# Pulmonary Ventilation

# MEDEMIA ACADEMY


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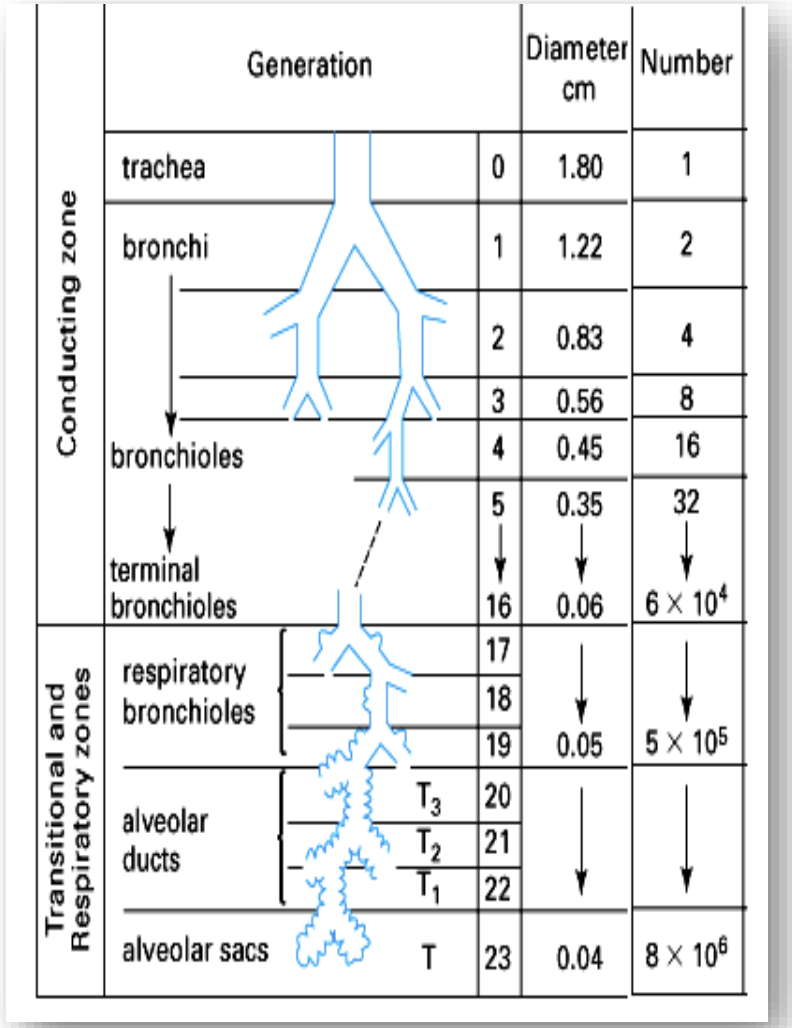
# AIR PASSAGE

- **Anatomically is divided into:**
  - **A. Upper airway passages:** Nose and pharynx.
  - **B. Lower airway passages:** Larynx, trachea, bronchi and bronchioles.
- **Physiologically is divided into:**
  - **A. Conducting Zone (Dead Space):**
    - Nose to terminal bronchioles (branching number 16).
    - The wall of the conducting part is thick, so no gas exchange, therefore it called dead space.
  - **B. Respiratory Zone:**
    - Site for gas exchange (thin wall).
    - Respiratory bronchioles to alveoli.
    - Air present in it is called alveolar air.



# DEAD SPACE(D.S)

- **Definition:**
- 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(average).
- N.B: Dead space may be predicted by multiply the weight in Kgm by 2.
  - 68kgm=150 pound.
  - Kgm=2.2

# Types of Dead Space

1. **Anatomical Dead Space:** = 150 ml.
2. **Alveolar Dead Space:** always pathological, that normally equal to zero.
   - **Non functional alveoli are present in:**
     A. Alveoli not received blood e.g., pulmonary thrombosis.
     B. Collapsed alveoli(no air in 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.

❖**Note:** 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

1. **No gas exchange between dead space air and blood in the alveolar capillary?**
   - Because it has a thick wall.
2. **The anatomical dead space is equal to the physiological dead space ?**
   - Because under normal condition, there is no alveolar dead space.

# Function of Dead Space

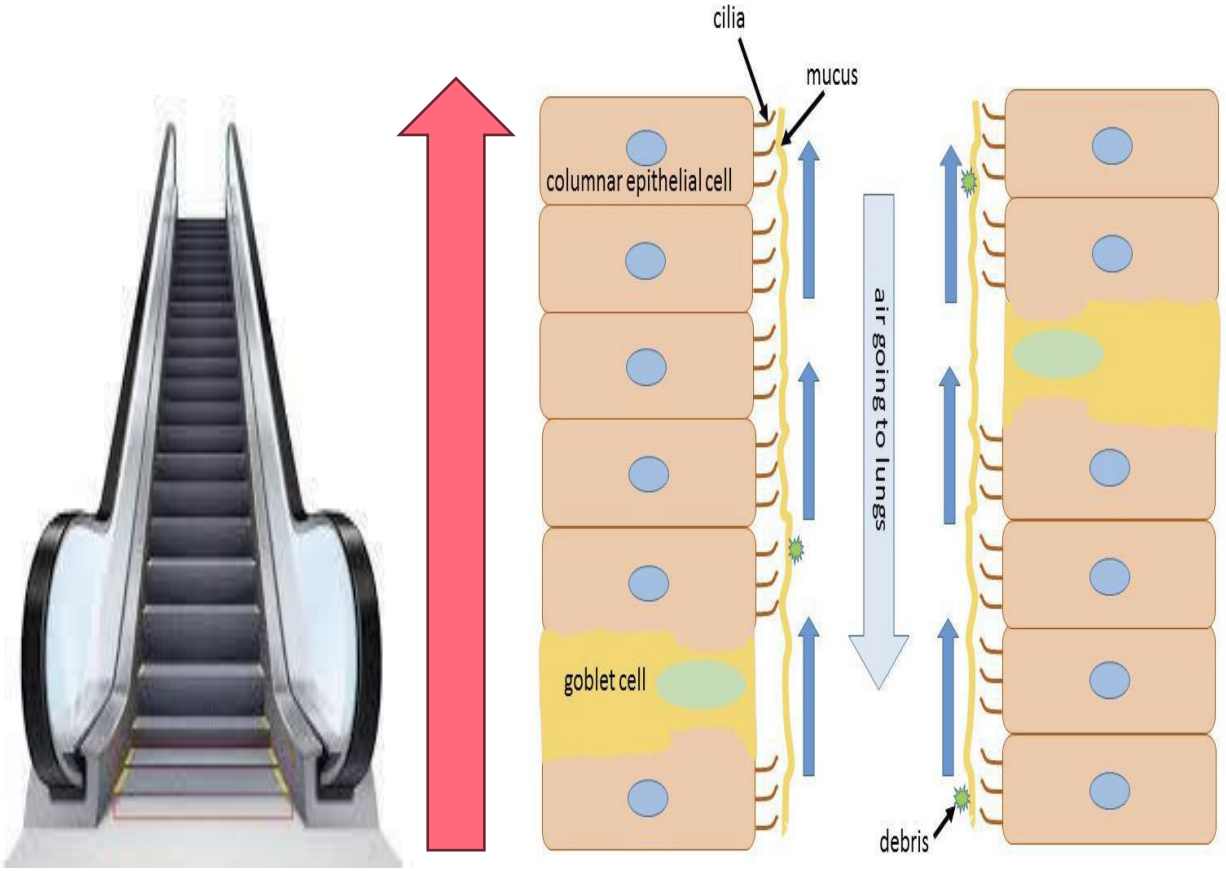
1. Air conduction.
2. Air condition(warmness or coldness the incoming air to body temperature, which helps prevent damage to the delicate lung tissues and facilitates gas exchange).
3. Moistening of air by mucous secretion(helps to humidify the incoming air, this is crucial because dry air can irritate the delicate alveolar surfaces and impair gas exchange and collapse of alveoli).
4. Filtration and cleaning of the air(from pathogen, according to it's size and diameter).
   - <2 Micrometers: Removed by alveolar macrophages in the alveoli(also known as dust cells).
   - 2-10 Micrometers: Expelled through coughing.
   - >10 Micrometers: Trapped in the nasal cavity by hairs and mucus.

# Function of Dead Space Cont.

5. **Protection of lung due to presence of:**
   A. **Trachea and bronchi cilia(ciliary escalator).**
      - Smoking and genetic disorders like Kartagener syndrome impair ciliary function, lead to mucus accumulation in the airways.
      - Accumulated mucus creates an environment for bacterial growth, leading to recurrent respiratory infections.
   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

❖**Definition:**

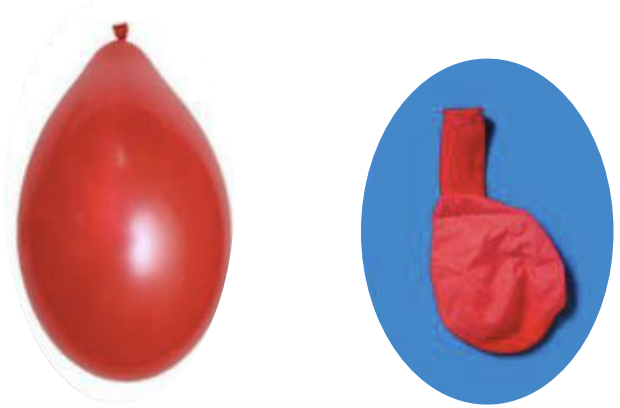
❖When the lung is inflated, it tends to recoil(collapse).

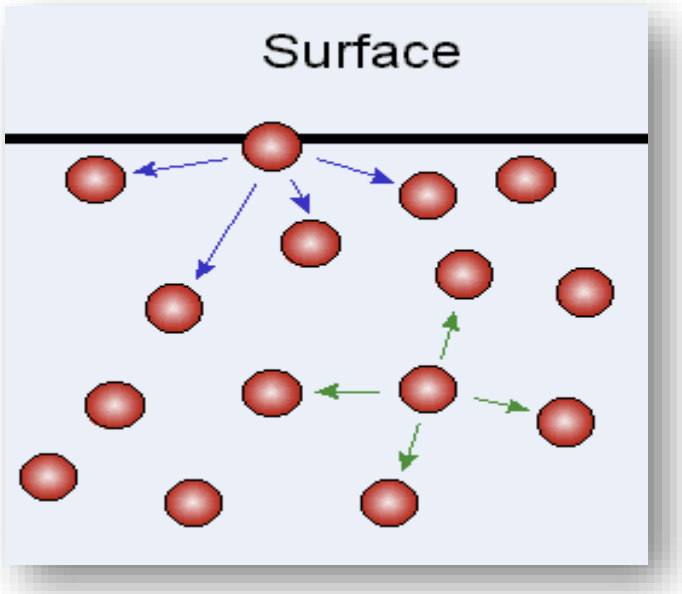
❖**Causes:**

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

❖**Types of Alveoli cell :**

- Type I Alveolar Cells (Pneumocytes):
  - These thin cells cover the majority (95%) of the alveolar surface and facilitate gas exchange.
- Type II Alveolar Cells (Pneumocytes):
  - These cells produce surfactant, smoking may reduce it levels and forms Adult RDS.





# THE PULMONARY SURFACTANT

❖ **Definition:**

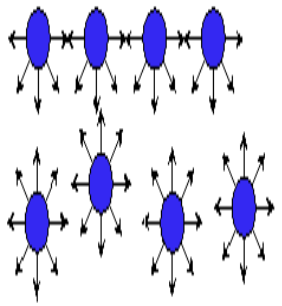
- It is a surface active agents secreted by type II alveolar epithelial cell to decrease the surface tension of fluid lining alveoli and antagonist lung collapse.

❖ **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).

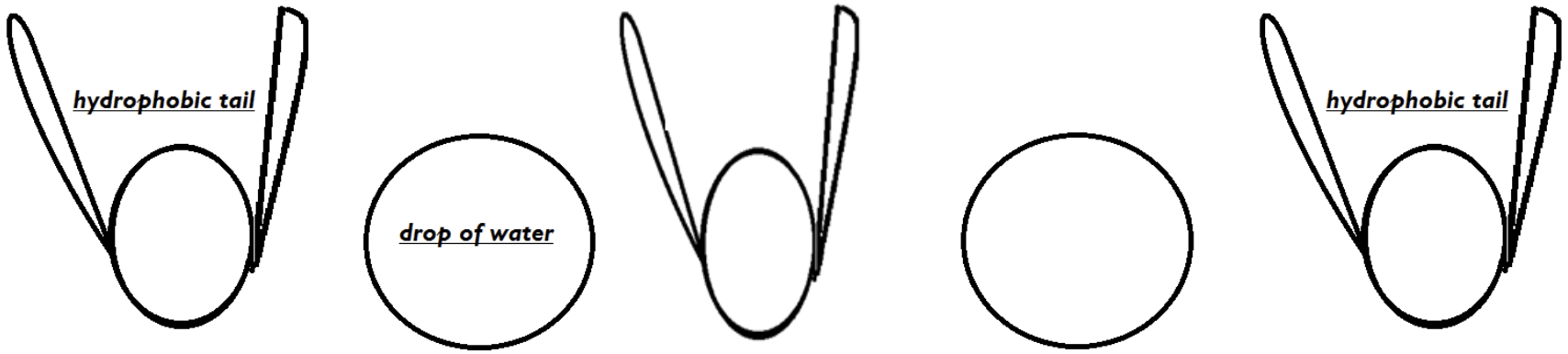
❖ **Composition:**

- **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):**

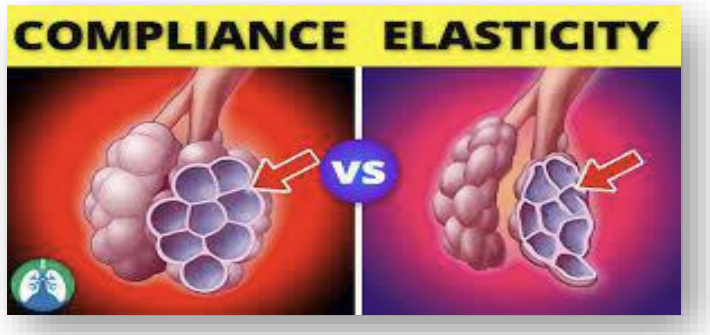
- **is common in:**
  A. Premature babies(↓Glucocorticoids secretion from adrenal cortex).
  B. Babies with diabetic mothers(those babies were subjected to fetal hyperinsulinemia, in which decrees glucose concentration in alveoli type 2 that suppress the surfactant formation).
- **Effect:** The surface tension is abnormally high; the work of breathing is markedly increased, alveoli are collapsed and death may occur.

# Give a reasons

1. **Respiratory distress syndrome is common in premature babies?**
   - Due to immature alveolar epithelium which fails tosecrete the surfactant because deficiency of glucocorticoid.
2. **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.
3. **Presence of fetal hyperinsulinemia in babes with diabetic mothers?**
   - Diabetic mother has hyperglycemia that causes fetal hyperinsulinemia due to presence of healthy fetal pancreas and easy diffusion of glucose by placenta.

# COMPLIANCE OF THE LUNGS

- **Definition:**
  - 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.
- **Complaince** $= \frac{\Delta V}{\Delta P}$ where $\Delta V$ is the change in volume, and $\Delta P$ is the change in pressure.
  - Increase in emphysema, higher surfactant concentration and aging.
  - Decrease in Pulmonary fibrosis.



# Respiratory muscles - Muscles of Inspiration

1. **Main Muscles:**
   - **A. Diaphragm:**
     - Supplied by the phrenic nerve(origin from C3 to C5).
       - Damage to phrenic in cases of hanging where there is injury above the C3 level, there is a risk of diaphragm paralysis, and death.
     - 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.
       - The ribs move upward and outward during contraction, this expansion increases the anteroposterior diameter of the thoracic cavity, also increases the lateral (side-to-side)diameter.
2. **Accessory Muscles:**(Act only in forced inspiration)
   - Sternomastoid(elevates the sternum).
   - Scaleri(elevates the 1st rib).
   - Seratus posterior superior .
   - Seratus posterior inferior(elevate the remaining ribs).

# Respiratory muscles - Muscles of Expiration

❖They act only in forced expiration

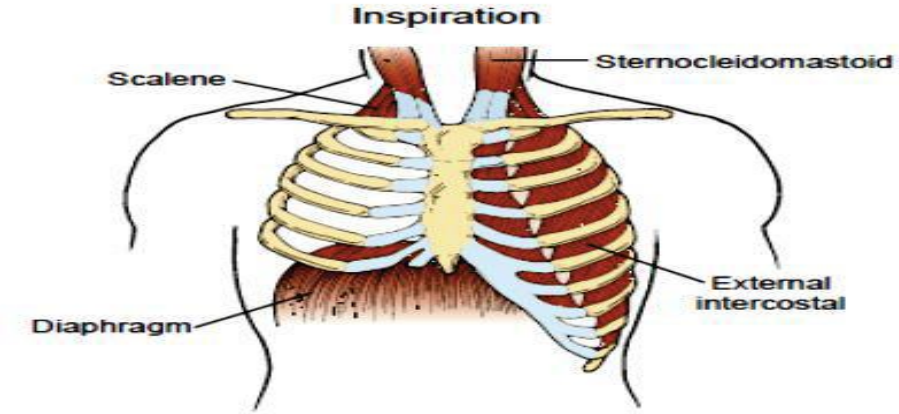
**A. Abdominal Wall Muscles:**

- 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.

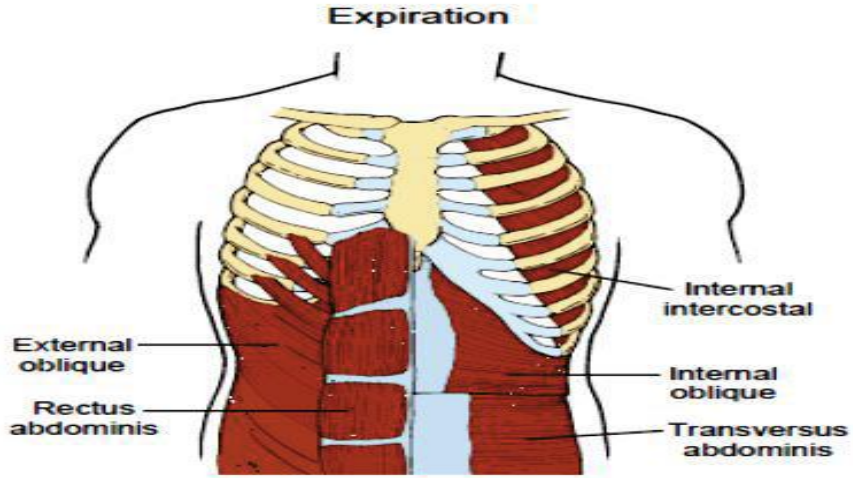
**B. Internal Intercostal Muscles:**

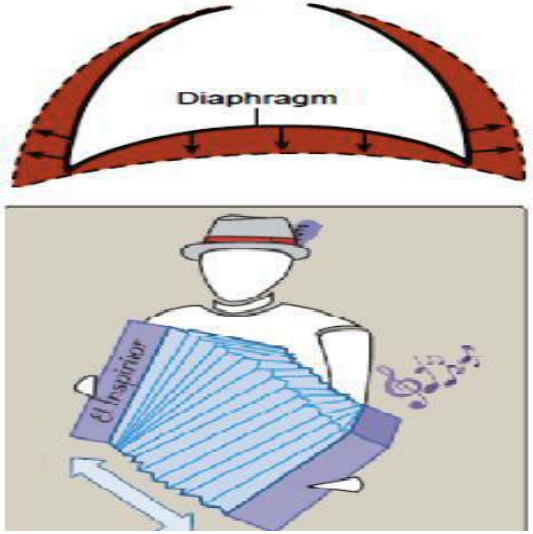
- Contraction of the internal intercostal muscles leads to depression and inversion of the ribs.

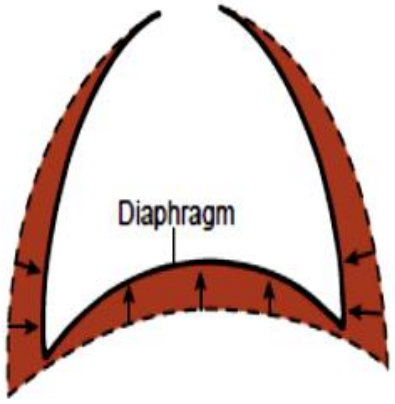
# RESPIRATORY MUSCLES




**D**iaphragm = **D**escend

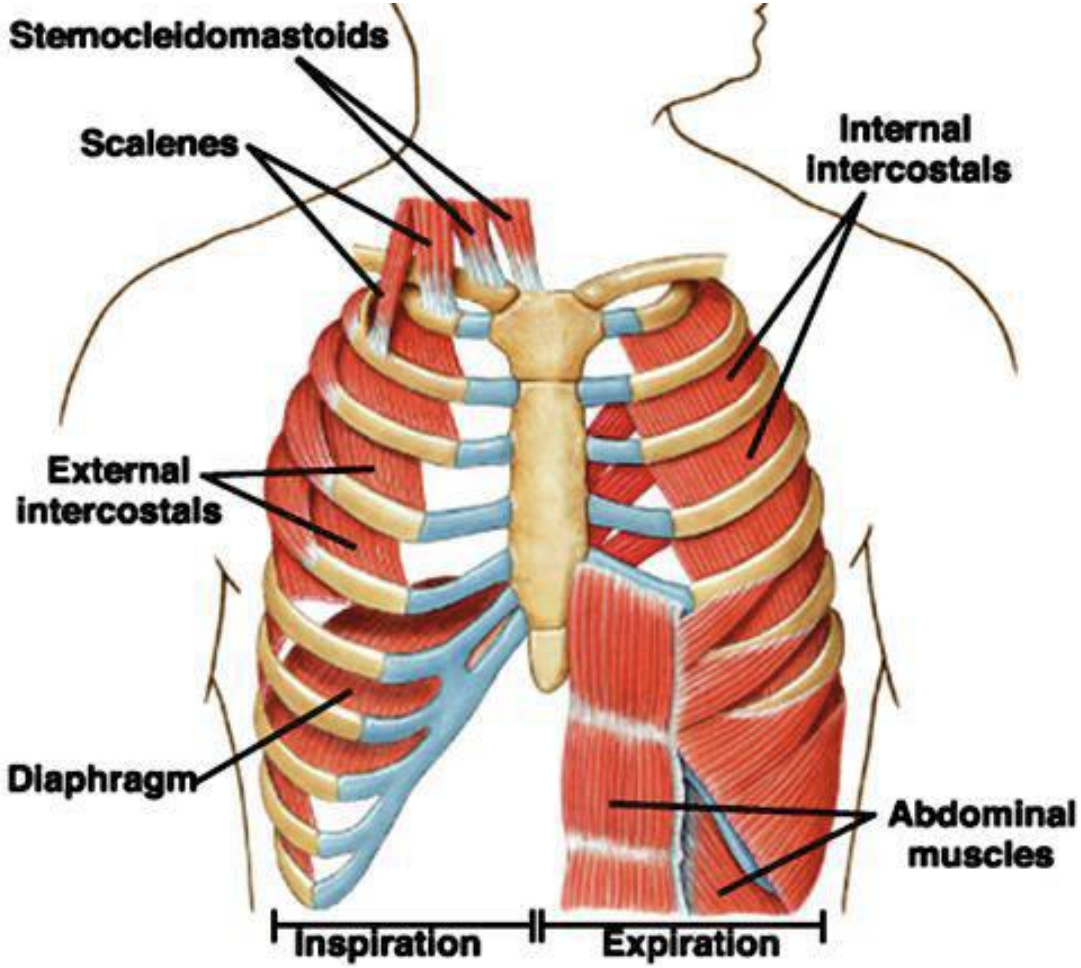


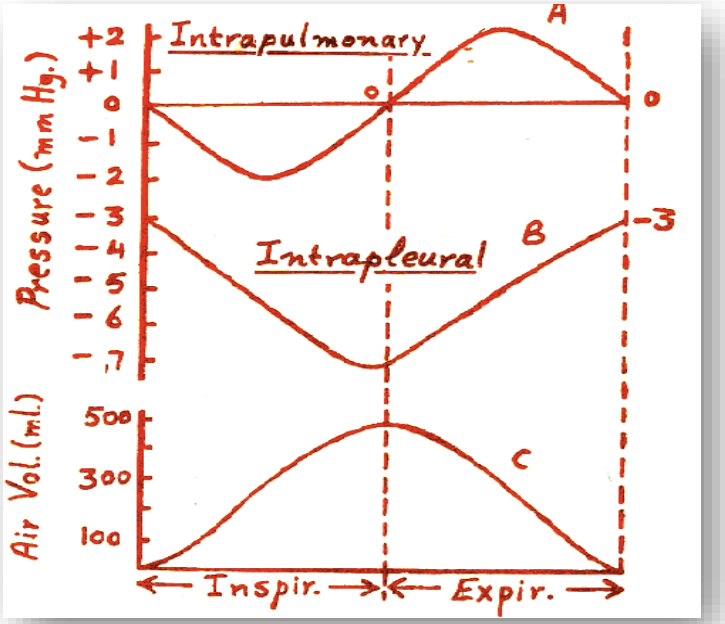

**E**xternal = **E**levation and **E**version

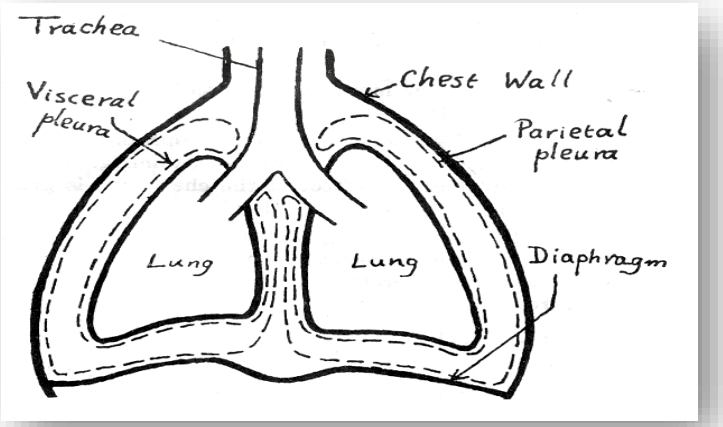
# RESPIRATORY MUSCLES



# Pulmonary Pressures

1. **Atmospheric pressure:**
   - Zero mmHg = 760 mmHg.
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(759mmHg) mmHg during inspiration and increases to +1(761mmHg) mmHg during expiration.

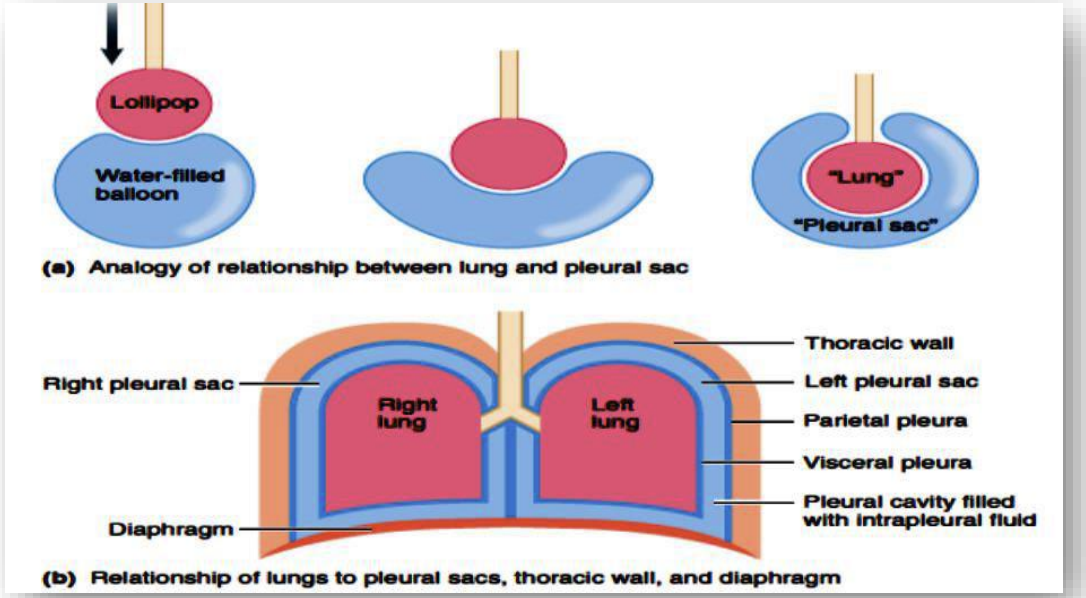


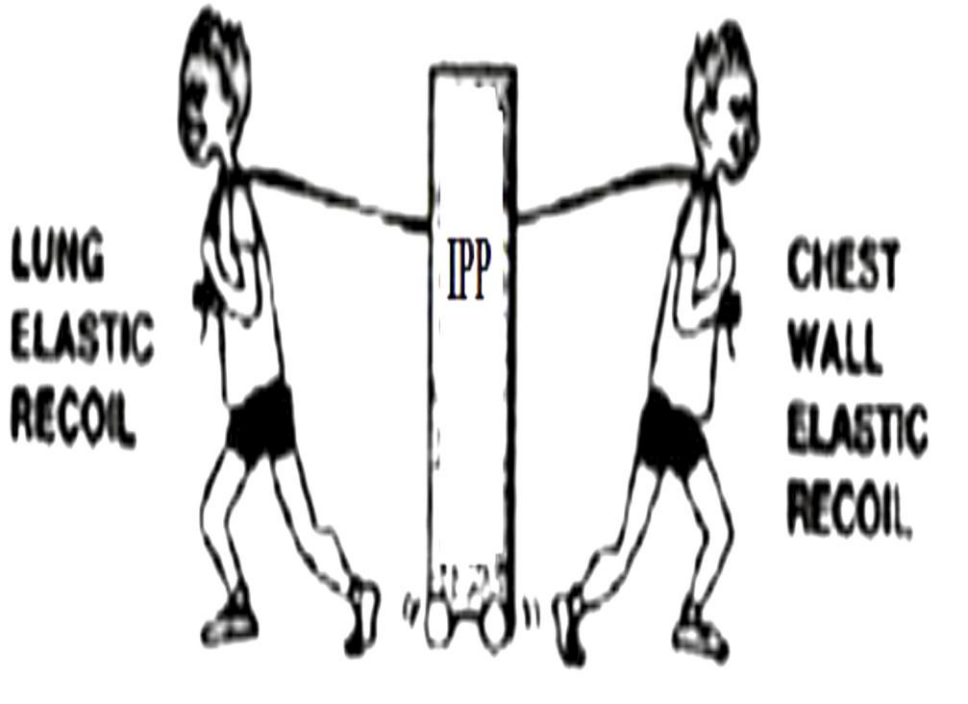
# INTRAPLEURAL PRESSURE(IPP)

❖**Definition:**

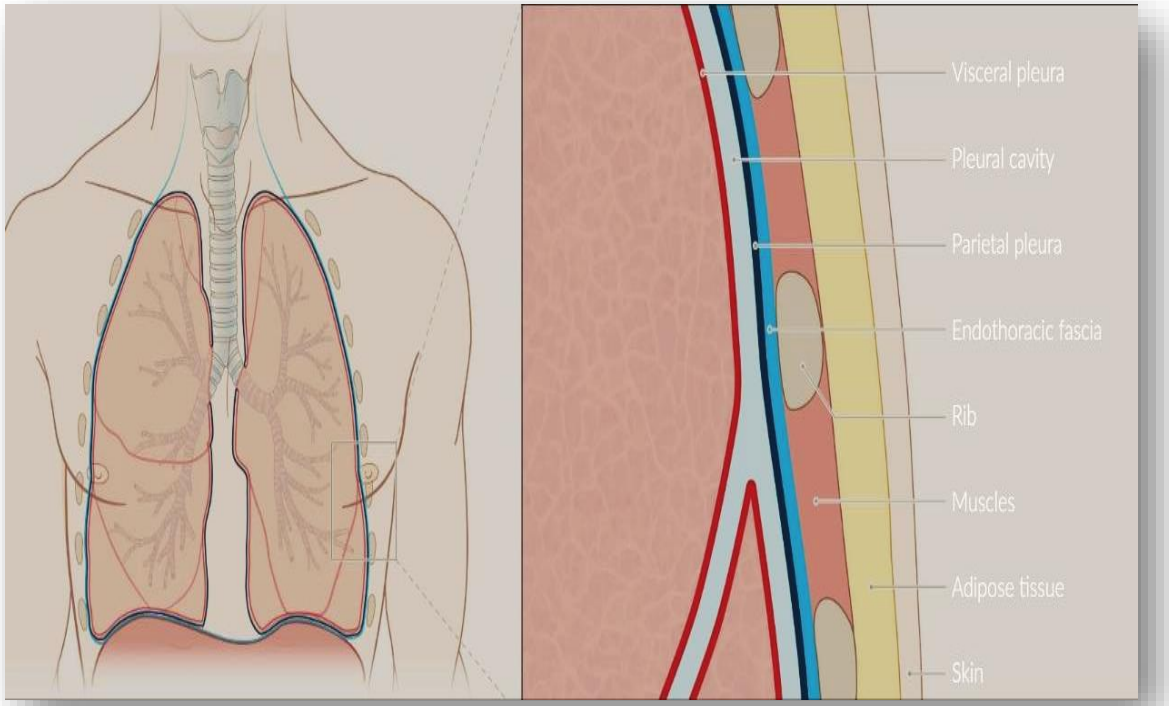
- It is the pressure of the fluid in the pleural cavity.
- It is always negative = Subatmospheric



# Causes of Negativity of IPP




Spiral elastic fiber



# Functions of IPP

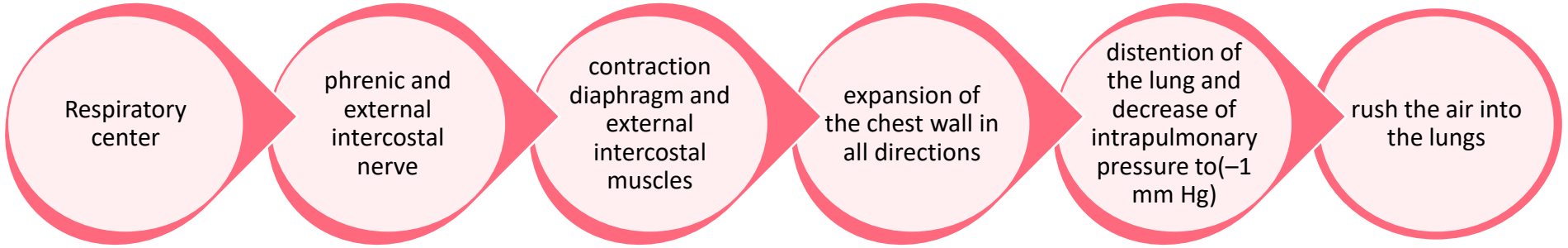
1. Maintains the lung inflated and prevents its collapse specially during expiration.
2. It helps the expansion of the lungs during inspiration.
3. It helps the venous return from extra-thoracic veins(+ve pressure)to intra-thoracic veins(–ve pressure).
   - **Valsalva Maneuver**: When people strain during bowel movements (as in constipation)especially in old age, they often perform a Valsalva maneuver, forcefully exhaling against a closed airway. This increases the intrathoracic pressure, which also increases (IPP) and decrease VR that may precipitate syncope 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:




## 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.

# MECHANISM OF BREATHING Cont.

**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)and 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 VS Expiration

| | *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)<br>• *I mean the intrapulmonary (intra-alveolar pressure)*<br>pressure decreasd to **-1 cmH2O**<br>*assuming that the atmospheric pressure is zero.* | decreased volume —> increased pressure (Boyle's law)<br>• *I mean the intrapulmonary (intra-alveolar pressure)*<br>pressure increasd to **+1 cmH2O**<br>*assuming that the atmospheric pressure is zero.* |
| Muscles | • Diaphragm: decends.<br>• external intercostals:<br>◦ —> elevate ribs —> increase transverse diameter.<br>◦ —> evert ribs —> increases AP diameter | — passive—<br><br>-diaphragm ascends, lungs shrink by their elastic recoil. |
| Accessory muscles for forced... | Forced inspiration:<br>• Sternocleidomastoid.<br>• serratous anterior<br>• scalene muscles | Forced expiration (voluntary "musical instuments", obstruvtive [COPD], restrictive [fibrosis]):<br>• internal intercostals<br>• abdominal muscles "abdominal recti" [abdominal breathing] |


MCQs
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# MCQs

1. **Which of the following does NOT cause a positive intrapleural pressure(IPP)?**
   a. Valsalva maneuver
   b. Hemothorax
   c. Tension pneumothorax
   d. Muller maneuver
2. **What is the main cause of the negativity of intrapleural pressure?**
   a. Pressure inside the alveoli during the respiratory cycle
   b. Dynamic harmonious antagonism between the chest wall and the lungs
   c. Different forces between the parietal and visceral pleura

# MCQs Cont.

3. **One of the following matched pairs is NOT TRUE?**
   a.Abdominal Muscles -Forced Expiration
   b.External intercostals and diaphragm- Quit Inspiration
   c.Sternocloidmastoid and scalenes- Quit Expiration
   d.Intrapleural pressure minus 7mmHg- Forced inspiration
   e.Intrapleural pressure minus 3mmHg-Forced Expiration
4. **To Increase the thoracic cavity and keep Intrapleural pressure negative, all the followings are correct EXCEPT?**
   a.Gravity
   b.Lymphatic drainage
   c.Elasticity of the chest wall
   d.Elasticity of the lung
   e.Surface tension

# MCQs Cont.

5. **A 25- year old pregnant female who is 24 weeks gestation presented to the labor and delivery ward. She has no underlying medical problems. She was 5 cm dilated and having continuous contractions.The newborn(700 gram)was delivered 10 hours later.Shortly after birth the neonate manifested with fast breathing, fast heart rate, chest contractions and blue discoloration of the skin.He was admitted to the neonatal ICU and placed on conventional ventilator, the condition is most probably due to?**

a.Inadequate alveolar collateral ventilation

b.Lack of oxygen due to insufficiency of type II pneumocytes

c.Immaturity of type II pneumocytes

d.Damage of type I pneumocytes by Covid-19 virus

e.The alveoli are loaded with dust cells

# MCQs Cont.

6. **Hereditary RDS due to SP-B deficiency, choose the wrong statement?**

   a.Infants with hereditary SP-B deficiency develop progressive respiratory failure that dose not respond to all treatment and death

   b.SP-B protein is encoded by a single gene on human chromosome 2

   c.The mutation causes complete absence of proSP-B but the presence of matureSP-B

   d.The common SP-B mutation is a net 2 base pair(bp)insertion into codon 121

   e.Hereditary SP-B deficiency is an autosomal recessive disorder

7. **Wrong about Blood gas barrier?**

   a.Thick

# MCQs Cont.

8. **According to the physiological and pathological factors that affect the negativity of intrapleural pressure, all are true except:**

a.Stab wound without valve(positive)

b.Emphysema(less negative)

c.Stab wound tension pneumothorax with valve(positive)

d.Valsalva's maneuver(positive)

e.At birth(zero)

# MCQs Cont.

**9. The surfactant proteins, choose the wrong statement of the following?**

a.SP-A and SP-C have overlapping surface film-promoting properties

b.SP-C bind bacterial lipopolysaccharides thus play a role in pulmonary host defense

c.SP-A and SP-D bind to pathogens causing its neutralization

d.SP-B is the only surfactant protein essential for life

e.SP-D and SP-A are hydrophilic protein