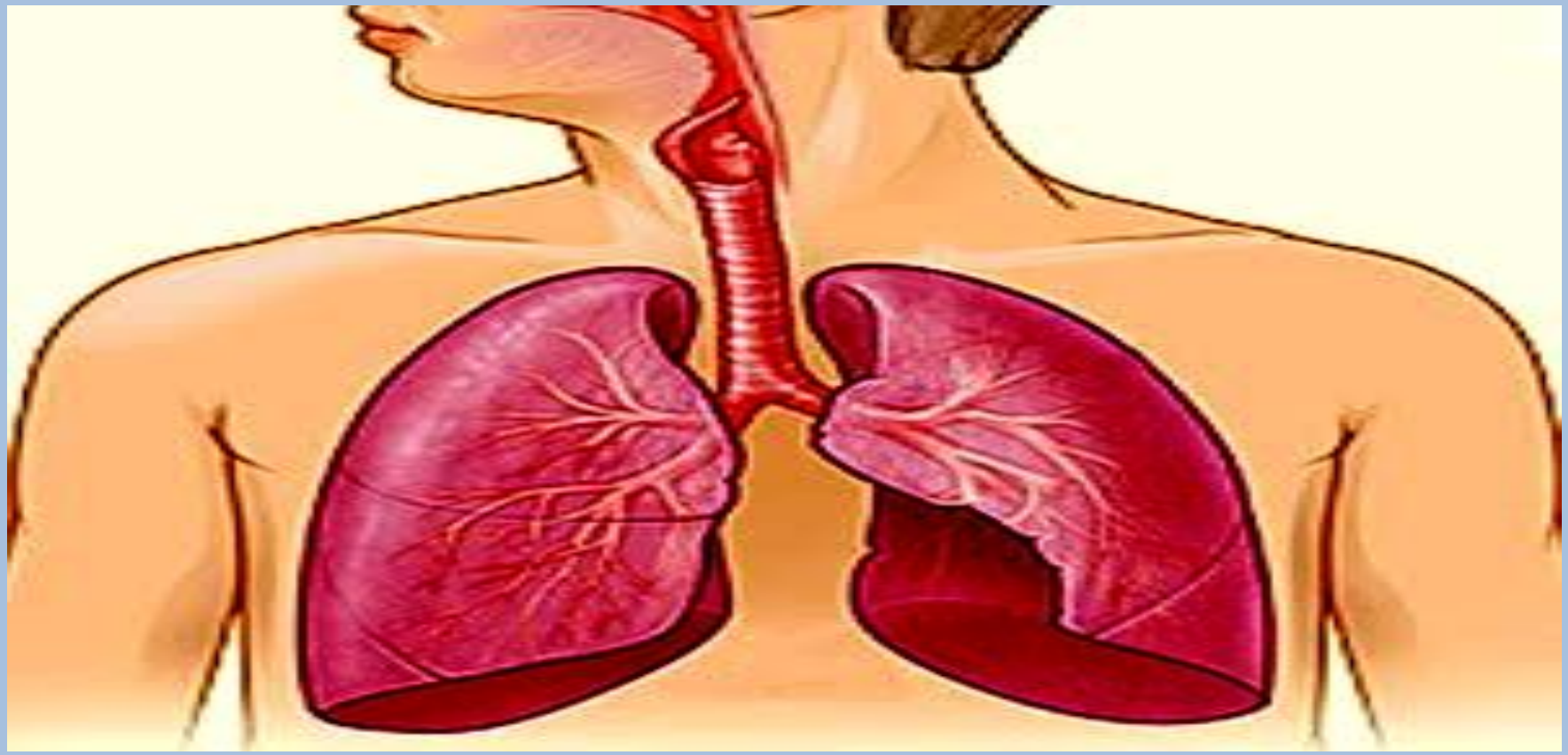
PHYSIOLOGY OF

Pulmonary Ventilation



Prof. Khaled Abdel-Sater, MD

AIR PASSAGE

Anatomically is divided into:

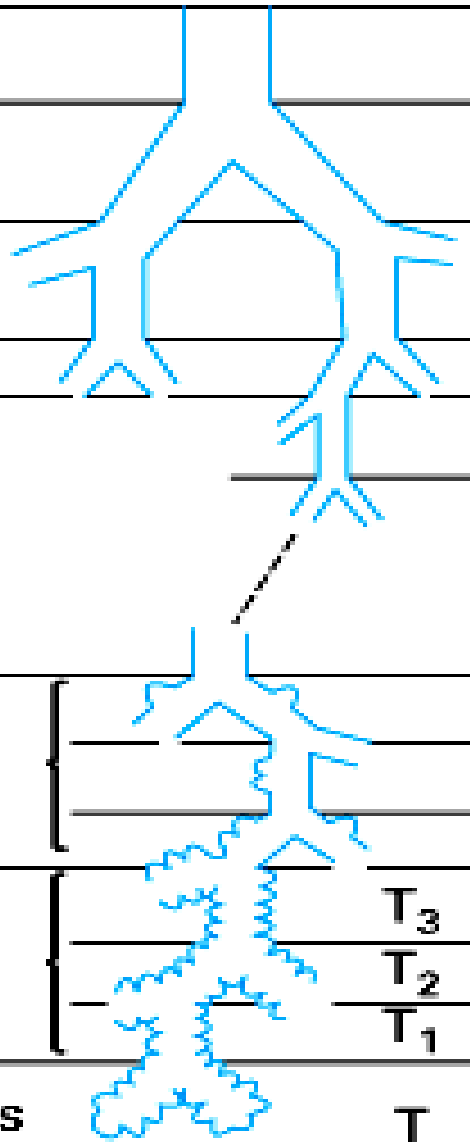
- Upper airway passages: Nose & pharynx.
- Lower airway passages: Larynx, trachea, bronchi & bronchioles.
- **Physiologically is divided into:**

❑ **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
	bronchioles	4	0.45	16
	terminal bronchioles	5 ↓ 16	0.35 ↓ 0.06	32 ↓ 6×10^4
Transitional and Respiratory zones	respiratory bronchioles	17 18 19	↓ 0.05	↓ 5×10^5
	alveolar ducts	T_3 20 T_2 21 T_1 22	↓	↓
	alveolar sacs	T 23	0.04	8×10^6

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.

N.B

68 kgm=150 Pound

1 Pound=2.2 kgm

كمية الهواء يلي بتدخل عالجسم مع كل نفس تساوي 500ml منهم 150ml ما بصيرلهم تبادل غازات وبكونوا موجودين بال *Dead Space*

●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.

Give reasons

No gas exchange between dead space air and blood in the alveolar capillary.

Because it has a thick wall

The anatomical dead space is equal to the physiological dead space.

Because under normal conditions, there is no alveolar dead space.

***أسئلة الدكتور

● Functions of Dead space :

1- Air conduction.

لو الهواء الناشف دخل لل alveoli بدون ترطيب
راح يصير للحويصلات Collapse and irritation

2- Air condition. → Add heat or cold

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).

Function 2 : Air condition (what we mean by add heat or cold ?)

ال dead space بشتغل على انه مبادل حراري طبيعي في الجسم بحيث أنه إذا كان الهواء الداخل للجسم بارد ف dead space بكون محتفظ بهواء دافئ داخله وبعادل الحرارة عشان الهواء الخارجي البارد ما يسبب تلف في أنسجة الرئة ونفس الفكرة في حالة كان الهواء الخارجي جدا سخن

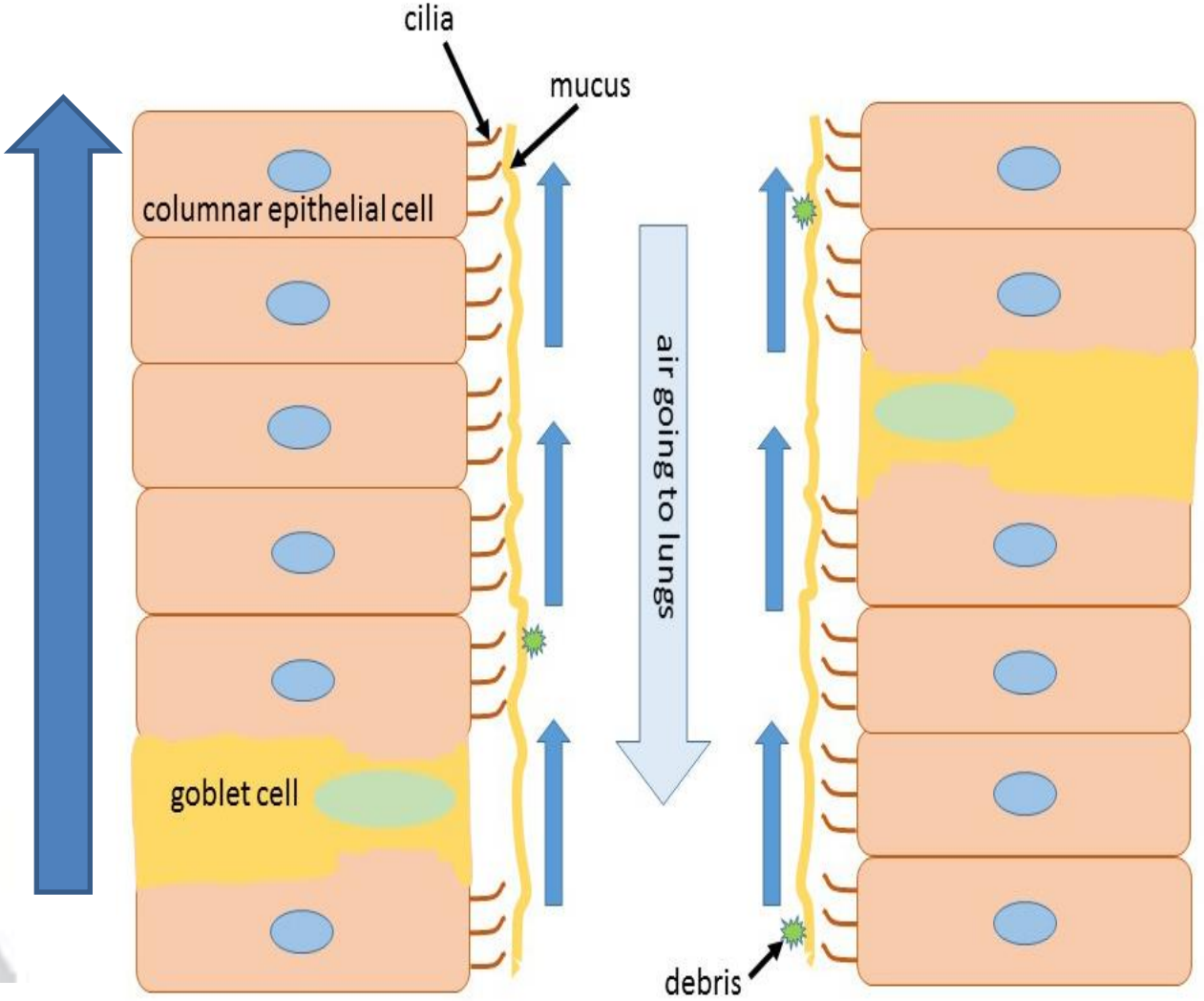
Function 4 : Filtration and cleaning of the air

أكثر من 10micron : يتم حجزها بواسطة الشعيرات الموجودة داخل الأنف.

بين 2 و 10 micron : يتم طردها عن طريق العطس أو السعال.

أقل من 2 micron : تتولى macrophages مهمة التعامل معها والتخلص منها داخل الجسم.

Ciliary Escalator



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

التدخين بدمر ال cilia الموجودة بال trachea and bronchi
(يعني بالأحرى بدمر ال ciliary escalator) فبصير مع
المدخنين مرض يسمى ب Kartagener syndrome
وأقل عرض من أعراض هاد المرض انه بكونوا معرضين
للعدوى بشكل متكرر

ELASTIC RECOIL OF THE LUNGS

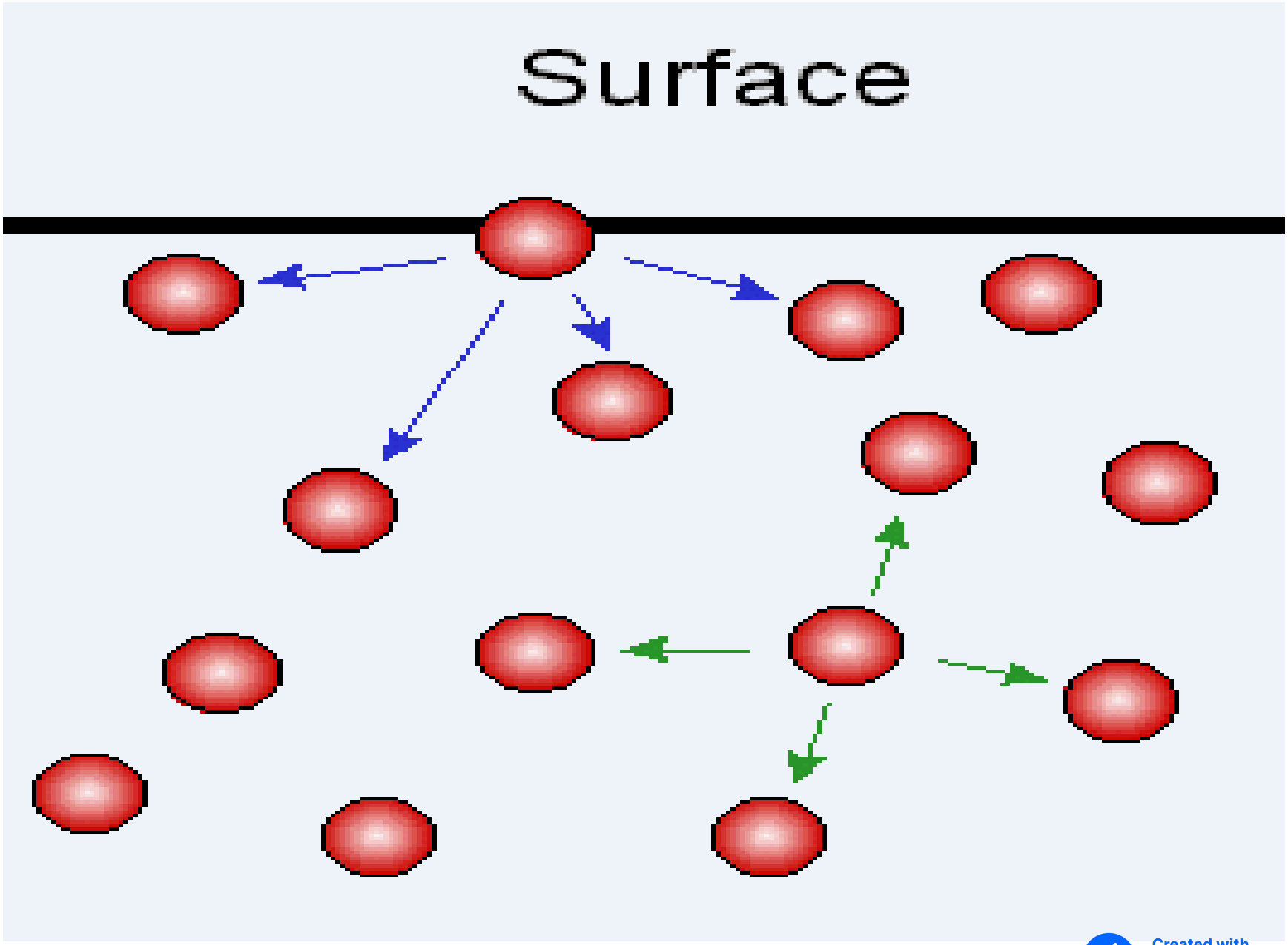
●Def.,

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%).



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.**

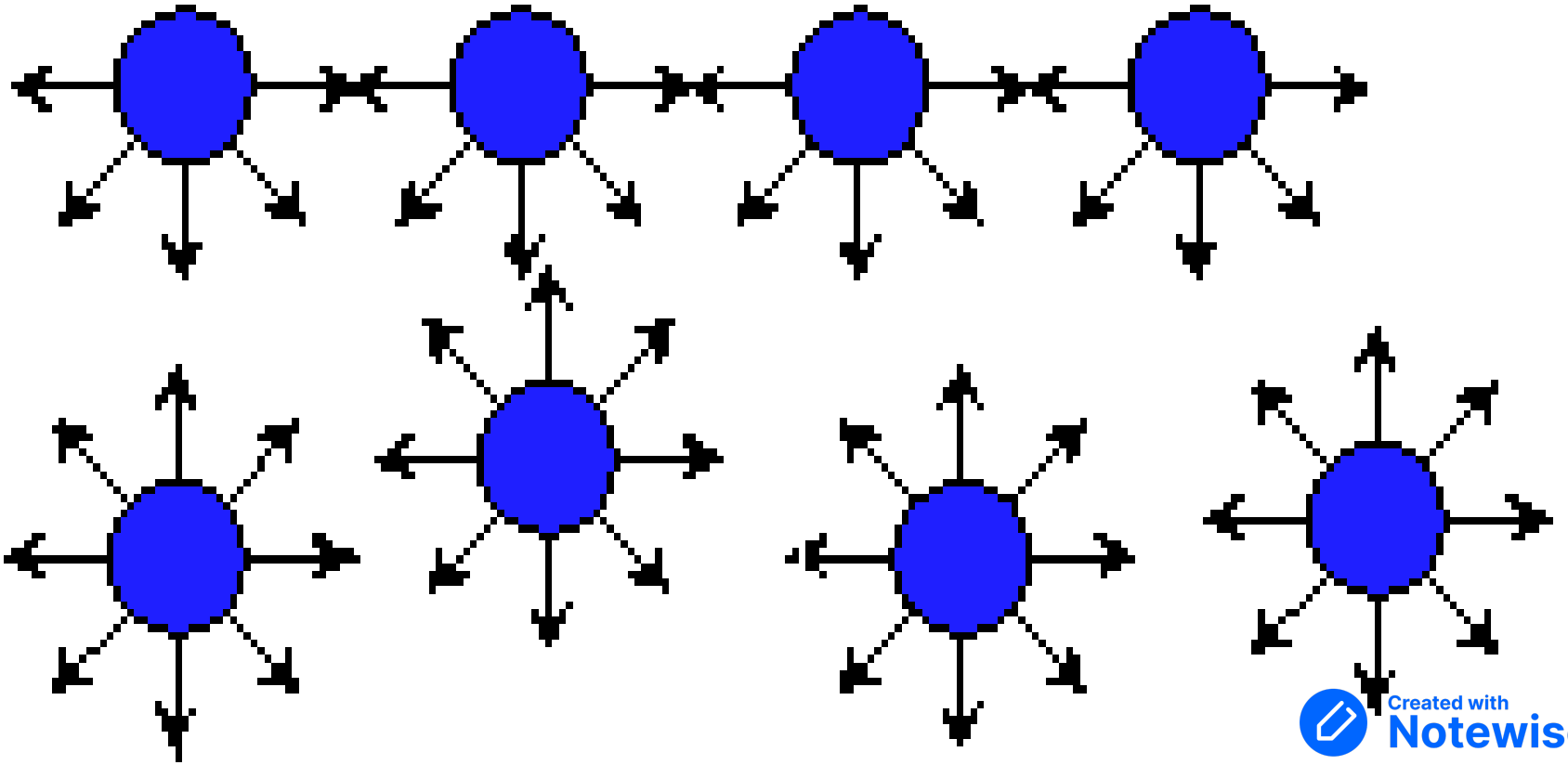
2- **Decreases the muscular work of breathing** and increases the pulmonary compliance (reducing the effort needed to expand the lungs).

● **Composition:** It is mixture of:

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

2-Lipoprotein 8%

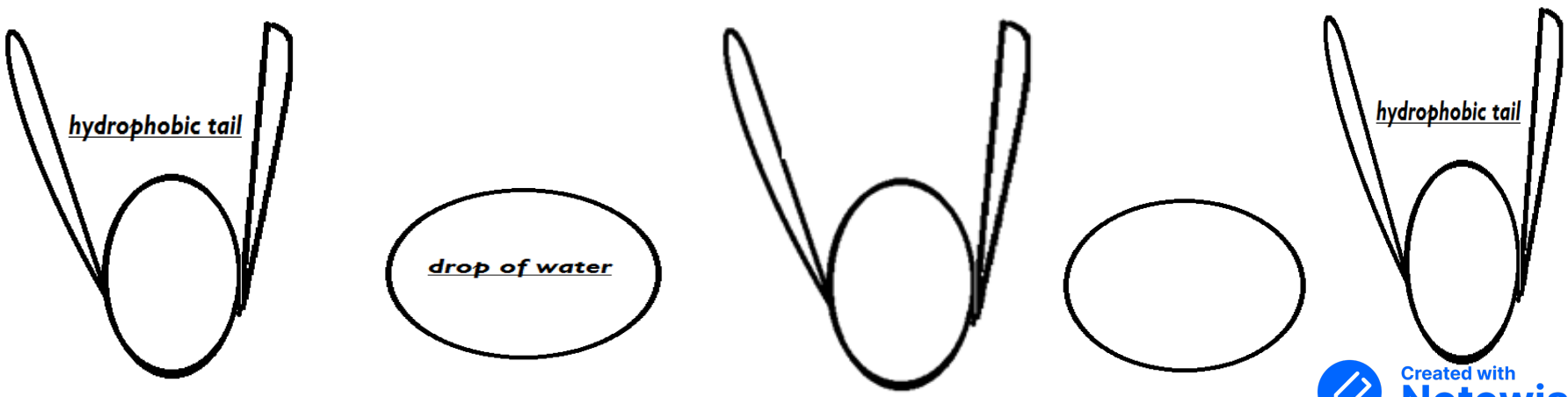
3-Calcium ions 15%.



●Mechanism of Action

-Physpholipid 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.

why Premature babies have surfactant deficiency ?

Premature babies, born before 34 weeks of gestation, often experience respiratory distress syndrome (RDS). This is primarily due to a deficiency of surfactant, a substance that coats the tiny air sacs in the lungs, reducing surface tension and preventing them from collapsing.

Surfactant production, a crucial step in lung development, is typically stimulated by glucocorticoids, hormones produced by the adrenal cortex. In premature babies, the adrenal cortex may not be fully developed, leading to insufficient glucocorticoid secretion and, consequently, delayed surfactant production. This immaturity in surfactant levels can result in respiratory distress, as the lungs struggle to expand and contract effectively.

Why do babies of diabetic mother have surfactant deficiency ? Fetal hyperinsulinemia can cause low blood sugar, which can stunt fetal growth, including lung development. This can lead to less surfactant production, increasing the risk of respiratory distress syndrome (RDS).

تفسيرات للسلايد يلي قبل اجتهاد شخصي الدكتور ما طلبهم

Give Reasons

Respiratory distress syndrome is common in premature babies. Due to immature alveolar epithelium which fails to secrete surfactant because deficiency of glucocorticoid.

***أسئلة الدكتور

Surfactant decreases surface tension?

Because 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.

Presence of fetal hyperinsulinemia in babies with diabetic mothers.

Diabetic mother has hyperglycemia that causes fetal hyperinsulinemia due to the presence of healthy fetal pancreas and easy diffusion of glucose by placenta.

طواعية الرئة

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 inrapleural pressure and alveolar pressure. This pressure is called the transpulmonary (transmural) pressure.

الضغط الخارجي

الضغط الداخلي

$$Compliance = \frac{\Delta V}{\Delta P}$$

where ΔV is the change in volume, and ΔP is the change in pressure

compliance = قابلية الرئة للتمدد / **elastic recoil** = **collapse** (عكس بعض)

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.

up-down movement

B-External Intercostal Muscles: Contraction of them lead to **e**levation and **e**version of the ribs.

increase the thoracic space
side to side anteroposterior

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

-They are **s**ternomastoid (elevates the sternum), **s**caleri (elevates the 1st rib), **s**eratus posterior superior and **s**eratus posterior inferior (elevate the remaining ribs).

إذا زادت المساحة بقل الضغط ، فعضلات الinspiration بتشتغل انه تزيد مساحة
الthoracic cage ف intrapleural Pressure بصير أقل من ال atmospheric
pressure فبدخل الهواء من الضغط الاعلى للأقل (من الخارج إلى الداخل)

II-Muscles of Expiration: Passive Process

Expiratory muscles act only in forced expiration.

relaxation of the inspiration muscles make the expiration process

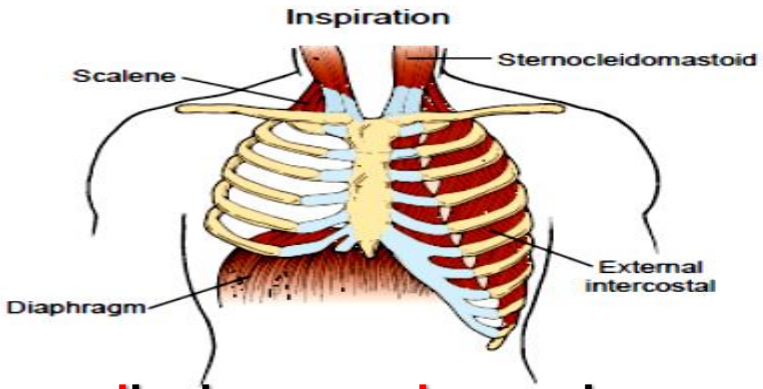
A-Abdominal Wall Muscles:

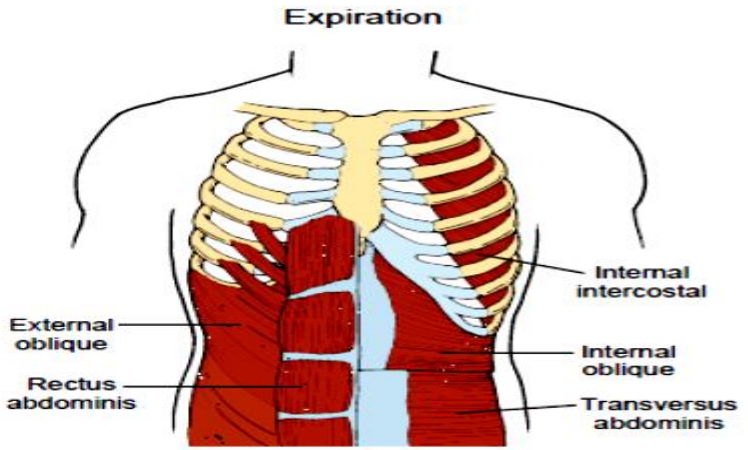
(i.e. abdominal recti, transverses abdominis, internal and external oblique muscles). Contraction leads to compression of abdominal contents which increases the intra-abdominal pressure and elevates the diaphragm upward.

B-Internal Intercostal Muscles:

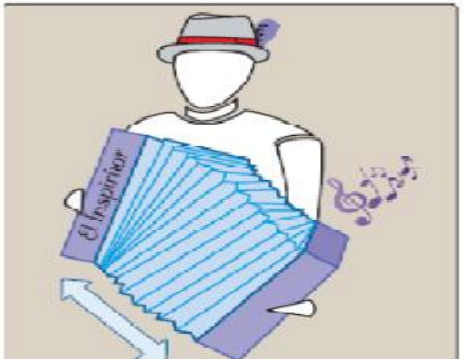
Contraction of the internal intercostal muscles leads to depression and inversion of the ribs.

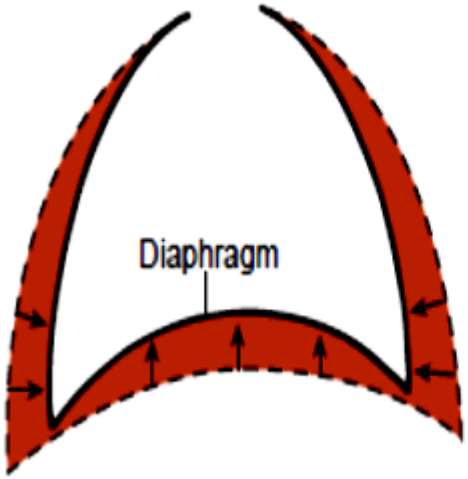
RESPIRATORY MUSCLES





diaphragm = descend



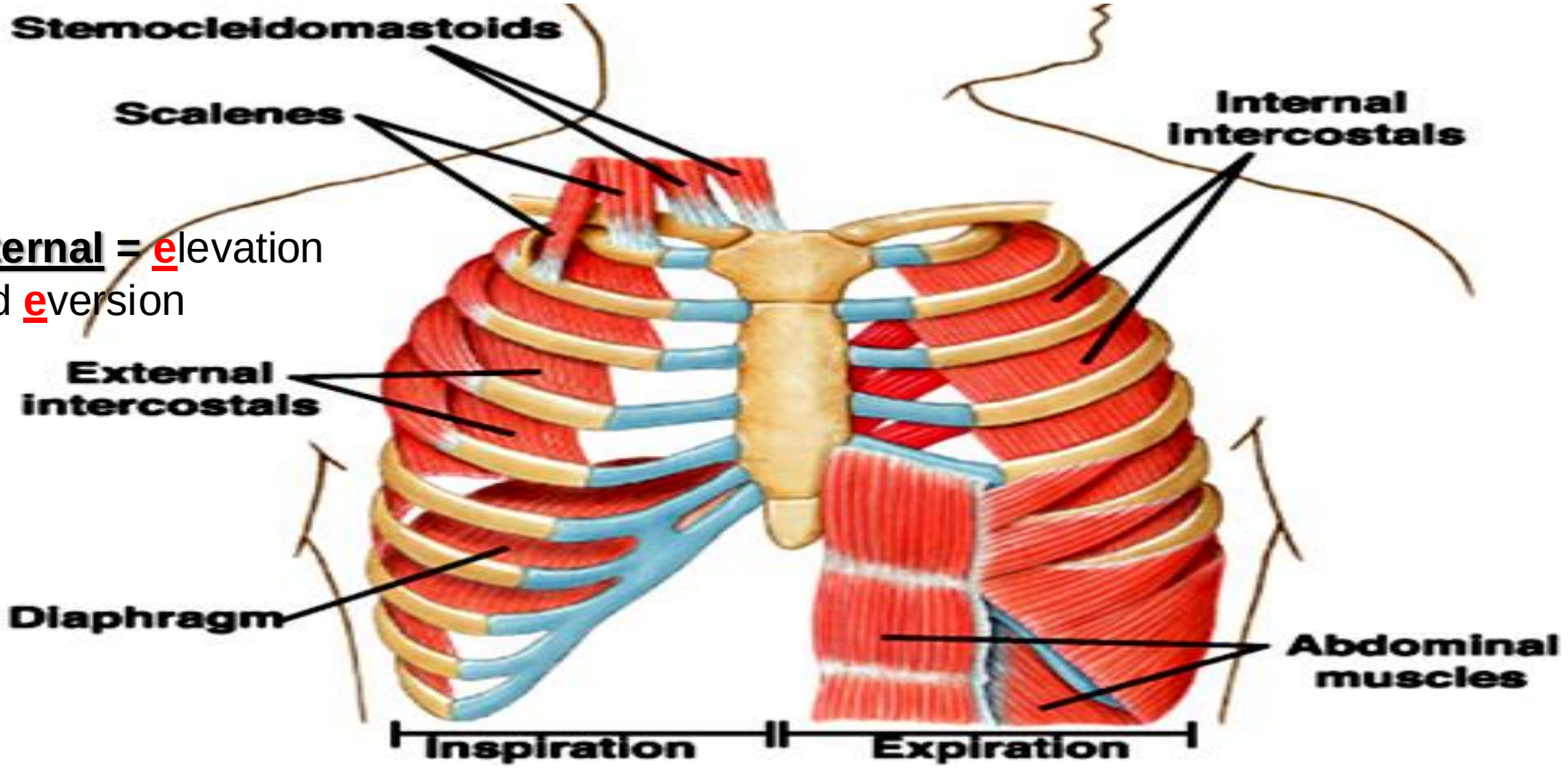


external = elevation and eversion

RESPIRATORY MUSCLES

diaphragm = descend

external = elevation and eversion



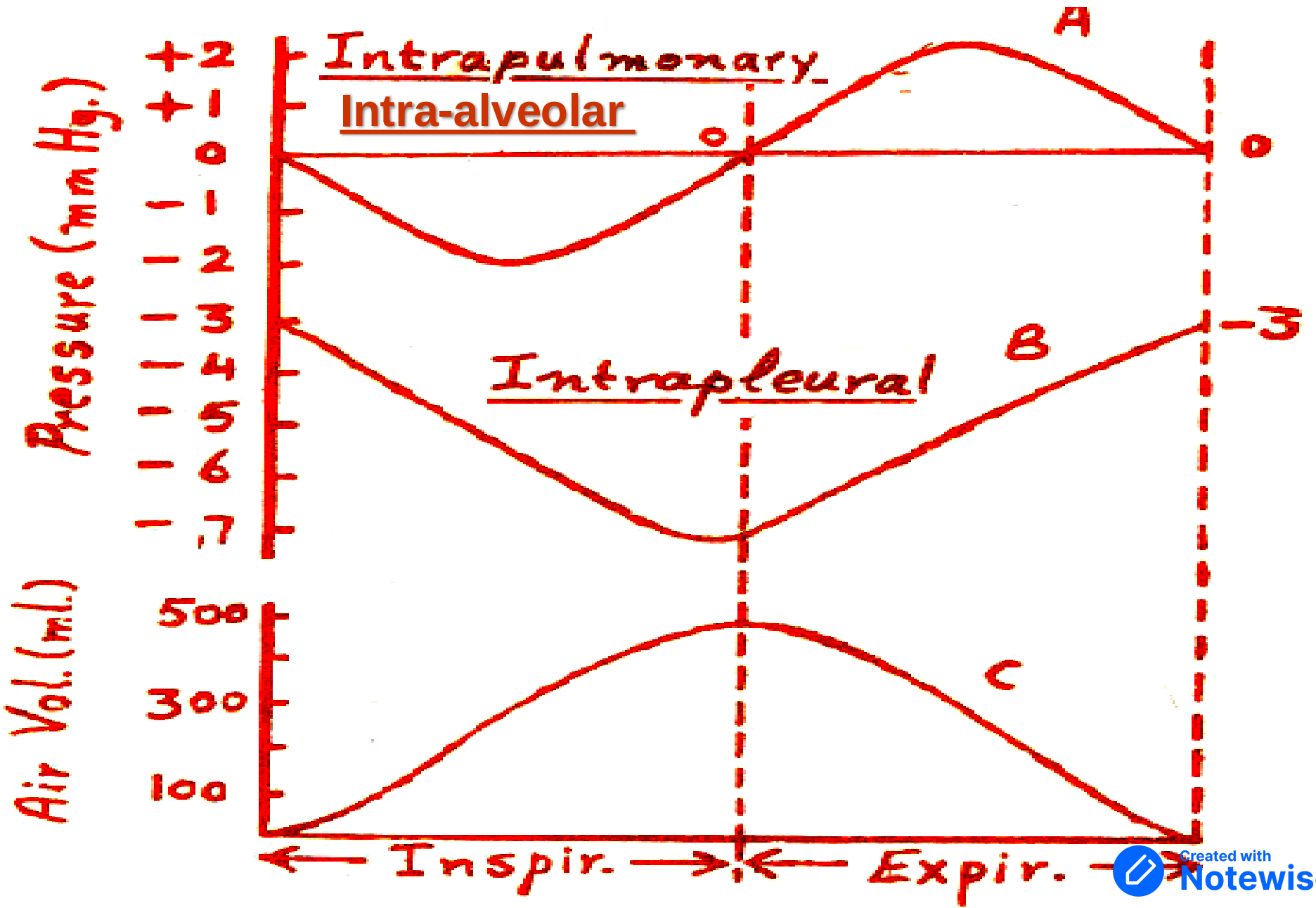
Pulmonary Pressures

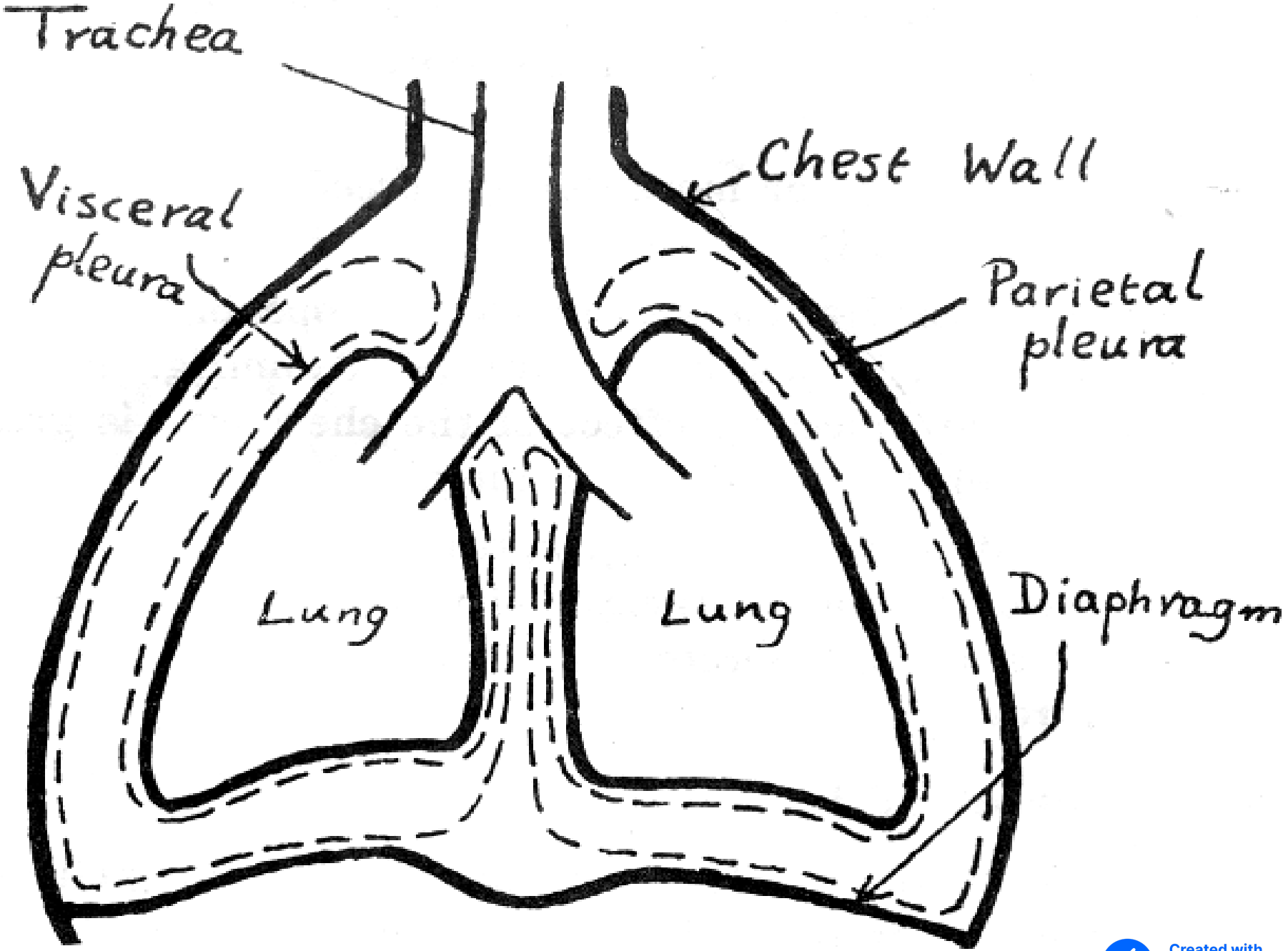
1-Atmospheric pressure (zero mm Hg = 760 mm Hg).

2-Intrapleural pressure (always negative = –3 mmHg).

3-Intrapulmonary (intra-alveolar) pressure: It is equal to atmospheric pressure so it called zero mmHg. It deceases to -1 mmHg during inspiration and increases to +1 mmHg during expiration.

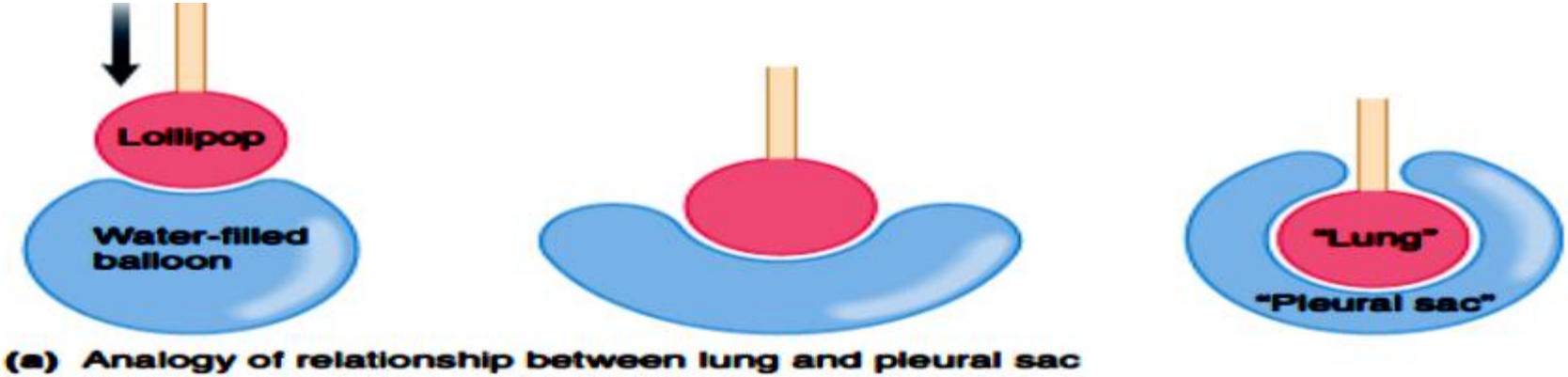
Pulmonary Pressures



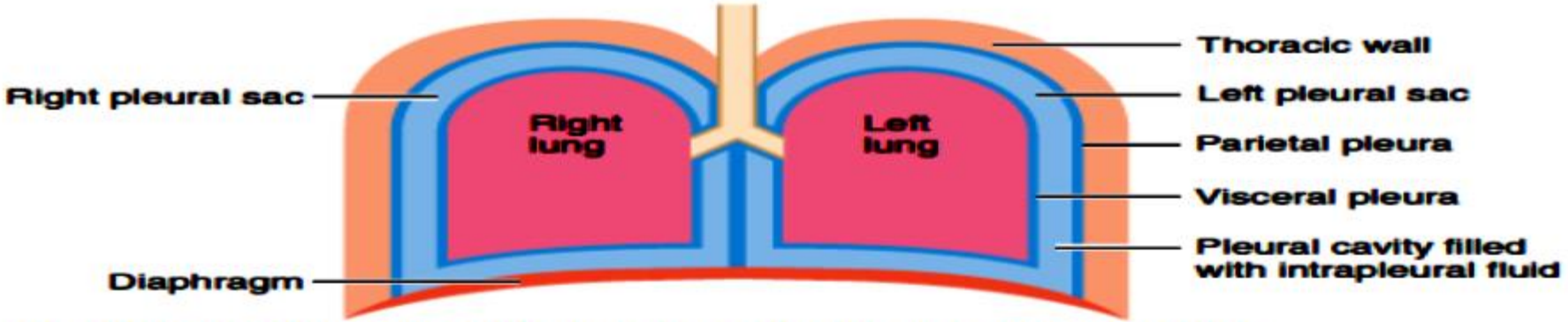


INTRAPLEURAL PRESSURE (IPP)

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



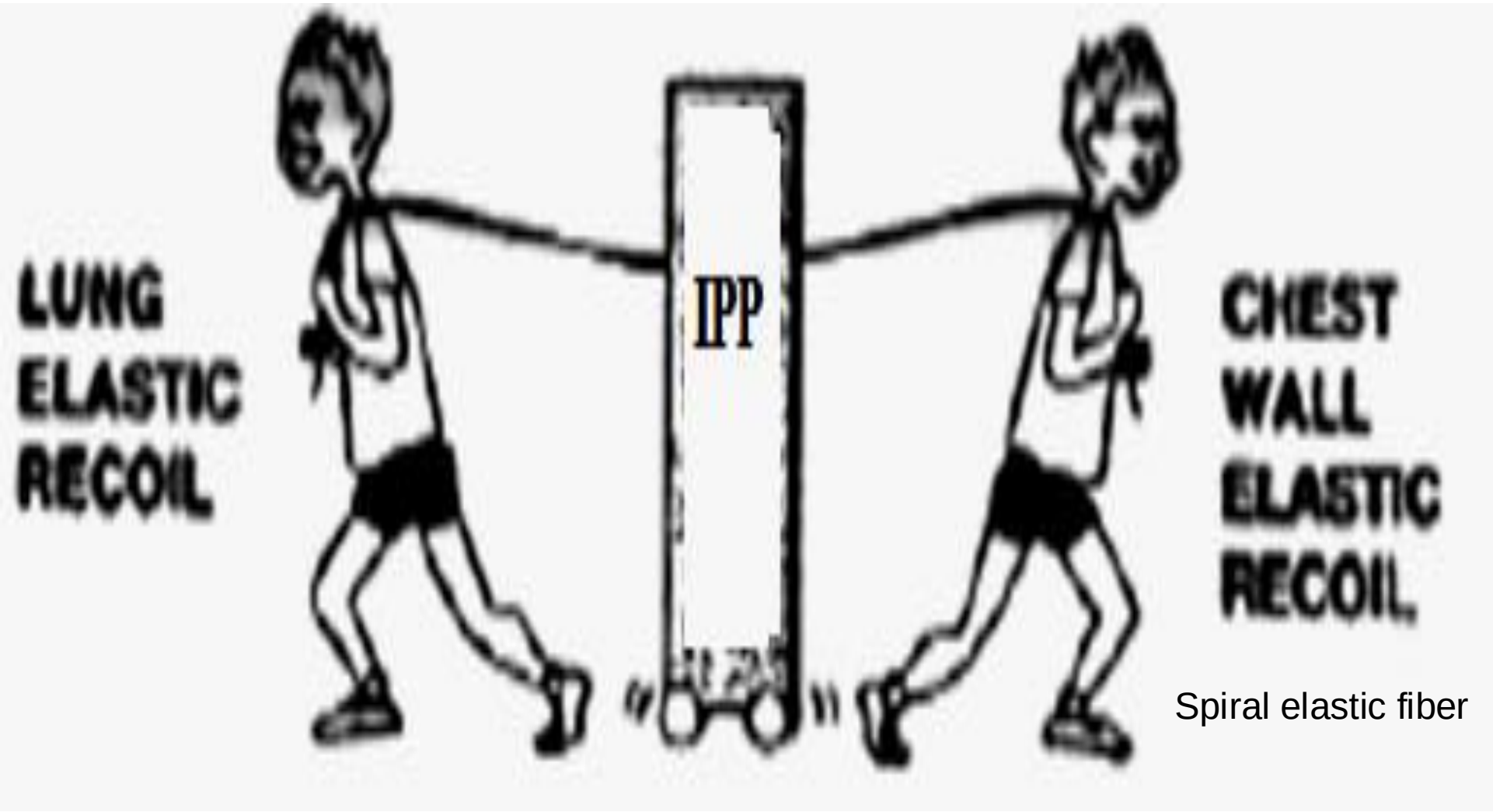
(a) Analogy of relationship between lung and pleural sac



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

●Causes of Negativity of IPP

ذى الشفاط و المكنسه الكهربائيه



Spiral elastic fiber

●Functions

+ surfactant

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).

When intrapleural pressure becomes positive, it can disrupt the normal mechanisms of venous return, particularly in elderly patients, and this disruption may contribute to syncopal attacks.

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.

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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.

	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-alveolar 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-alveolar pressure)* pressure increasd to +1 cmH2O *assuming that the atmospheric pressure is zero.*
Muscles	• Diaphragm: decends. • external intercostals: ◦ –> elevate ribs –> increase transverse diameter. ◦ –> overt 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]

Thanks