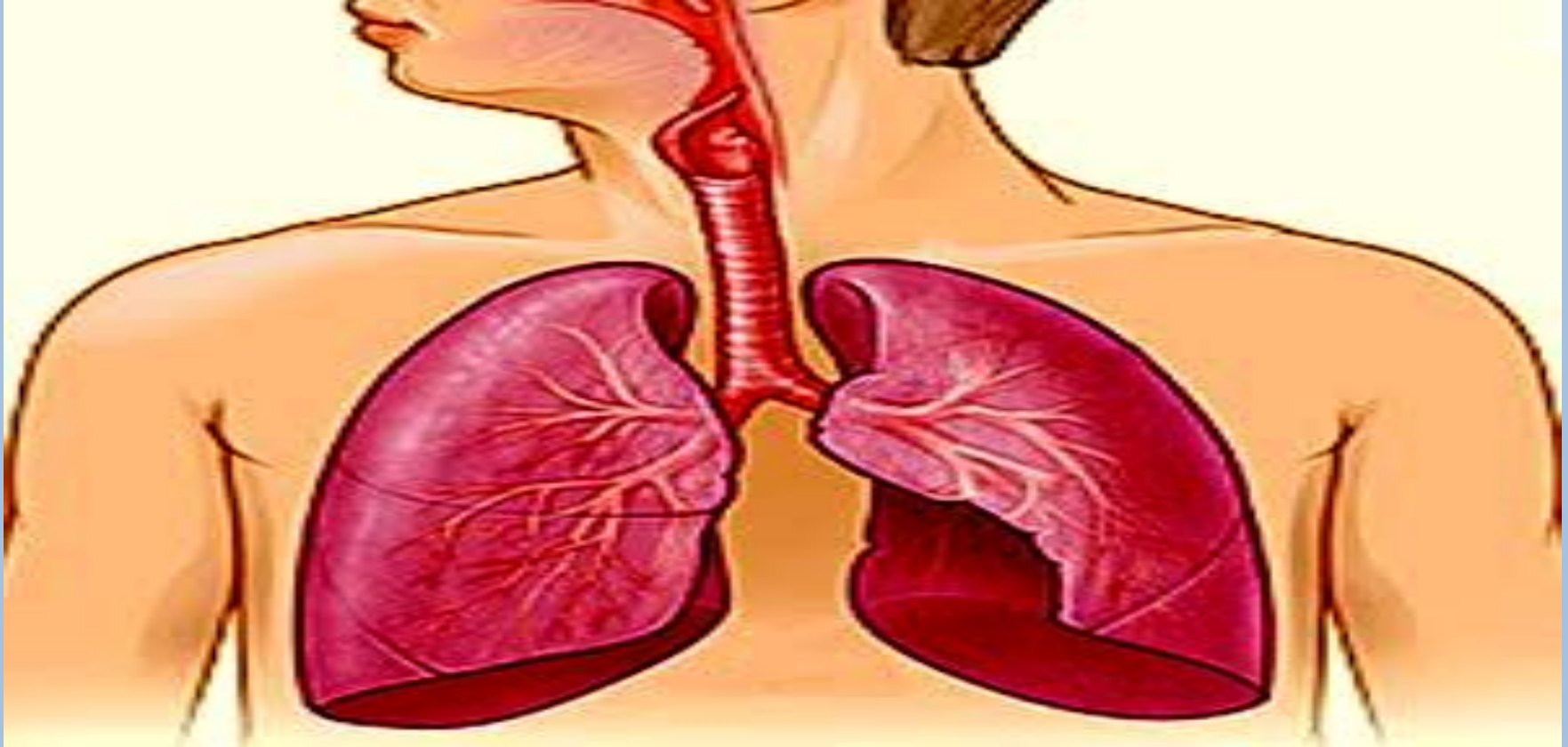


# PHYSIOLOGY OF Pulmonary Ventilation



Prof. Khaled Abdel-Sater, MD

# AIR PASSAGE

## Anatomically is divided into:

- Upper airway passages: Nose & pharynx.
- Lower airway passages: Larynx, trachea, bronchi & bronchioles.
- Physiologically is divided into:

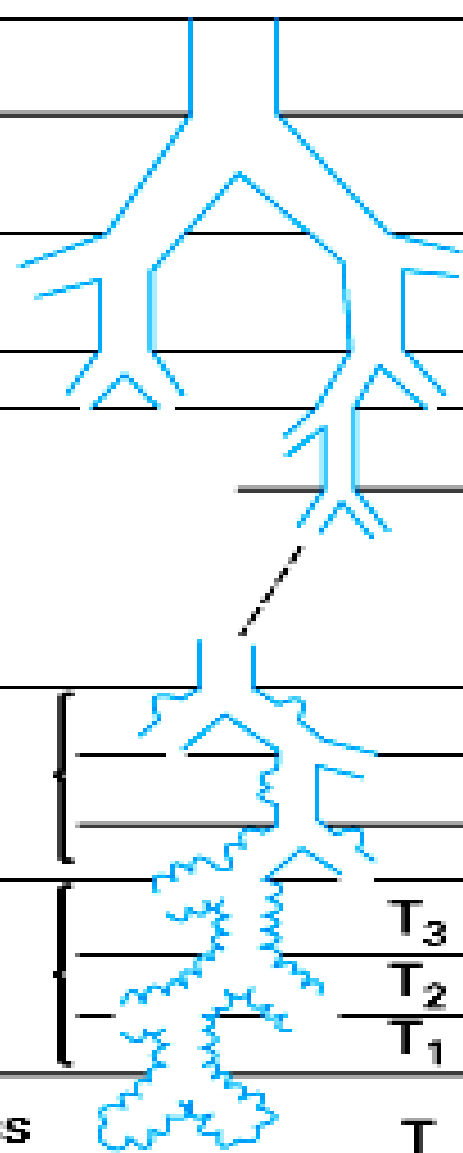
### Conducting Zone (=Dead Space):

- Nose to terminal bronchioles.
- The wall of the conducting part is thick, so no gas exchange. Therefore, it called dead space.

### Respiratory Zone:

- Site for gas exchange (thin wall).
- Respiratory bronchioles to alveoli.
- Air present in it is called alveolar air.

		Generation		Diameter cm	Number
Conducting zone	trachea		0	1.80	1
	bronchi		1	1.22	2
			2	0.83	4
			3	0.56	8
			4	0.45	16
	bronchioles		5	0.35	32
terminal bronchioles		16	0.06	$6 \times 10^4$	
Transitional and Respiratory zones	respiratory bronchioles		17	↓	↓
			18		
			19		
	alveolar ducts	$T_3$	20	↓	↓
		$T_2$	21		
		$T_1$	22		
alveolar sacs	T	23	0.04	$8 \times 10^6$	



# DEAD SPACE (D.S)

## ● Def.,:

It is the volume of air which does not undergo gas exchange with blood in the lung.

## ● Normal Value:

Normally it is equal to 150 ml.

# ● Types

**1-Anatomical Dead Space:** = 150 ml.

**2-Alveolar Dead Space:** always pathological.

**Non functional alveoli are present in:**

- a- Alveoli not received blood e.g. pulmonary thrombosis.
- b- Collapsed alveoli.

**3-Physiological Dead Space (Total Dead Space):**

- It is a total volume of air which does not undergo gas exchange.
- Physiological D.S = anatomical D.S + alveolar D.S.
- Normally there is no alveolar dead space and so normally physiological dead space = anatomical dead space, but in patient with non-functioning alveoli the physiological dead space may be equal to 10 times more than anatomical dead space.

# ● Functions

1- Air conduction.

2- Air condition.

3-Moistening of air by mucous secretion.

4-Filtration and cleaning of the air. <2, 2-10, > 10

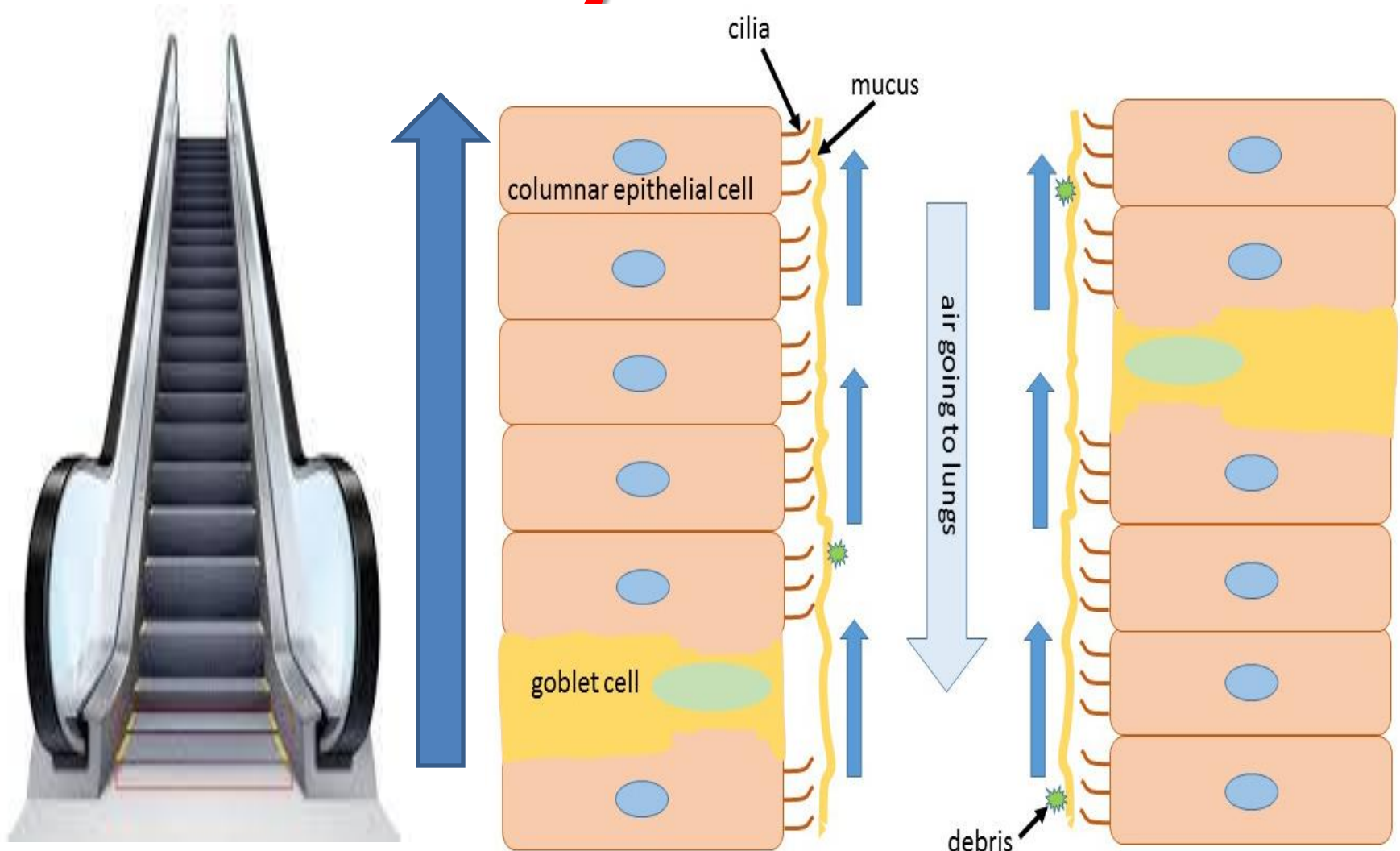
5-Protection of lung due to presence of:

a-Trachea and bronchi cilia (**=ciliary escalator**).

b-Macrophages (dust cells).

6- Regulation of body temperature (heat loss by evaporation).

# Ciliary Escalator



The foreign bodies are brushed upwards, away from the lungs, thereby ensuring that air is clean prior to entry into the lungs.

# ELASTIC RECOIL OF THE LUNGS

## ●Def.,

When the lung is inflated, it tends to recoil (collapse).

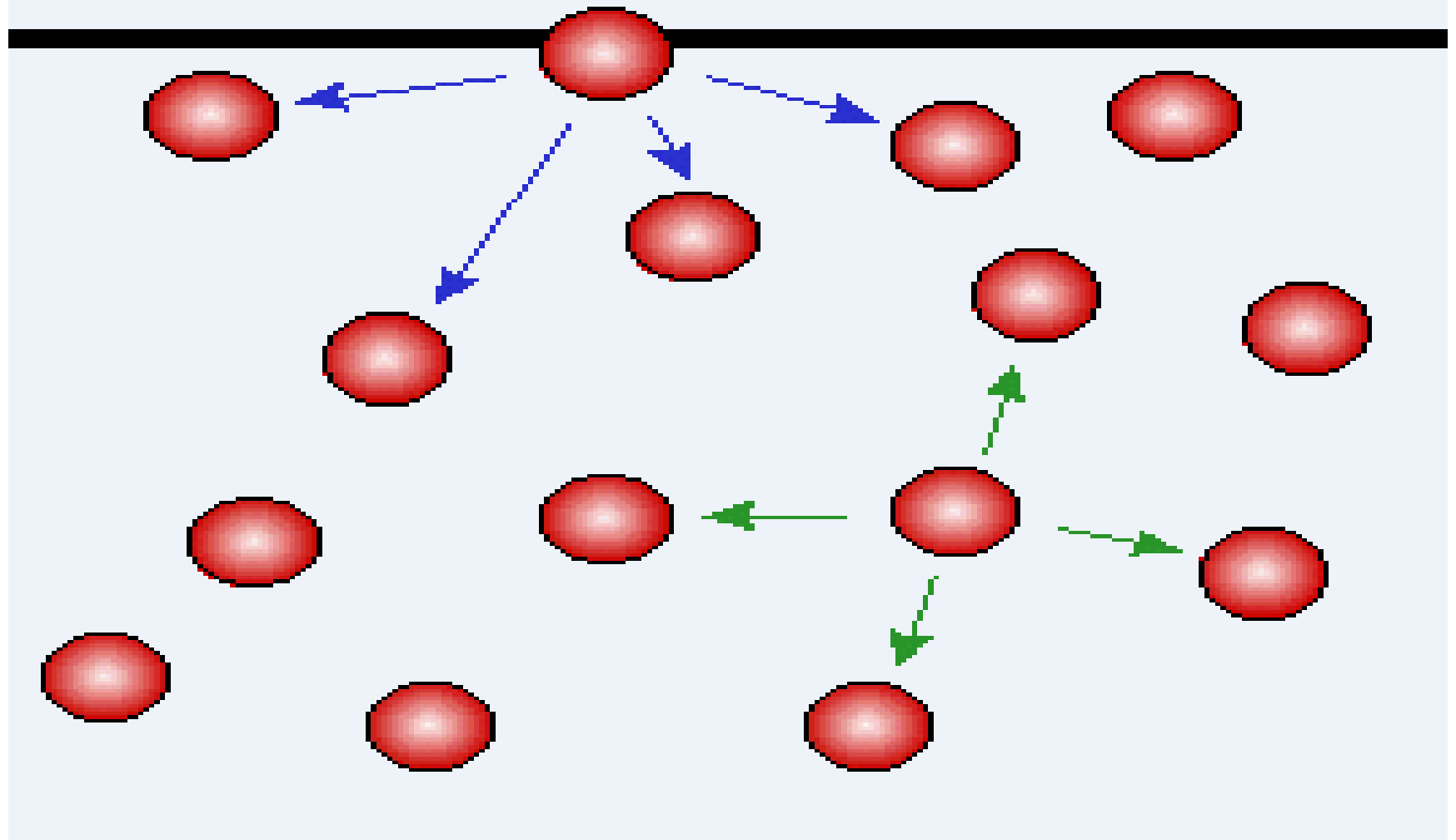
## ●Causes:

- 1-Elastic fiber in the lung (responsible only for 30%).
- 2-Surface tension of fluid lining alveoli (responsible for 70%).





# Surface



# THE PULMONARY SURFACTANT

● Def., It is a surface active agents secreted **by type II alveolar epithelial** cell to decrease the surface tension of fluid lining alveoli and antagonist lung collapse.

## ● Functions:

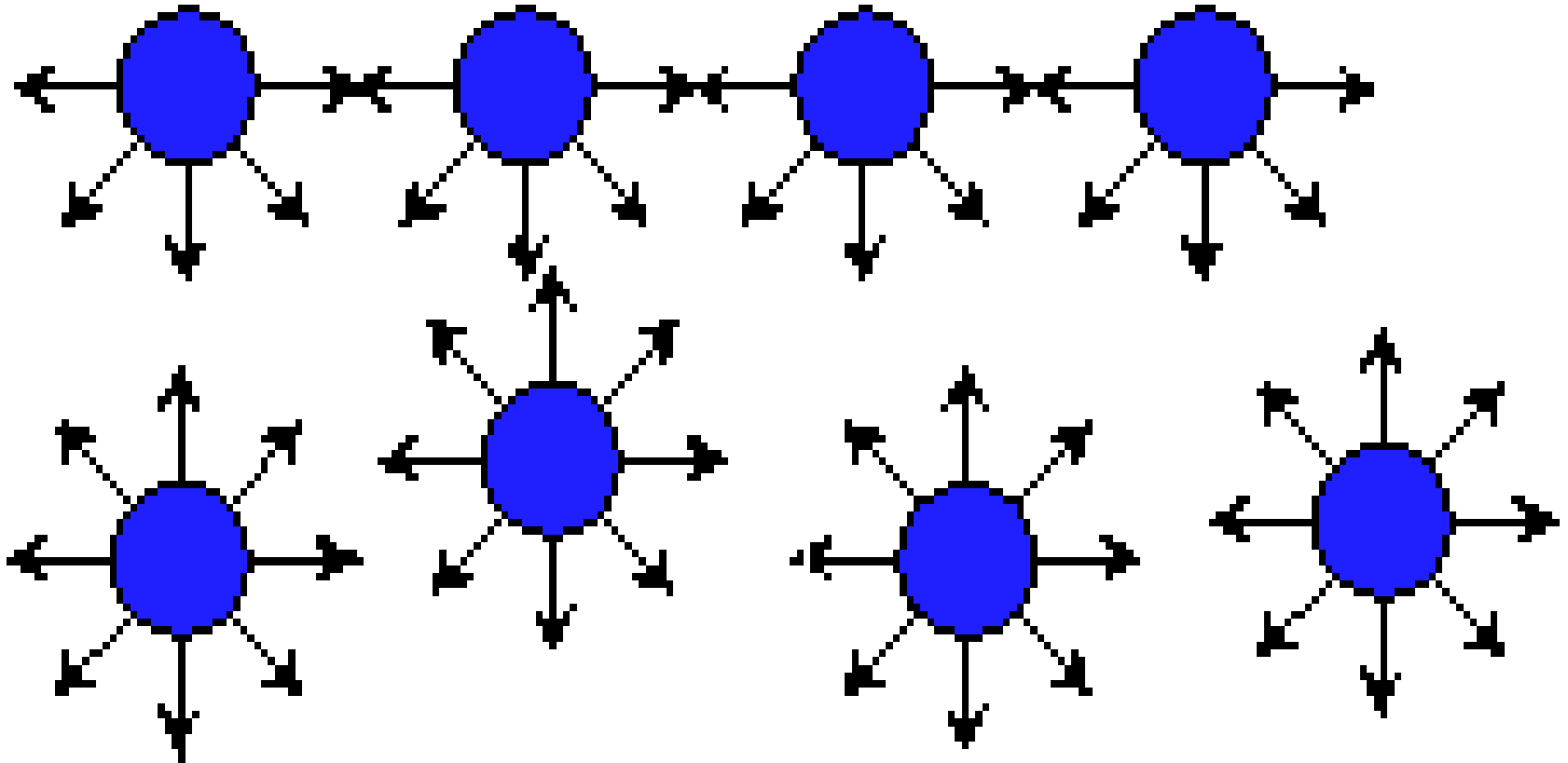
- 1- Prevents of the alveoli collapse.
- 2- Decreases the muscular work of breathing and increases the pulmonary compliance (reducing the effort needed to expand the lungs).

● **Composition:** It is mixture of:

1-Phospholipids 77% = (diplmintoyl phosphatidyl cholin and glycerine).

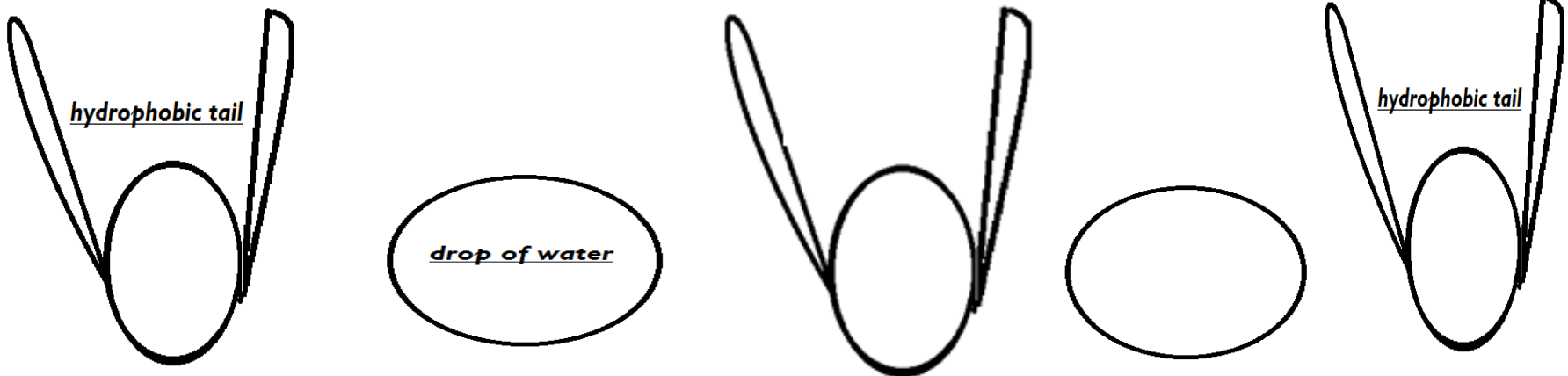
2-Lipoprotein 8%

3-Calcium ions 15%.



# ● Mechanism of Action

- Phospholipid is formed of two parts; hydrophilic head (towards the fluid) and hydrophobic tail (towards air). It interdigitates between the water molecules and prevents the attractive force between them.
- Calcium and lipoproteins allow for rapid and better spread of surfactant over the fluid surface.



# Surfactant Deficiency

Respiratory Distress Syndrome (= Hyaline Disease).

is common in

A- premature babies (↓ Glucocorticoids)

B-Babies with diabetic mothers (those babies were subjected to fetal hyperinsulinemia, which suppress the surfactant formation).

-Effect: The surface tension is abnormally high; the work of breathing is markedly increased. alveoli are collapsed & death may occur.

# COMPLIANCE OF THE LUNGS

- Def., A unit change in lung volume per the unit change in distending pressure.

Distending pressure of the lung is the difference between intrapleural pressure and alveolar pressure. This pressure is called the transpulmonary (transmural) pressure.

$$\text{Compliance} = \frac{\Delta V}{\Delta P} \text{ where } \Delta V \text{ is the change in volume, and } \Delta P \text{ is the change in pressure}$$



# RESPIRATORY MUSCLES

## I-Muscles of Inspiration: 1-Main Muscles:

A- Diaphragm:- (supplied by the phrenic nerve (origin from C3 to C5). 70% Contraction (= descent) of the diaphragm leads to enlargement of the thoracic cavity vertically.

B-External Intercostal Muscles: Contraction of them lead to elevation and eversion of the ribs.

2-Accessory Muscles:-Act only in forced inspiration.

-They are sternomastoid (elevates the sternum), scaleri (elevates the 1<sup>st</sup> rib), seratus posterior superior and seratus posterior inferior (elevate the remaining ribs).

## **II-Muscles of Expiration:**

Expiratory muscles act only in forced expiration.

### **A-Abdominal Wall Muscles:**

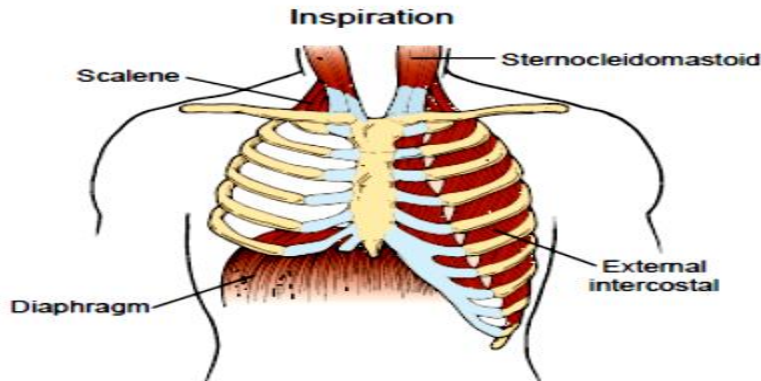
(i.e. abdominal recti, transverses abdominis, internal and external oblique muscles). Contraction leads to compression of abdominal contents which increases the intra-abdominal pressure and elevates the diaphragm upward.

### **B-Internal Intercostal Muscles:**

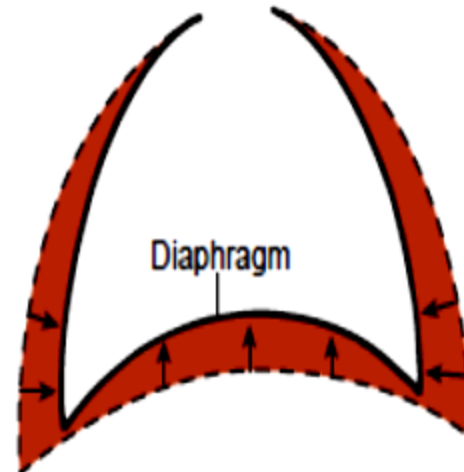
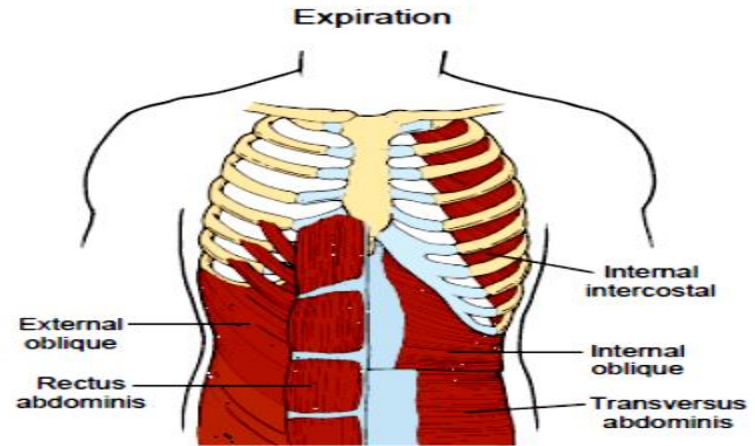
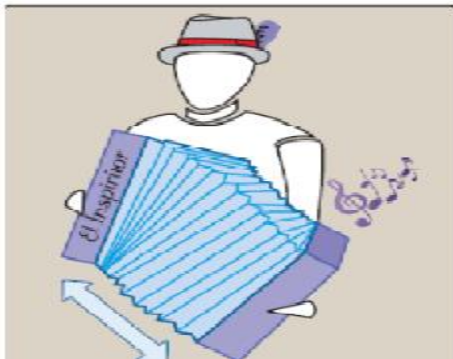
Contraction of the internal intercostal muscles leads to depression and inversion of the ribs.



# RESPIRATORY MUSCLES



**daphragm = descend**

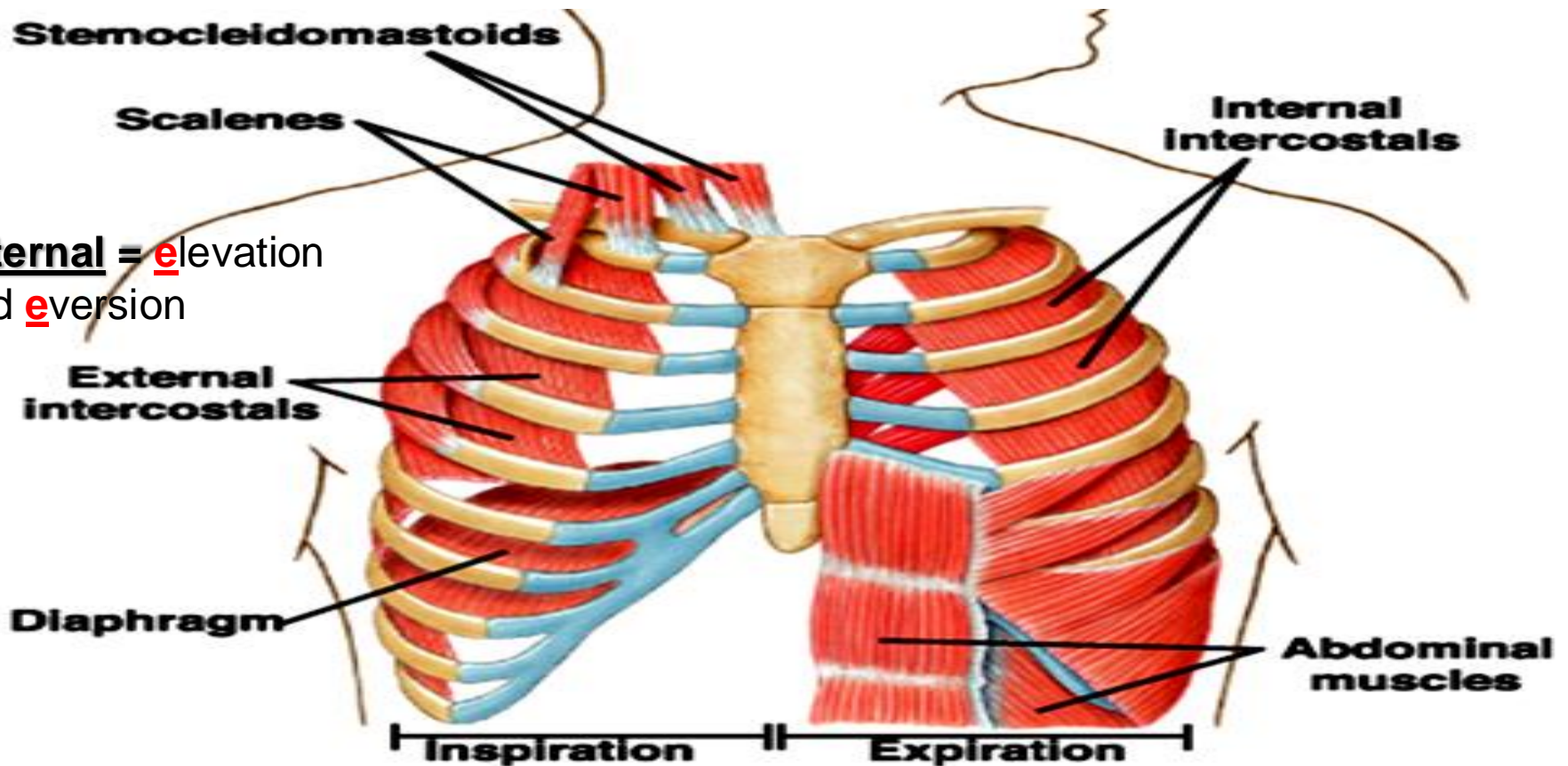


**external = elevation and eversion**

# RESPIRATORY MUSCLES

**d**iaphragm = **d**escend

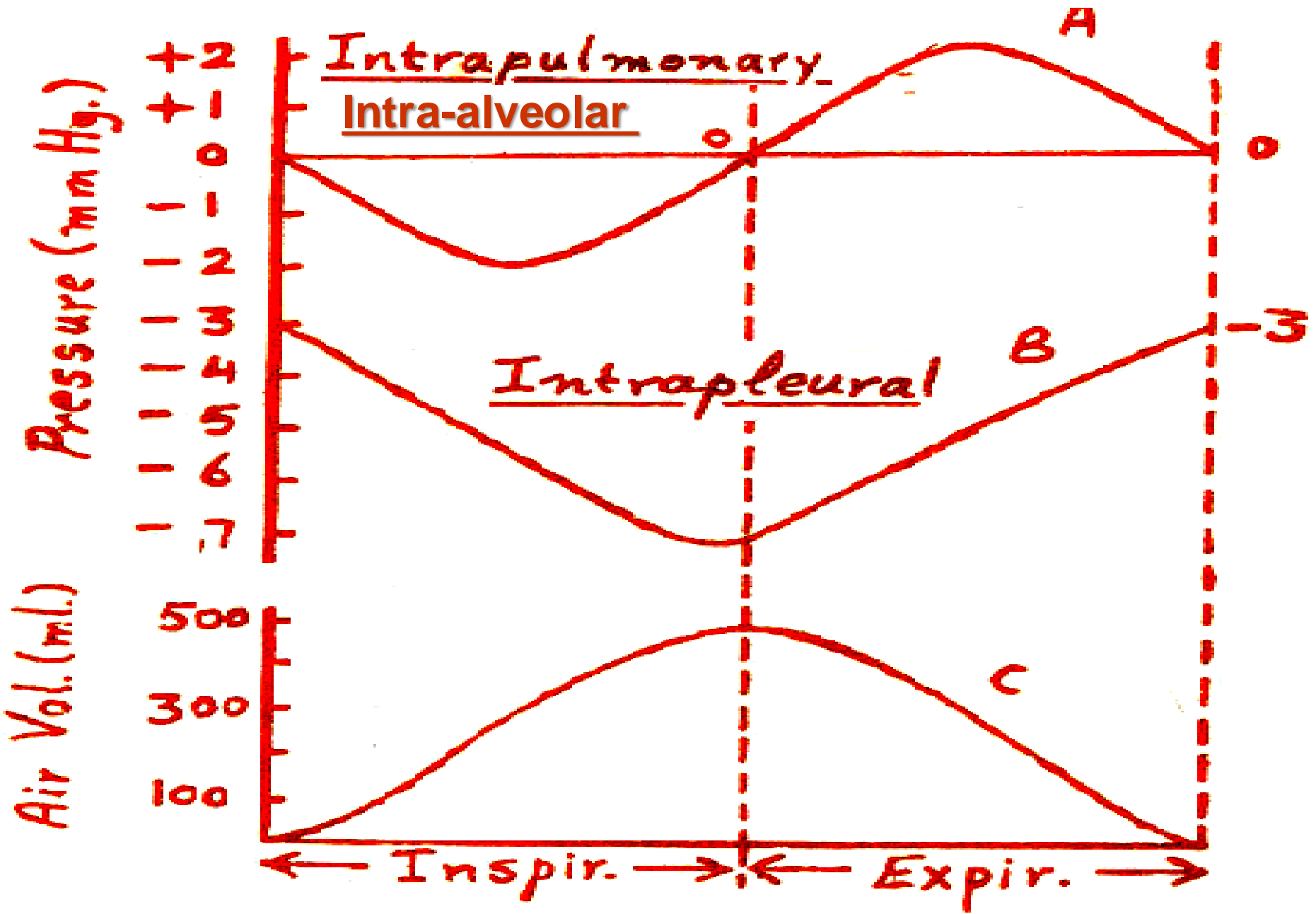
**e**xternal = **e**levation  
and **e**version

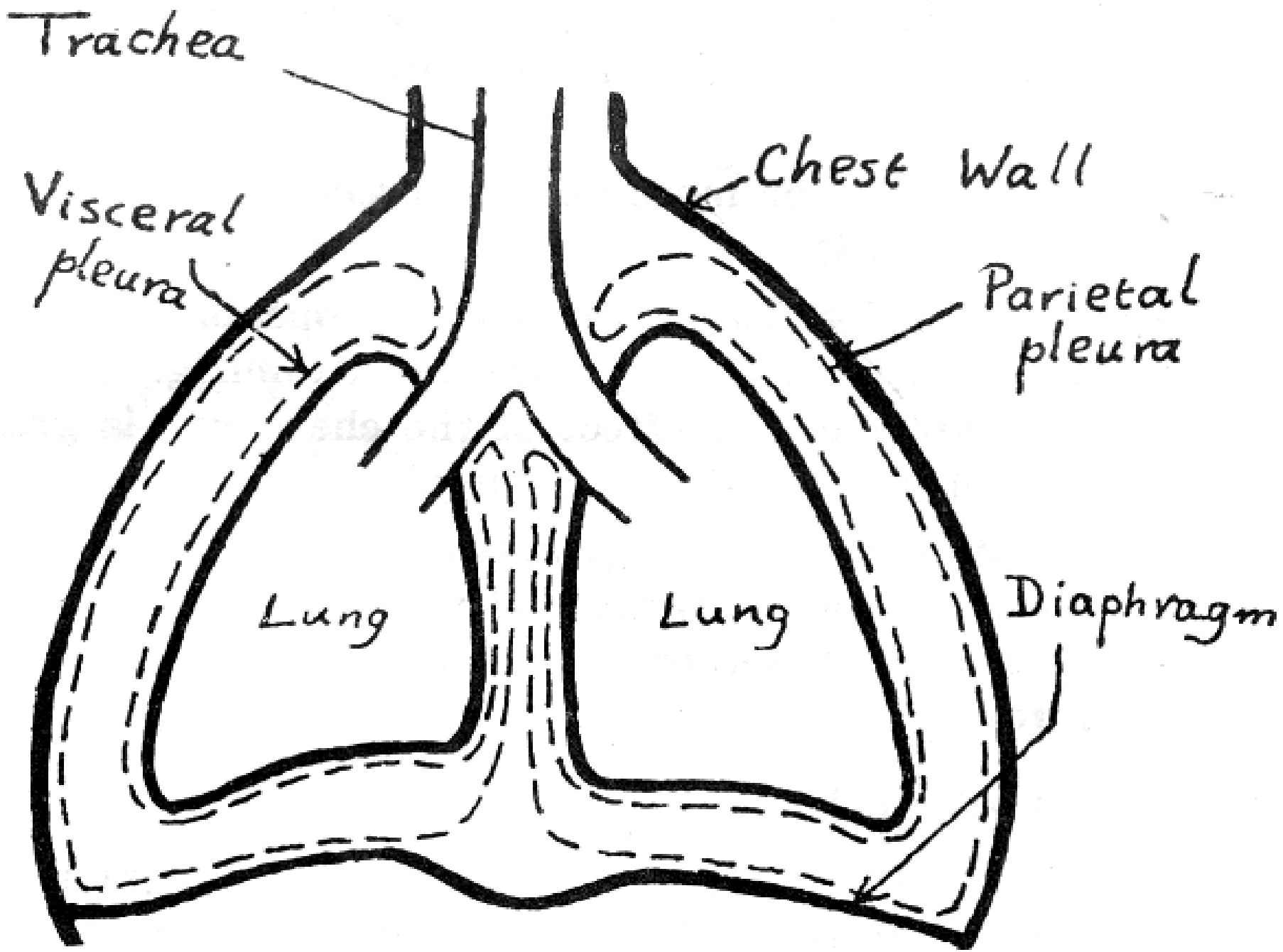


# Pulmonary Pressures

- 1-Atmospheric pressure (zero mm Hg = 760 mm Hg).
- 2-Intrapleural pressure (always negative =  $-3$  mmHg).
- 3-Intrapulmonary (intra-alveolar) pressure: It is equal to atmospheric pressure so it called zero mmHg. It decreases to  $-1$  mmHg during inspiration and increases to  $+1$  mmHg during expiration.

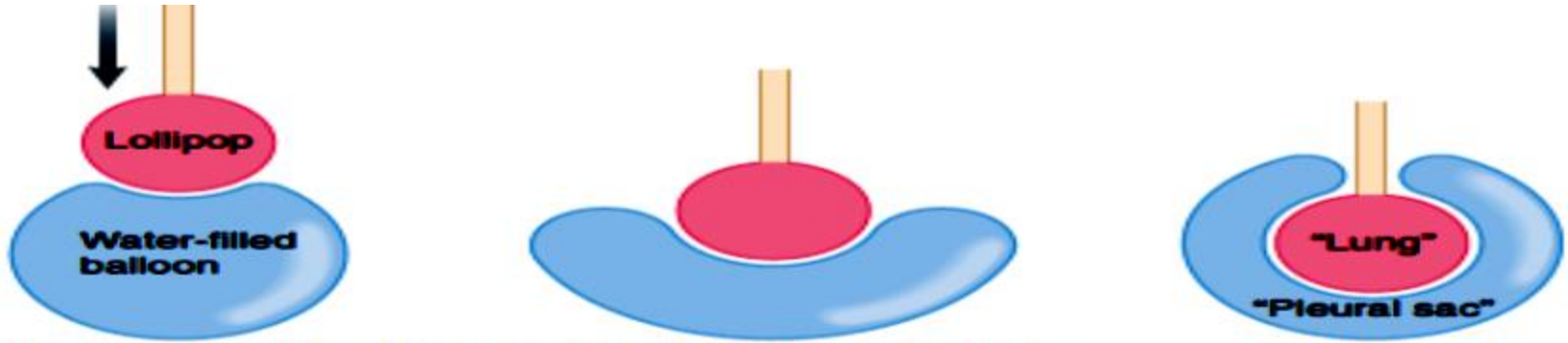
# Pulmonary Pressures



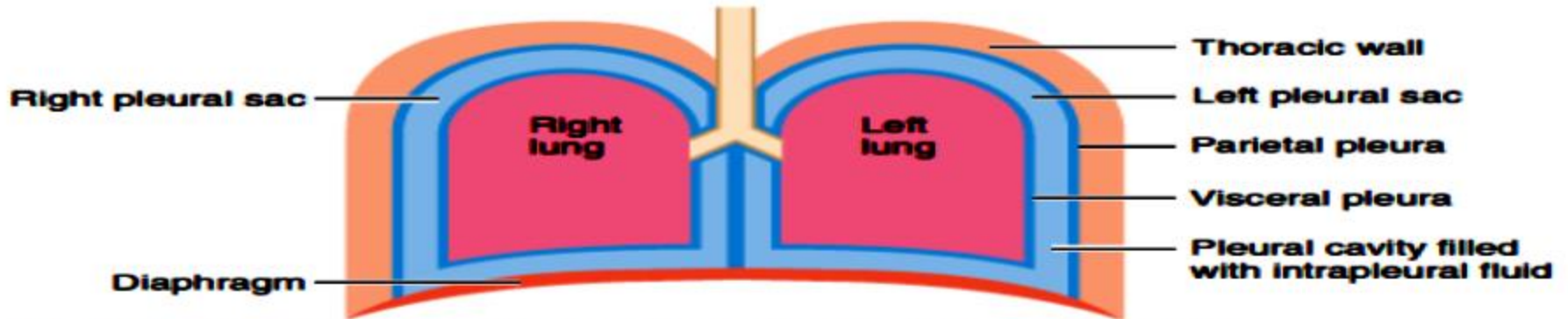


# INTRAPLEURAL PRESSURE (IPP)

● Def., It is the pressure of the fluid in the pleural cavity. It is always negative = SUBATMOSPHERIC



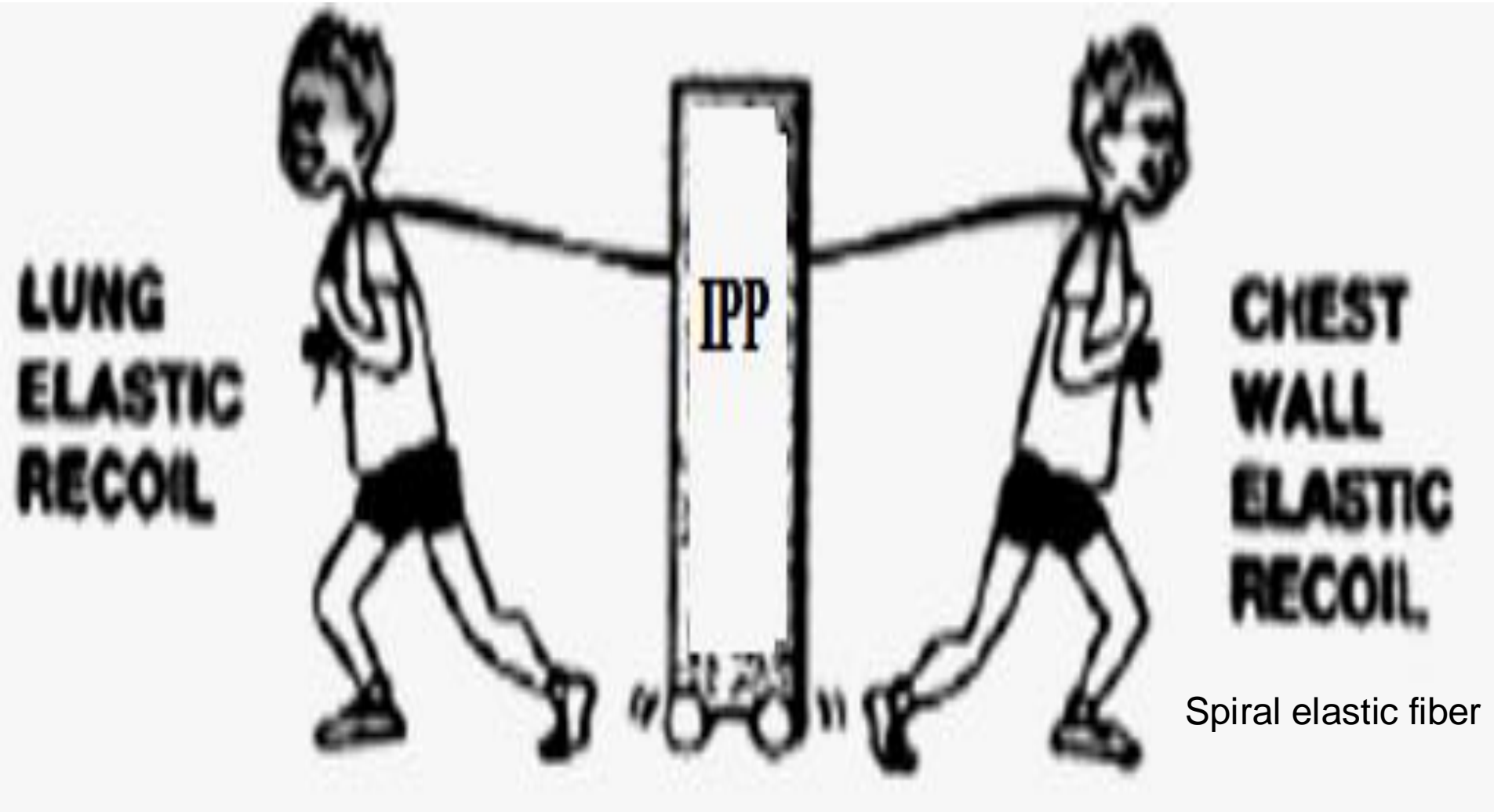
(a) Analogy of relationship between lung and pleural sac



(b) Relationship of lungs to pleural sacs, thoracic wall, and diaphragm

# ● Causes of Negativity of IPP

ذی الشفایط و المکنسه الکهربائیه



## ● Functions

- 1-Maintains the lung inflated and prevents its collapse specially during expiration.
- 2-It helps the expansion of the lungs during inspiration
- 3-It helps the venous return from extra-thoracic veins (+ve pressure) to intra-thoracic veins (-ve pressure).
- 4-It helps the lymph return from extra-thoracic veins (+ve pressure) to intra-thoracic veins (-ve pressure).



# MECHANISM OF BREATHING

## 1-Inspiration:

### A-In Normal Resting Inspiration:

Respiratory center → phrenic and external intercostal nerves → contraction diaphragm and external intercostal muscles → expansion of the chest wall in all directions → distention of the lung and decrease of intrapulmonary pressure to (-1 mm Hg) & rush the air into the lungs.

### B- In Forced Inspiration:

The main and the accessory muscles contract strongly so, greater increase in the thoracic cavity and rush more volume to the lungs.

## 2-Expiration:

### A-In Normal Resting Expiration:

-Normal expiration is the passive process. It is produced by relaxation of inspiratory muscles. Drop of the thoracic cage and elevation of diaphragm increase of intrapulmonary pressure to (+1 mm Hg) & rush the air out the lungs.

### B- In Forced Expiration:

The expiratory muscles contract strongly so, more depression and inversion of ribs more decrease in the thoracic cavity and rush more volume out of the lungs.

	<i>inspiration</i>	<i>expiration</i>
Nature	active	passive
Duration	longer	shorter
Dimensions	expansion in 3 dimensions	decrease in 3 dimensions (lung recoils)
	<p>Increased volume → decreased pressure (Boyle's law)</p> <ul style="list-style-type: none"> <li>• I mean the intrapulmonary (intra-alveolar pressure)</li> </ul> <p>pressure decreased to -1 cmH<sub>2</sub>O</p> <p>assuming that the atmospheric pressure is zero.</p>	<p>decreased volume → increased pressure (Boyle's law)</p> <ul style="list-style-type: none"> <li>• I mean the intrapulmonary (intra-alveolar pressure)</li> </ul> <p>pressure increased to +1 cmH<sub>2</sub>O</p> <p>assuming that the atmospheric pressure is zero.</p>
Muscles	<ul style="list-style-type: none"> <li>• Diaphragm: descends.</li> <li>• external intercostals: <ul style="list-style-type: none"> <li>◦ → elevate ribs → increase transverse diameter.</li> <li>◦ → evert ribs → increases AP diameter</li> </ul> </li> </ul>	<p>— passive—</p> <p>-diaphragm ascends, lungs shrink by their elastic recoil.</p>
Accessory muscles for forced...	<p>Forced inspiration:</p> <ul style="list-style-type: none"> <li>• Sternocleidomastoid.</li> <li>• serratus anterior</li> <li>• scalene muscles</li> </ul>	<p>Forced expiration (voluntary "musical instruments", obstructive [COPD], restrictive [fibrosis]):</p> <ul style="list-style-type: none"> <li>• internal intercostals</li> <li>• abdominal muscles "abdominal recti" [abdominal breathing]</li> </ul>

Thanks

