired air.
- 3- Contains the olfactory receptors for smell sensation.
- 4- Conducts the sound during speech.
- 5- Helps in coughing and sneezing reflexes for protection.

## Respiratory Zone. Also called (Respiratory unit)

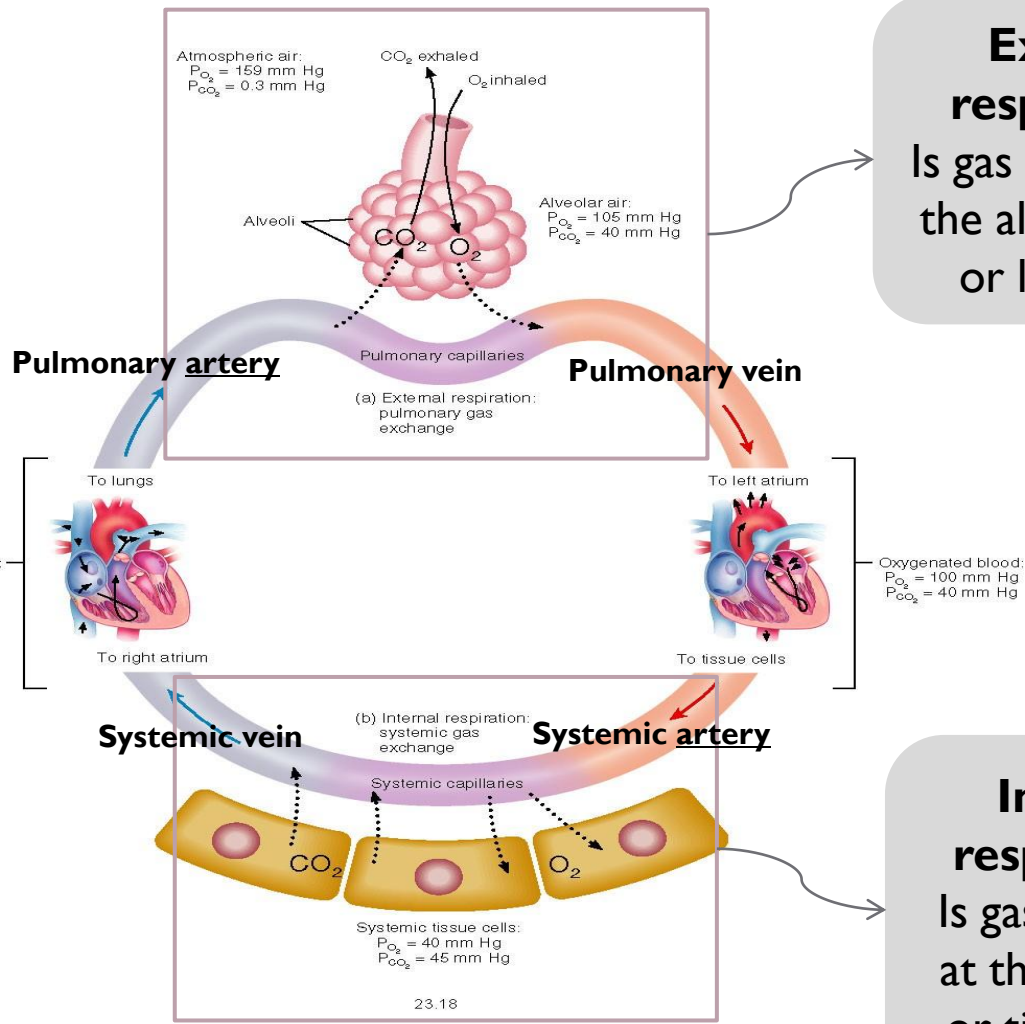
- **Main function is : Gas exchange.**
- Includes: respiratory bronchioles, alveolar ducts, alveolar sacs, alveoli (acix)





# Internal & External Respiration

## “EXTRA NOTES”



**External respiration :**  
 Is gas exchange at the alveolar level or lung level.

**Internal respiration :**  
 Is gas exchange at the cell level or tissue level.

The vessels which are going to and coming from the pulmonary circulation are called: **Pulmonary arteries + Pulmonary veins.**

- Any blood that travels from tissues to the heart, goes through **veins**
- Any blood that comes from the heart, comes through **arteries**

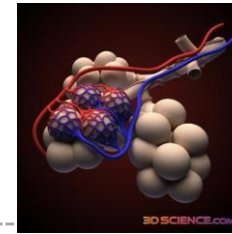
**-Always: red vessels carry oxygenated blood.**  
**-Always: blue vessels carry deoxygenated blood.**

To memorize :  
 External > Near to the outside.  
 Internal > Near to the inside (Deep)

systemic arteries “Oxygenated blood”	pulmonary arteries “deoxygenated blood”
systemic veins “deoxygenated blood”	pulmonary veins “Oxygenated blood”



# Internal & External Respiration "Extra Explanation"

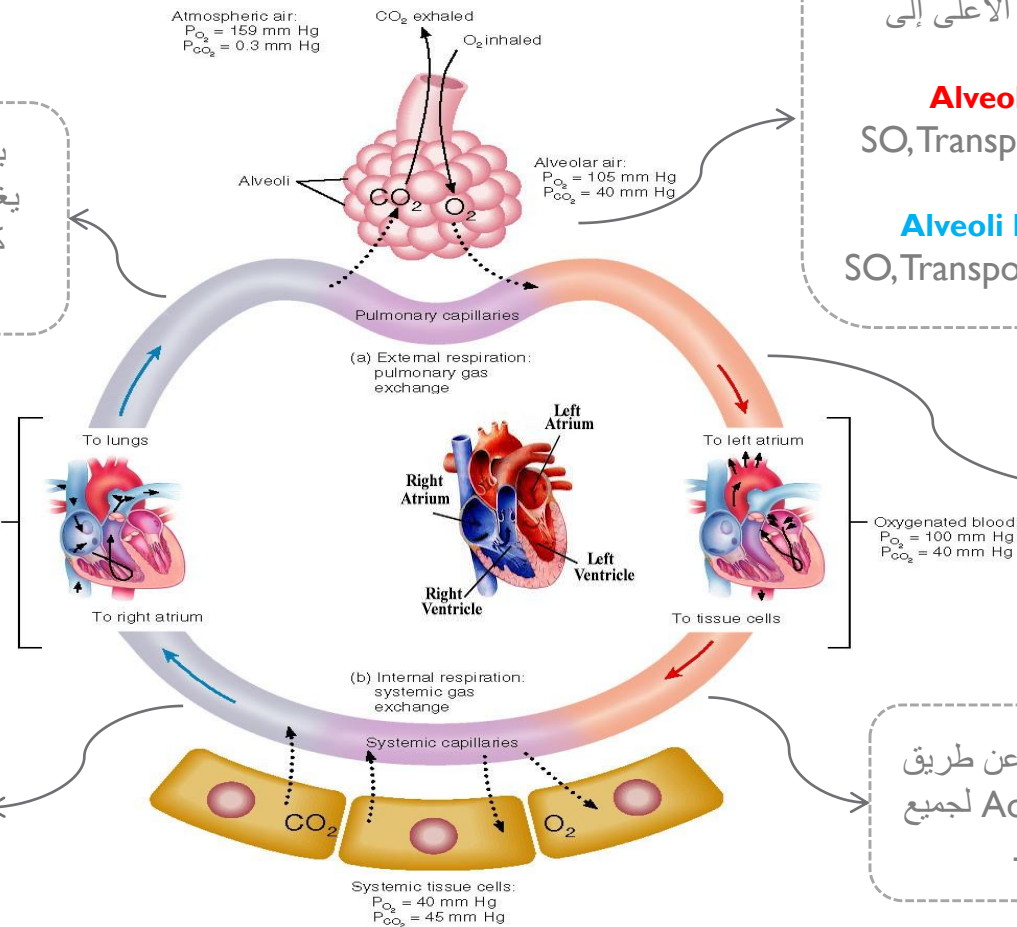


عندما يصل الدم إلى alveoli تكون مغلقة بالـ pulmonary capillaries.. ويتم انتقال الأوكسجين وثنائي أكسيد الكربون من المستوى الأعلى إلى المستوى الأقل.

**Alveoli More O<sub>2</sub> (Inhaled) = Capillaries Less O<sub>2</sub>**  
SO, Transport O<sub>2</sub> from Alveoli to pulmonary Capillaries.

**Alveoli Less CO<sub>2</sub> (Exhaled) = Capillaries More CO<sub>2</sub>.**  
SO, Transport CO<sub>2</sub> from pulmonary capillaries to Alveoli.

بعد ذلك الدم الغير مؤكسج يغادر القلب عن طريق وريد كبير اسمه pulmonary artery.



ثم، تتجمع الـ capillaries لتصبح veins وهم أربعة أو عية دموية اثنين لكل رئة من الرئتين.  
تنقل الدم المؤكسج وتدخل القلب في الـ left atrium then left ventricle.

الدم الغير مؤكسج القادم من خلايا الجسم هو دم مستهلك عن طريق الأنسجة أنتجت ثاني أكسيد الكربون مما جعله يأخذ اللون الأزرق. هذا الدم يدخل الـ Right ventricle في القلب بعد ذلك إلى الـ Right atrium.

بعد ذلك يغادر القلب عن طريق وريد كبير اسمه Aorta لجميع خلايا الجسم.

# External Respiration

-The respiration is considered to be “resting” during normal breathing.

- The respiration is considered to be “forced” or “maximal” during heavy conditions like during exercises, or like with patients with asthma, allergy...etc.

It occurs with 3 major functional events:

Pulmonary ventilation

Inward and outward movement of air between lung and atmosphere.

Diffusion

When gases exchange during diffusion of  $O_2$  and  $CO_2$  between the alveoli and the pulmonary capillary blood.

Transport

Transport of  $O_2$  and  $CO_2$  in blood and body fluids to and from the cells.

**External respiration** : is basically the transfer of gas between respiratory organs such as lungs and the outer environment.

**Regulation** : To regulate the air concentration ,and it depends on the number of surfactant available.(Some books consider it number “4” of the functional events)

# Lining cells of the alveoli

Type I alveolar cells  
(Type I pneumocytes)

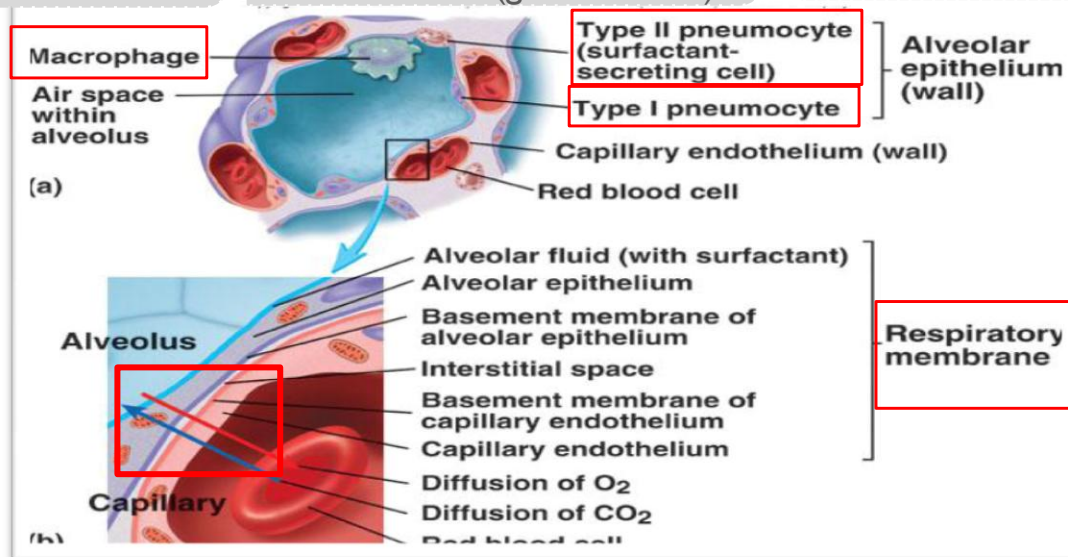
Function: Share in the formation of the respiratory membrane.

Type II alveolar cells  
(type II pneumocytes)

Function: Secrete substance called "surfactant" (granular cells)

Alveolar macrophages

Function: engulf smaller particles.



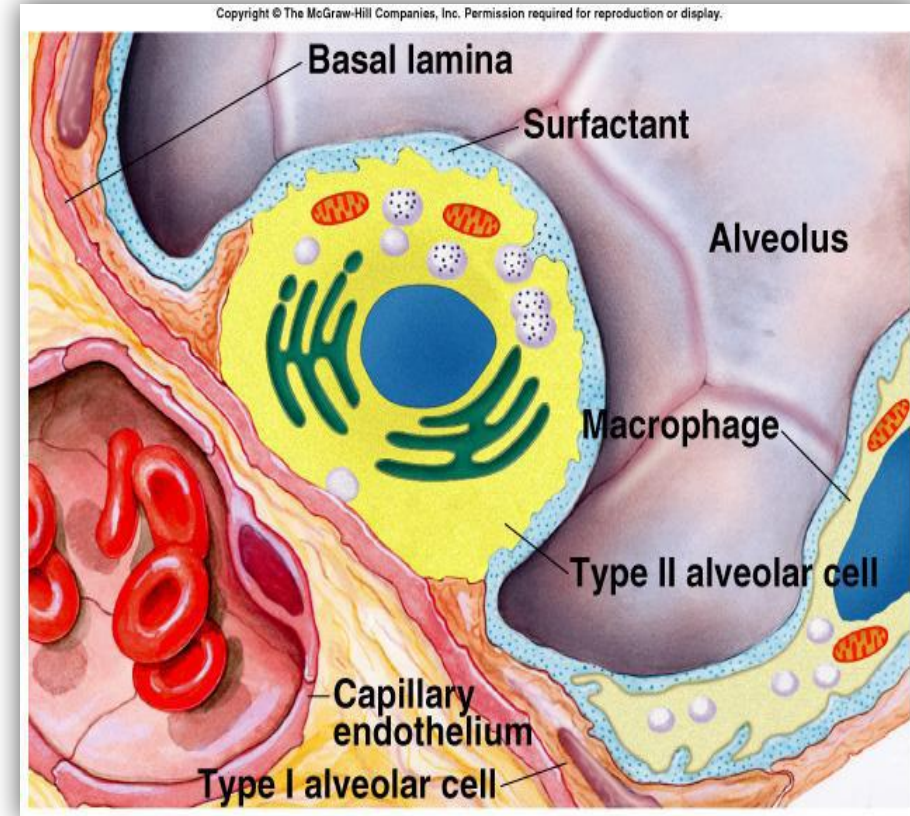
- If we could choose one main element for external respiratory it's going to be the alveoli.
- Type I alveolar epithelial cells + Endothelial cells + Interstitial tissue > those are the pulmonary membrane or Respiratory membrane.

Type I alveolar cells تركيب الخلية (الرفيع والرقيق) على الجدار يجعلها مؤهلة لعملية ال Diffusion لغازي الأوكسجين وثاني أوكسيد الكربون.

# Surface Tension

**Surfactant** : is a substance covering and lining the inner surface of the alveoli.

- $H_2O$  molecules at the surface are attracted to other  $H_2O$  molecules by attractive forces that resist distension called surface tension.
- Surface tension tends to oppose alveoli expansion.
- Pulmonary surfactant **reduces surface tension**.
- Surfactant is a complex substance containing **phospholipids** and a **number of apoproteins**.
- Secreted by **the Type II alveolar cells**.
- The earliest detection from fetal alveoli begins between 6-7<sup>th</sup> month but this could be delayed in others to **week 35** of intrauterine life.



- ماذا يحدث عندما يقترب جزيء هواء من جدار الalveoli الذي يحتوي على الماء ؟ تقوم جزيئات الماء بالتجاذب بينها وبين بعضها "خاصية التوتر السطحي" وتنكمش الخلية وهذا خطير جدًا على حياتنا فلذلك هنالك مواد تقوم بإيقاف هذه العملية وهي السورفاستانت فهي تقوم بعزل الماء عن الهواء لتوفر مساحة سطح اكبر، إذا العوامل التي تزيد ال Surface tension هو التعرض للهواء. الهدف أثناء التنفس هو زيادة مساحة السطح بالتالي لا بد من منع انكماشه بسبب تفاعل الماء مع الهواء.

- الطفل عند بلوغه ٧ اشهر في بطن امه فيكون بذلك تخطى مرحلة الخطر.

- أخطر مرحلة يخاف منها على المولود هو اول نفس اذا عدى بسلام فالطفل سليم ولا عليه خوف.

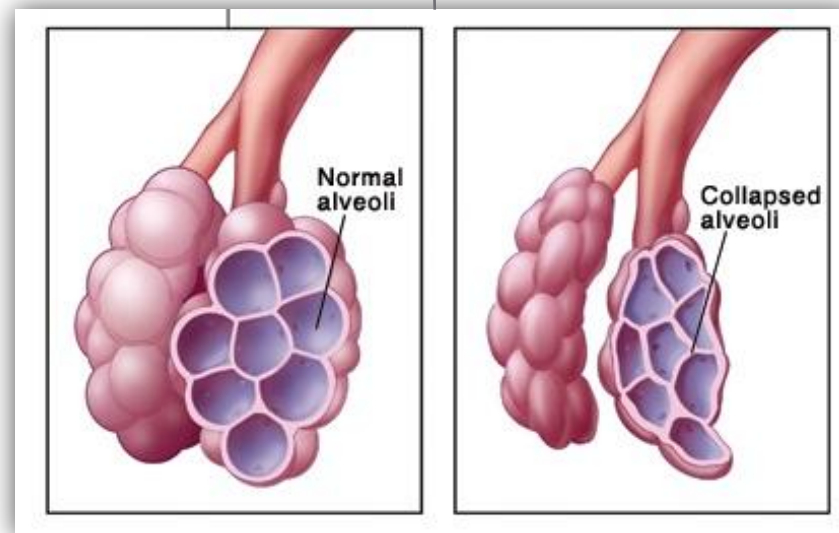


# Surfactant functions

1-reduces surface tension throughout the lung.

2-prevents alveolar collapse.

3-decreases airway resistance and the work of breathing.



كل ممرات التنفس في الجسم عندها مقاومة بسيطة لمرور الهواء عبرها، لأن الهواء يعتبر مركب غريب بالنسبة لها، والمقاومة هذه تتم عن طريق ال Smooth muscles. وهذا هو الطبيعي لكن، لو زادت انقباضات العضلات بالتالي increase airways resistance. بالتالي تعمل مادة السورفاكتنت على تقليله.

# Pulmonary diseases

- **Deficiency in premature babies** cause “**respiratory distress syndrome**” of the new born (RDS) (hyaline membrane disease).
- Their lungs aren't able to make enough surfactant.

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 Alveoli.

Boy's Notes:

**VEDIO**

- **Smoking in adults**, hypoxia or hypoxemia (low oxygen in the arterial blood) or both, decrease the secretion of surfactant and cause adult “**respiratory distress syndrome**”.
- Hypoxia > Low O<sub>2</sub> in air.
- Hypoxemia > Low O<sub>2</sub> in blood.



Healthy alveolus



Alveolus damaged by pulmonary disease



# Innervations of lungs and bronchi

Is by autonomic nerves.

Sympathetic stimulation causes dilatation of the bronchi.

Parasympathetic stimulation causes constriction of the bronchi.




**Locally secreted factors:** Histamine, Slow reacting substances of anaphylaxis (SRSA) by mast cells, due to allergy (as in patients with asthma) often cause bronchiolar constriction and increase airway resistance.

Slow reacting substances Like :  
**Bradykinin , Prostaglandin ,  
Serotonin.**

عند دخول مواد غريبة مع الهواء كرائحة مادة  
عند الشخص حساسية منها ! هذه المواد تنهيج  
وتؤدي إلى حدوث Bronchospasm وبالتالي  
سيحصل increase airways resistance

2

## Mechanics of pulmonary ventilation

-  Very important
-  Extra information
-  Terms



The most effective way to do it , is to  
**DO IT!**

# Objectives

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

# Muscles That Expand and Contract The Lungs "BOYS & EXTRA NOTES"

## LUNGS CAN BE EXPANDED AND CONTRACTED:

Expansion	Contraction
<p><b>Downward movement</b> of the diaphragm to increase the chest cavity.</p>	<p><b>Upward movement</b> of the diaphragm to decrease the chest cavity.</p>
<p>By the <b>elevation</b> of the ribs to increase the antero-posterior diameter of the chest cavity.</p>	<p>By the <b>depression</b> of the ribs to decrease the antero-posterior diameter of the chest cavity.</p>

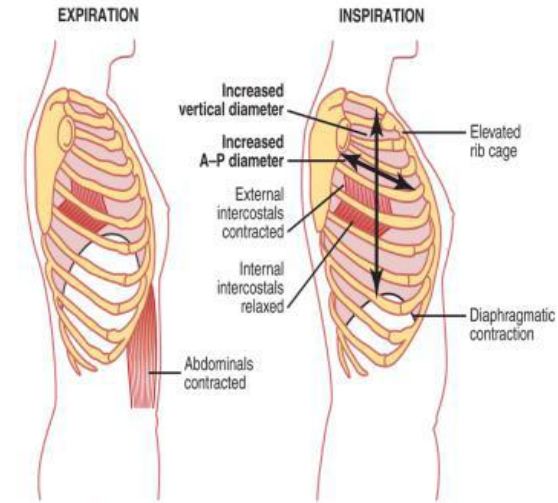
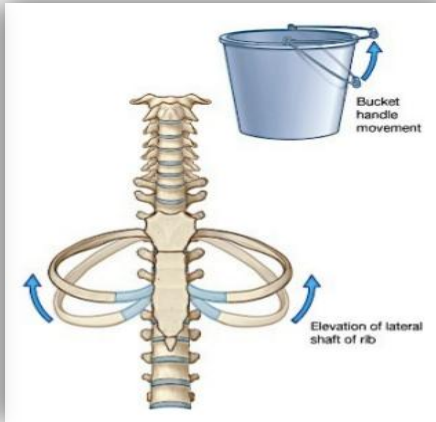
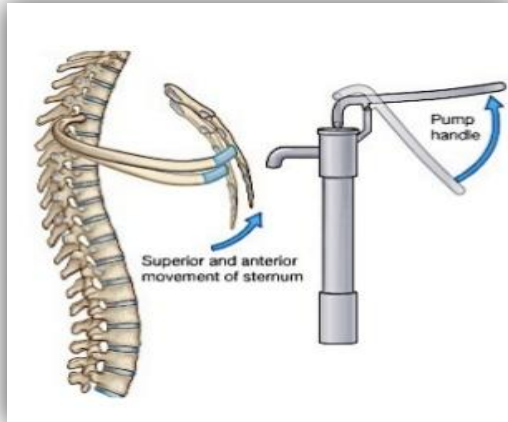


Figure 37-1 Contraction and expansion of the thoracic cage during expiration and inspiration...

Anterior increase in volume.

Lateral increase in volume.



- Allows the air to move > By the pressures.
  - Changes the pressures > By the muscles.
- The three dimensions of the chest cage are :  
(Vertical ,Transverse, Depth)

# Respiratory Muscles

## INSPIRATORY MUSCLES:

All the muscles that expand (elevate) the chest cage are classified as muscles of inspiration (**FORCED “DEEP” AND “RELAXED”**)

**RELAXED INSPIRATION:** During resting breath. Contraction of the :

- **Diaphragm muscle.** - Goes down to expand the chest cage. Increase the vertical dimension.
- **External intercostals** – Moves the ribs outward. Increase the transverse dimension.

**DEEP INSPIRATION** During exercise : (relaxed inspiration muscles + Accessory muscles) During deep forceful inhalation accessory muscles of inspiration participate to increase size of thoracic cavity: “Are Bellow”

## EXPIRATORY MUSCLES:

All the muscles that depress the chest cage are classified as muscles of expiration (**FORCED ONLY**)\*

### DEEP EXPIRATION:

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

- **Abdominal muscles**
- **Internal intercostals** – Moves the ribs inward. Decrease the transverse dimension.

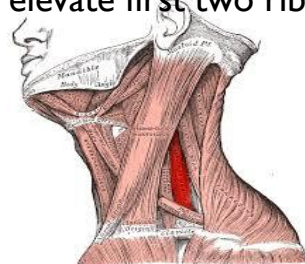
### A. Sternocleidomastoid

– elevate sternum



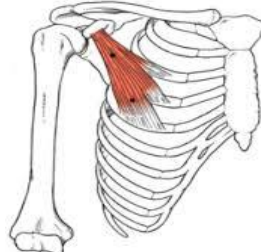
### B. Scalenes

– elevate first two ribs



### C. Pectoralis minor \*

– elevate 3rd–5th ribs



\***Pectoralis major** (mentioned in anatomy), increases the AP diameter when the arm is fixed.  
\***Resting expiration** is the passive relaxation of diaphragm, no muscle contraction necessary.



# Respiratory Muscles

## Muscles of inspiration

**Accessory**

- Sternocleidomastoid (elevates sternum)
- Scalenes Group (elevate upper ribs)
- Not shown: Pectoralis minor

**Principal**

- External intercostals
- Interchondral part of internal intercostals (also elevates ribs)
- Diaphragm (dome descends, thus increasing vertical dimension of thoracic cavity; also elevates lower ribs)



## Muscles of expiration

**Quiet breathing**

Expiration results from passive, elastic recoil of the lungs, rib cage and diaphragm

**Active breathing**

- Internal intercostals, except interchondral part (pull ribs down)
- Abdominals (pull ribs down, compress abdominal contents thus pushing diaphragm up)
- Note shown: Quadratus lumborum (pulls ribs down)

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**End of expiration**

Diaphragm relaxed

**End of inspiration**

Labored breathing: Additional muscles contract, causing additional expansion of the thorax.

Abdominal muscles relax.

**Quiet breathing:** The external intercostal muscles contract, elevating the ribs and moving the sternum.

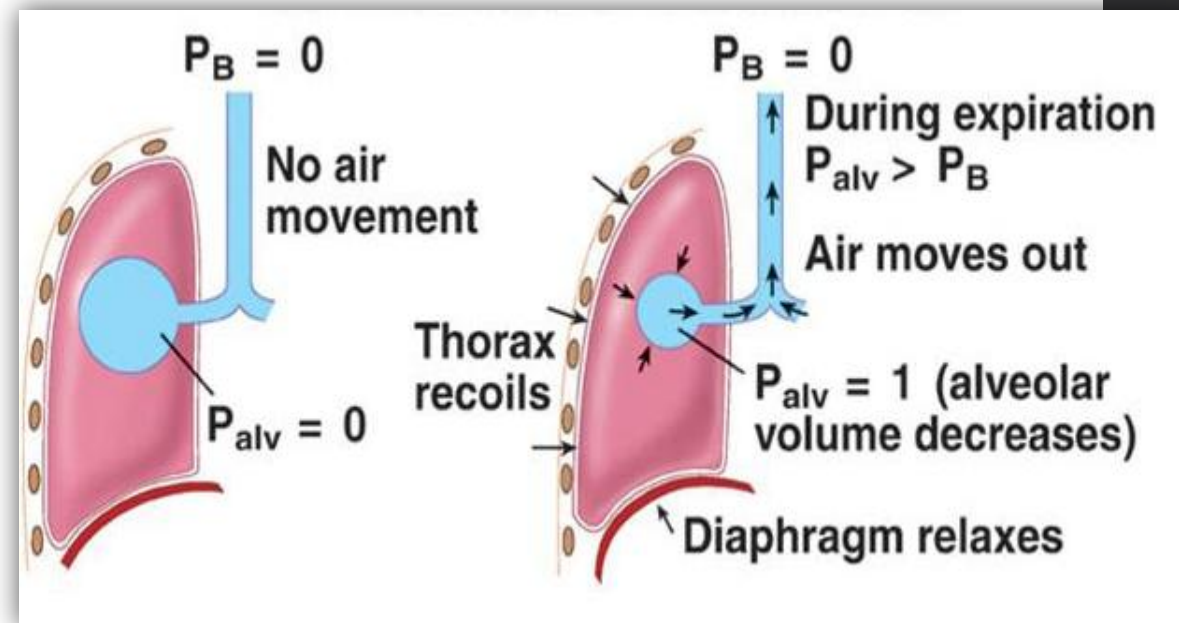
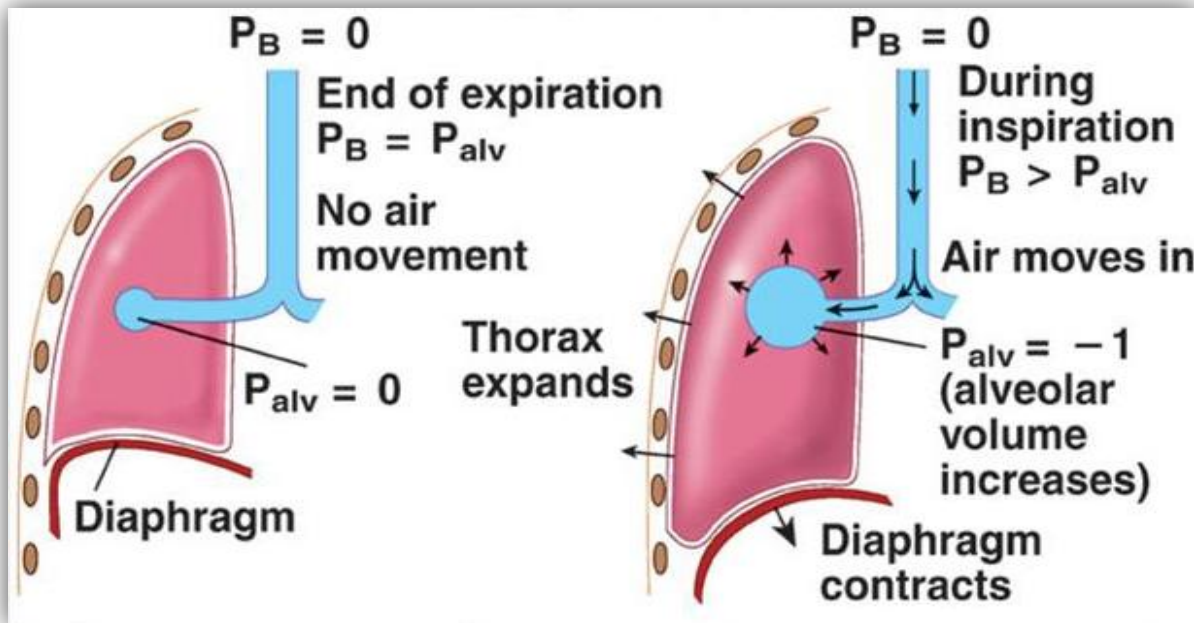
**The diaphragm contracts,** increasing the superior-inferior dimension of the thoracic cavity.



# Pressures changes in the lungs during breathing

◆ **CONCEPT:** Air will flow from a region of high pressure to one of low pressure-

The bigger the difference, the faster the flow. Atmosphere pressure =  $P_B$  (Barometric pressure) = 760 = 0



**End of expiration:**  
 $P_B = P_{alv} = 0 = 760$   
 "No more movement of the air outside"

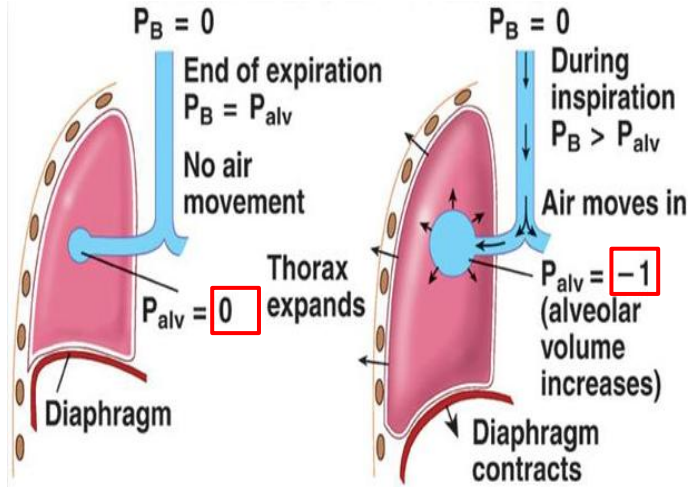
**During inspiration:**  
 $P_B = 0 > P_{alv} = -1$   
 $760 > 759$   
 "the movement of the air from high to low .. From outside to inside"  
 The thorax expands.

**End of Inspiration:**  
 $P_B = P_{alv} = 0 = 760$   
 "No more movement of the air inside"

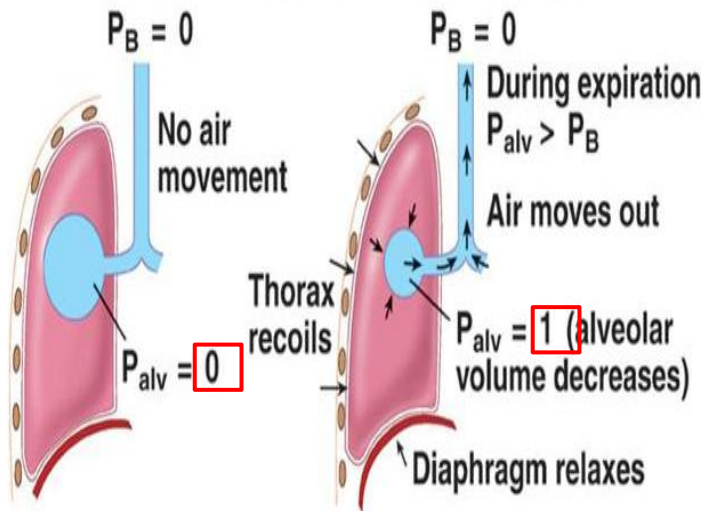
**During expiration:**  
 $P_{alv} = 1 > P_B = 0$   
 $761 > 760$   
 "the movement of the air from high to low .. From inside to outside"  
 The thorax recoils.



# Extra explanation



- Between breaths (Rest) = 0 = 760  
No air movement ,  $P_B = P_{alv}$
- During inspiration = -1 mmHg ( $P_{alv} < P_B$ )  
air flow from outside to inside the lungs. During inspiration we know that diaphragm will contract which allows the volume to **increase** while the pressure **decreases**. " $P_{alv} = -1 = 795$ "  
العلاقة بين الضغط والحجم علاقة عكسية ففي هذه الحالة ازداد لدينا الحجم مما يعني أن الضغط سيقبل ولذلك كانت الإشارة سالبة.



- At the end of inspiration = 0 (air flow stops, no more air movement , no more increase in the chest wall volume.
- During Expiration = +1 mmHg ( $P_{alv} > P_B$ )  
air flow out of the lungs. During expiration diaphragm relaxes which mean : **decrease** in chest volume "chest will recoil", **increase** in pressure  
ومن هنا نستنتج أن الإشارة الموجبة دلالة على زيادة الضغط أما الإشارة السالبة كما في الحالة السابقة فهي دلالة على نقصه.

# Pressures changes in the lungs during breathing

## THREE TYPES OF PULMONARY PRESSURE MENTIONED:

### INTRA-ALVEOLAR "INTRAPULMONARY" PRESSURE ( $P_{alv}$ )

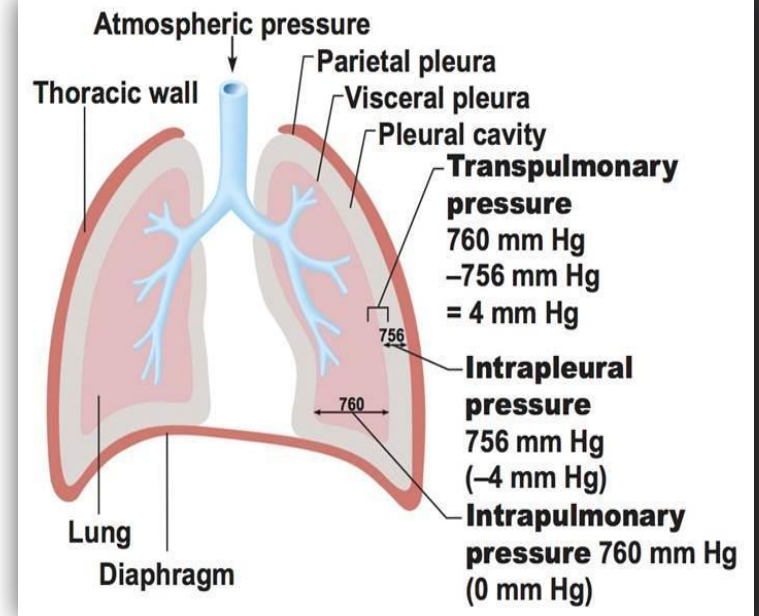
Alveolar pressure is the pressure of the air inside the lung alveoli.

### INTRAPLEURAL PRESSURE ( $P_{ip}$ )

- Pleural pressure is the pressure of the fluid in the thin space "imaginary space" between the lung pleura and the chest wall pleura.
- Pressure in the pleural space is **negative** with respect to atmospheric pressure at the end of normal expiration (**-5cmH<sub>2</sub>O**).

### TRANSPULMONARY PRESSURE ( $T_{Pp}$ )

- Called : (the recoil pressure) & (The extending pressure)
  - The difference between the **alveolar pressure ( $P_{alv}$ )** and the **pleural pressure ( $P_{pl}$ )**. ( هو ضغط حسابي وليس له مكان معين في التجويف الصدري )
- $T_{Pp} = P_{alv} - P_{pl}$  .. Ex : 1 - (-7) = +8mmHg**



#### Why it's called ?

Extending pressure : because it's the pressure that keeps the lungs inflated or extended , so the lungs never collapse.

Recoil pressure: because it's equal to the recoil force of the lungs. So when the pressure = to the force ,the recoiling will not happen, so no lung collapse.

# Pressures changes in the lungs during breathing

Continue..

## INTRA-ALVEOLAR "INTRAPULMONARY" PRESSURE ( $P_{alv}$ )

- Between breathes (Resting breath)= **Zero**
- Pressure During inspiration = **(-1 mmHg)**.. Air(tidal volume) flow from outside to inside the lungs).
- At the end of inspiration = **Zero**.. Airflow stops.
- Pressure During expiration = **(+1 mmHg)**.. Air flow out of the Lungs

## INTRAPLEURAL PRESSURE ( $P_{ip}$ )

### WHY NEGATIVE:

- The lung's elastic tissue causes it to recoil, while that of the chest wall causes it to expand. Because of these 2 opposing forces the pressure in the pleural cavity becomes negative.
- The pleural space is a potential space, empty due to continuous suction of fluids by lymphatic vessels.

### Values of IPP:

- (-5) cm H<sub>2</sub>O **during resting** position between breathes, and it becomes more -ve (-7.5) cm H<sub>2</sub>O **during resting inspiration**. (Due to the expanding of the chest wall)
- **Forced ventilation:** (Insp. :-20 to -40 cm H<sub>2</sub>O .. Exp. : + 30 cm H<sub>2</sub>O)

## TRANSPULMONARY PRESSURE ( $T_{Pp}$ )

- 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.**

### Opposing forces :

- Lung force (Recoiling)
- Chest wall force (Expanding)  
تعاكس هذين القوتين تعطي الإشارة السالبة للضغط.
- The lung is surrounded by a layer of visceral pleura, and the whole chest wall is covered by parietal pleura. Between the visceral pleura and the parietal pleura is a thin space of pleural fluid. This fluid provides suction.
- Suction (شفط) is expressed as a negative pressure, and the amount of suction increases as the thoracic diameter increases. In other words:

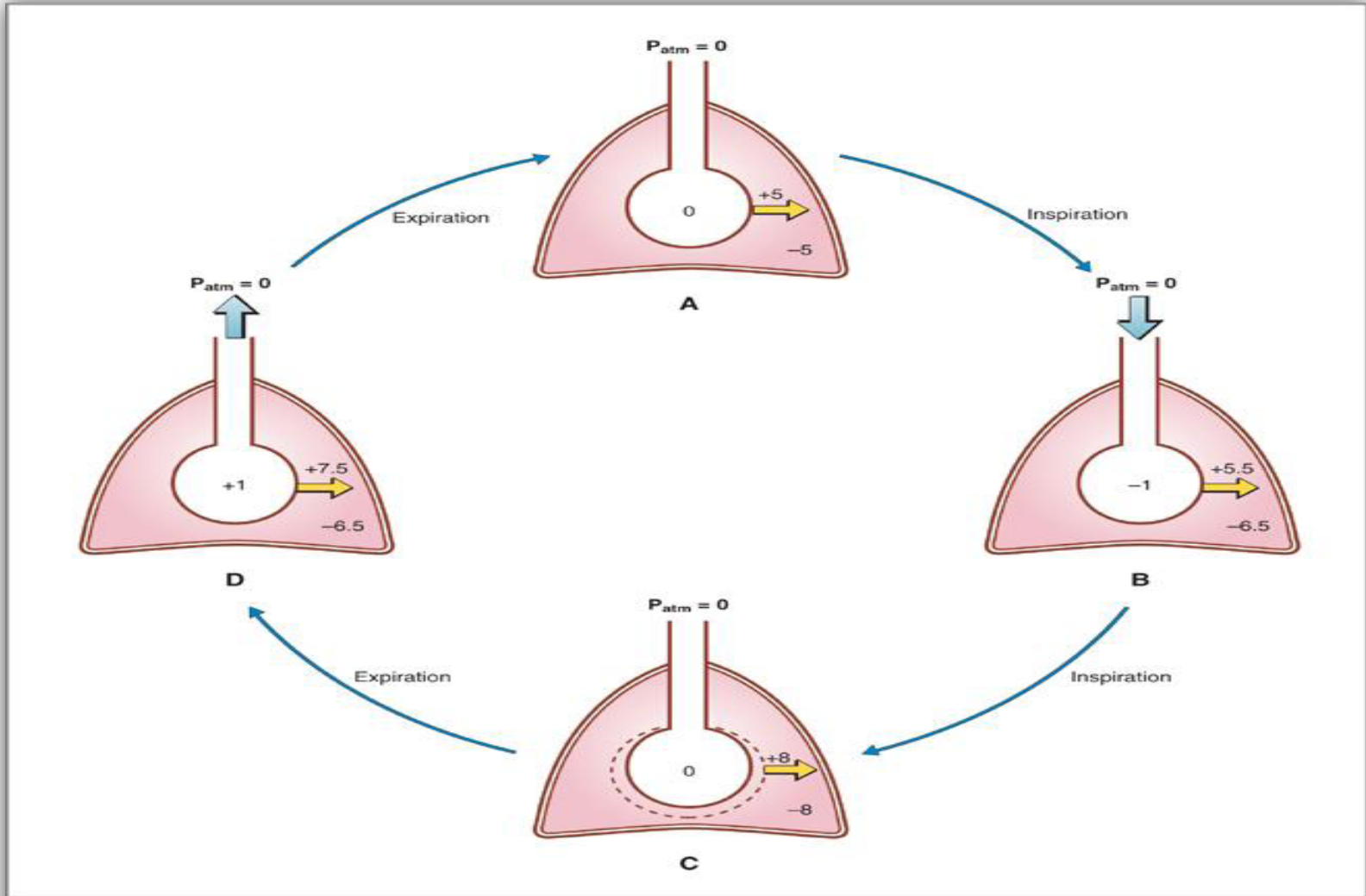
(١) يقل ضغط السائل كلما زادت مساحة القفص الصدري.

(٢) يقل ضغط السائل كلما زادت كمية الهواء.

(٣) يقل ضغط السائل كلما تمت عملية الاستنشاق.

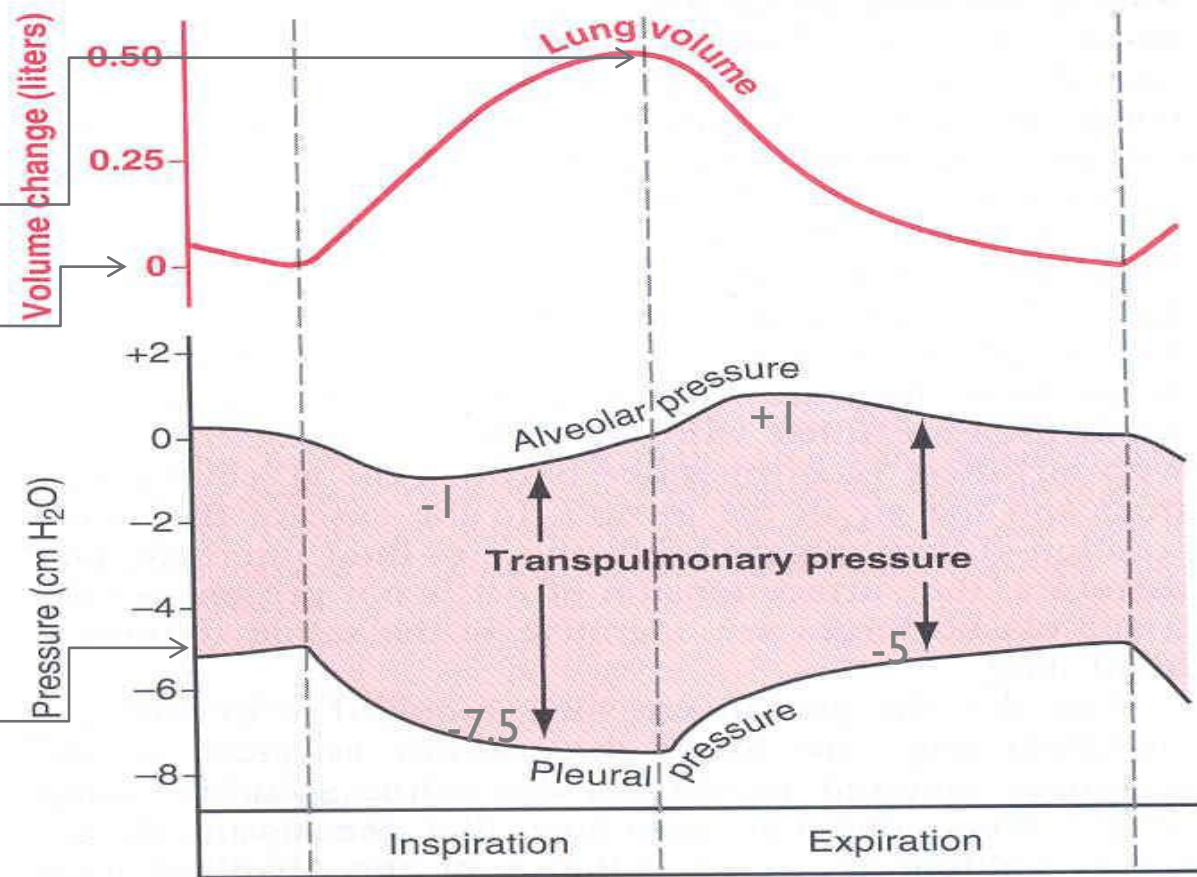


pressure and volume relationships in a  
single respiratory cycle.  
“Boy’s Slides”



# Compliance of the lung

## 'In a single respiratory cycle'



During inspiration the lung volume increase 500 ml = 0.50 (Half a liter)

Resting point (at the end of expiration)

During resting the TPP. was (-5cm H<sub>2</sub>O) (Normal)

- Is defined as, the ratio of the change in the lung volume produced per unit change in the **distending pressure(Transpulmonary Pressure)**
- The extent to which the lungs expand for each unit increase in the transpulmonary pressure.

$$CL = \frac{\text{Volume change } (\Delta V)}{\text{Transpulmonary pressure change } (\Delta P)}$$

**Compliance means :** Responsiveness & Flexibility (التجاوب والمرونة)

- Is The relationship between the change in the lung volume with the change in the transpulmonary pressure.
- Whenever this relation between the lung volume and the working pressure on it is as expected that means (The lung has normal compliance)
- Increase in transpulmonary pressure > Increase in lung volume. And vice versa.

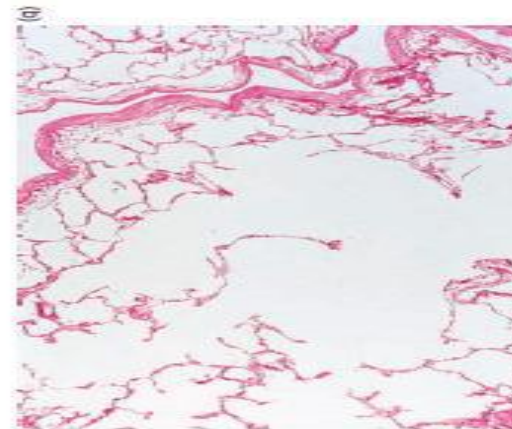
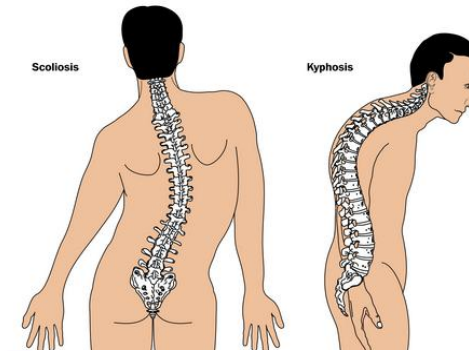


# Compliance of the lung

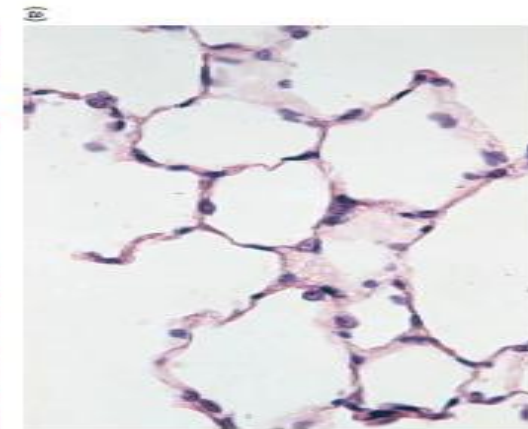
- For both lungs in adult (Free lungs)  
= **200ml of air /cm H<sub>2</sub>O.**
- For lungs and thorax together = **110 ml/cm H<sub>2</sub>O.**

## Compliance is:

- Decreased in pulmonary fibrosis , pulmonary edema, diseases of the chest wall ( kyphosis, scoliosis)
- Increased in **Emphysema** because it destroys the alveolar septal tissue rich with elastic fibers that normally opposes lung expansion.



Abnormal alveolar septal tissue. like in chronic smokers, they got repeated respiratory tract infection. Because of the bacteria the walls lysis by the **trypsin.**



Normal alveolar septal tissue. That increase surface area for gas exchange.

- The clinical benefit of the compliance: to know the structure of the lungs and chest wall.
- Whenever the lungs tissue is normal > No increase or decrease in fibers tissue, No fibrosis or chest deformity (that impedes the expansion of the lungs during inspiration) has a normal compliance.
- When compliance is decreased that means ether abnormal expiration or the chest deformity prevent the movement of the lungs.
- Generally all pulmonary diseases are decrease the compliance EXEPT Emphysema.



## Videos

- 1- [Pressure changes during breathing.](#)
- 2- [Compliance of the lung.](#)
- 3- [Respiratory system “overview”](#) very helpful.

## Quiz 1

<https://www.onlineexambuilder.com/physiology-1st/exam-56691>

## Quiz 2

<https://www.onlineexambuilder.com/physiology-2nd/exam-56701>

## SAQ's

**What are the defense factors in the pulmonary system?**

- Muco-cilliary barrier filter.
- IgA.
- Alpha-1 antitrypsin.
- Pulmonary macrophages.
- Sneeze and cough reflexes.

**How is the lung associated with the blood's PH level?**

The PH (acid-base status) is controlled by washing out CO<sub>2</sub> (Carbon-dioxide).

**Case:** A newborn was admitted to the extensive care right after delivery because of an alveolar problem.  
**How many months had the child been carried?**  
6 to 7 months.

**What is the name of the syndrome he has?**

RDS: Respiratory distress of the newborn (hyaline membrane disease)

## Physiology Team

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# THANK YOU FOR CHECKING OUR WORK

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