Over view of Respiratory System Physiology

by

Dr .Nour A. Mohammed

Associate professor of physiology

Faculty of Medicine, Mutah University

2024-2025

Respiratory System

- **Steps of respiration**

1) **Pulmonary ventilation**: between lung & atmosphere.

2) **Pulmonary perfusion**: it is the COP of right ventricle = 5 L/min.

3) **Exchange of gases**: between pulmonary ventilation & perfusion.

4) **Gas transport**: by the blood.

5) **Internal respiration**: by the tissues.

Respiratory passages

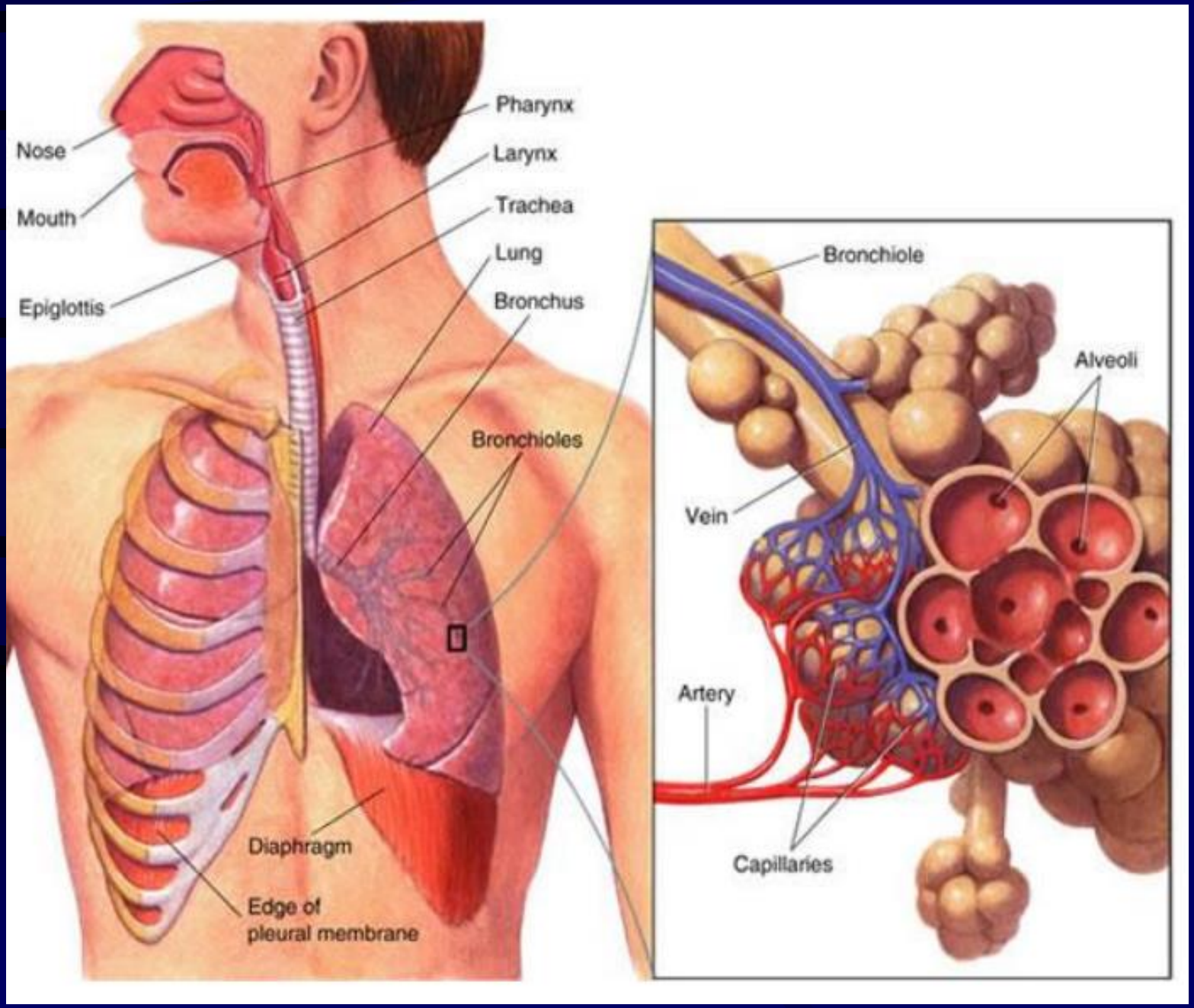
I. Air conducting zone (Dead space):

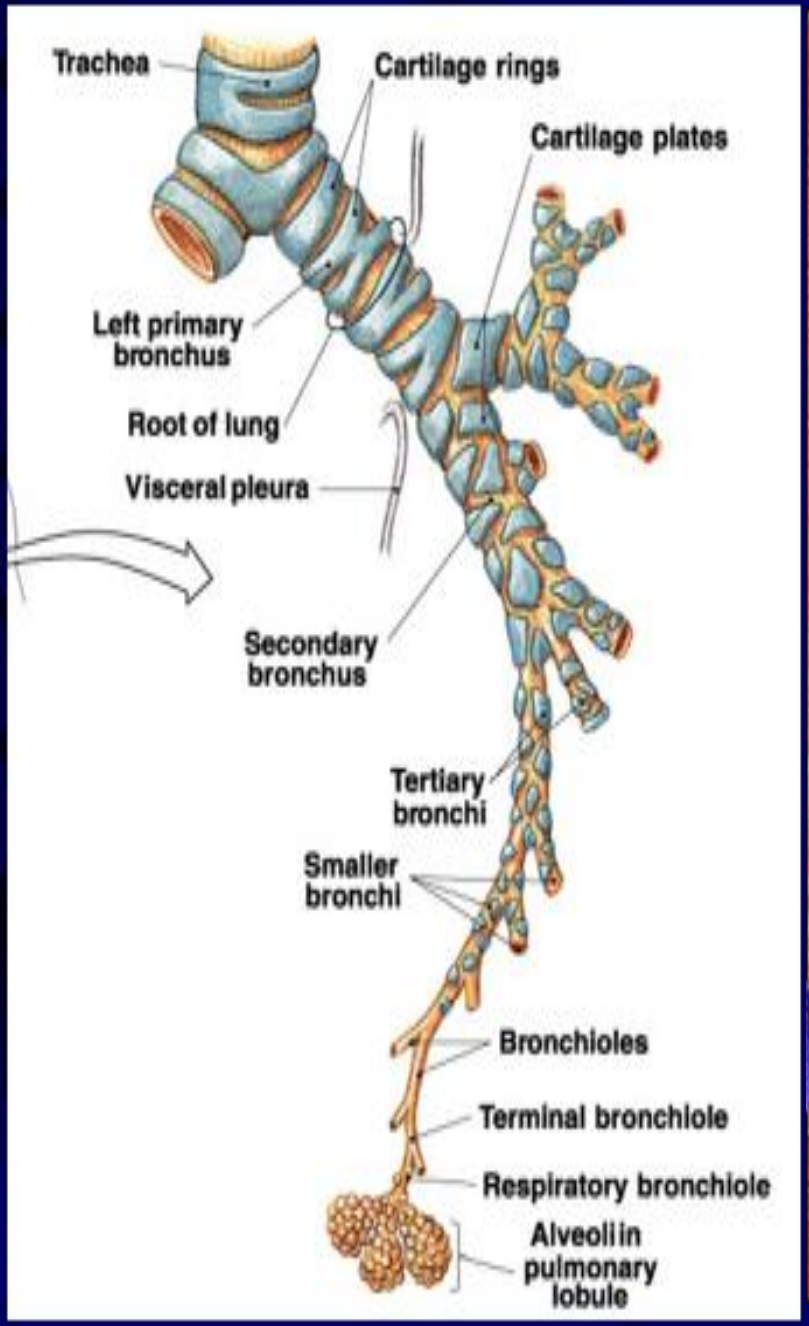
- Nose ð Pharynx ð larynx ð trachea ð bronchi ð bronchiole ð terminal bronchiole.
- No gas exchange due to thick wall.

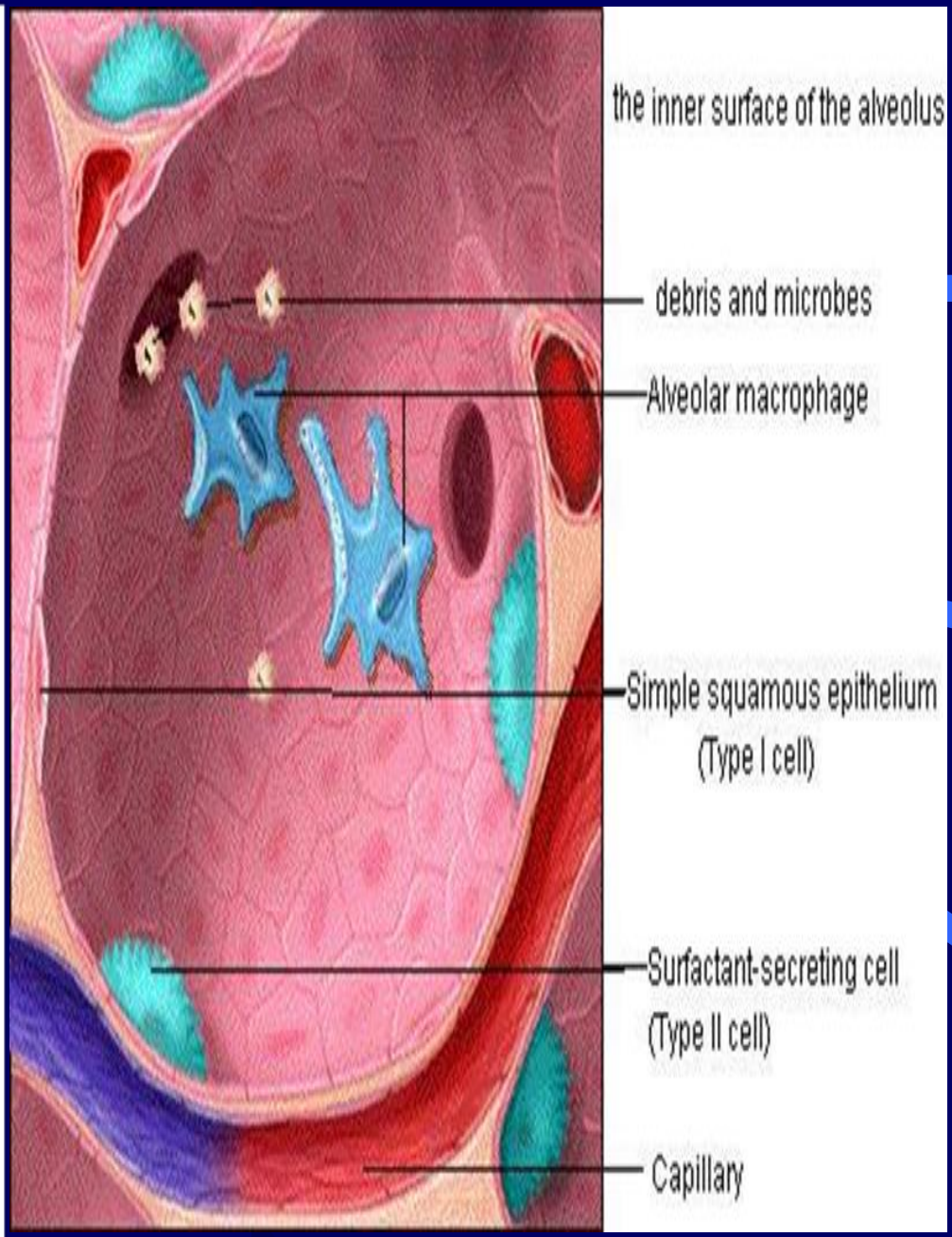
II. Respiratory zone (Exchange zone):

- Respiratory bronchioles ð alveolar duct ð alveoli. Gas exchange occurs with blood.
- Structure of alveoli: alveoli have 3 types of cells:

1) Type I cells (squamous pneumocyte).
2) Type II cells (granular pneumocyte) secrete surfactant.
3) Type III cells (alveolar macrophages or dust cell) phagocytic cells.


Nose
Mouth
Epiglottis
Pharynx
Larynx
Trachea
Lung
Bronchus
Bronchioles
Diaphragm
Edge of
pleural membrane
Bronchiole
Alveoli
Vein
Artery
Capillaries


Trachea
Cartilage rings
Cartilage plates
Left primary bronchus
Root of lung
Visceral pleura
Secondary bronchus
Tertiary bronchi
Smaller bronchi
Bronchioles
Terminal bronchiole
Respiratory bronchiole
Alveoli in pulmonary lobule


the inner surface of the alveolus
debris and microbes
Alveolar macrophage
Simple squamous epithelium
(Type I cell)
Surfactant-secreting cell
(Type II cell)
Capillary

Non respiratory functions of respiratory system

Smell: By olfactory receptors present in the posterior nasal cavity.

Taste: By oral cavity & pharynx.

Voice production: (phonation & articulation of speech)

- By changes in thickness & vibration & position of vocal cords in larynx.

Regulation of body temperature: By heat loss in expiration.

Regulation of pH (Acid-base balance)

- By controlling CO2 level.

Many drugs may be used by inhalation:

- As anesthetics & bronchodilators.

Protective functions

1) Air conditioning:

By warming & moistening of air due to rich blood supply & mucous so, prevent bad effect of cold air or dry air on the alveoli.

2) Protective reflexes:

- Irritation of nose causing sneezing reflex.
- Irritation of larynx, trachea or bronchi causing cough reflex.

3) Presence of Lymphoid tissue: in oro pharynx & naso pharynx.

4) Filtration of Large particles: (> 10 µ) by nasal hair.

5) Mucous blanket:

- It is produced by goblet cells - It prevents dust particles (less than 10 µ) to reach alveoli. - It contains immunoglobulin A

6) Muco-ciliary escalator mechanism:

- It is a wave of movement of cilia of respiratory mucosa, which drives mucus with particles to pharynx to be expelled.
- It is inhibited by: cigarette smoking, hypoxia, general anesthesia & dehydration.

7) Alveolar macrophages (Dust cells):

- They engulf dust particles (< 2 µ) and kill bacteria by its lysosomes.

Metabolic functions

1) **Synthesis of surfactant.**

2) **Release of** prostaglandins & interleukins & histamine & serotonin.

3) **Activation** of angiotensin I to form angiotensin II (important VC substance)by (ACE) convertase enzyme.

4) **Removal of some substances** as noradrenaline & serotonin.

5) **Contains fibrinolytic system** for lysis of any intra vascular thrombus. So, protect systemic circulation from emboli.

- **Respiratory cycle is composed of :**

Active inspiration then longer passive expiration then followed by pause.

- Respiratory rate during rest in adult male =

12 – 16 cycles/min.

- 500 ml of air inspired & expired per each cycle (tidal volume).

N.B: The expiratory **pause** is caused by:

1- Reflex stoppage of the activity of the inspiratory centers.

2- Time required for re-accumulation of Co2 after its wash by expiration to stimulate new inspiration.

Respiratory mechanics

Mechanism of inspiration

Active process under effect of inspiratory center.

Contraction of inspiratory muscles.

Normal inspiration:

a) Diaphragm ⇨ ⇧ vertical diameter.

(responsible for 75% of normal inspiration)

b) External intercostal ⇨ ⇧ transverse diameter.

Forced inspiration:

Sternomastoid & **S**erratus anterior
& **S**caleni & Elevator **S**capule
& Erector **S**pine.

⇧ Size of thoracic cavity.

Distention of the lung.

⇩ Intrapulmonary pressure. (-1 mmHg).

Air flow to inside the lung.

Mechanism of expiration

Passive process due to stoppage of the activity of the inspiratory center.

Normal expiration by:
Relaxation of inspiratory muscles.

Forced expiration by:
a) Internal intercostals.
b) Abdominal muscles.

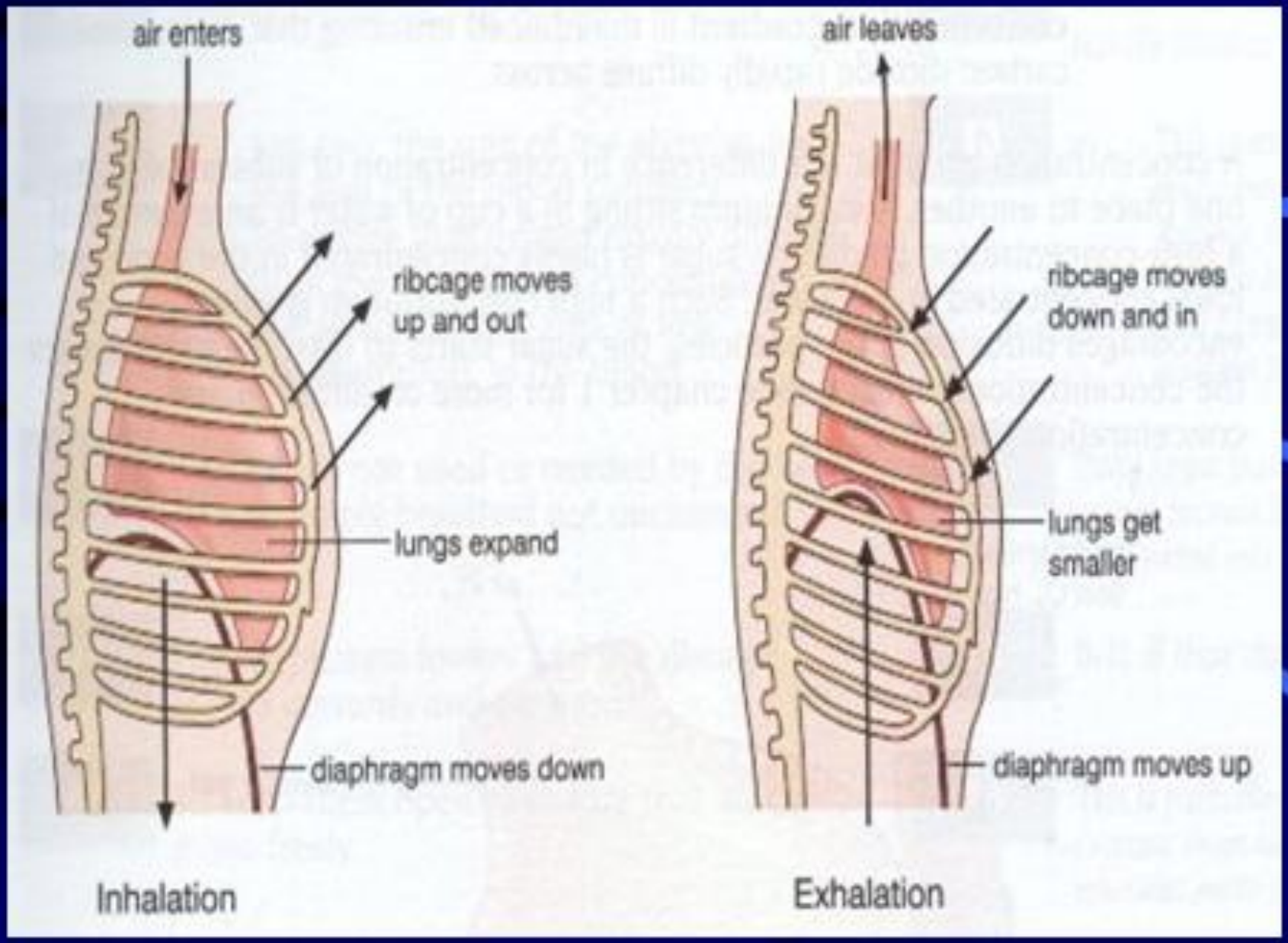
⇩ Size of thoracic cavity.

Recoil of the lung.

⇧ Intrapulmonary pressure. (+1 mmHg).

Air flow to outside the lung.

N.B: Expiration may be active in forced expiration


air enters
ribcage moves
up and out
lungs expand
diaphragm moves down
Inhalation
air leaves
ribcage moves
down and in
lungs get
smaller
diaphragm moves up
Exhalation

Respiratory pressures

1. Intra alveolar (Intra pulmonary) pressure:

Definition: It is the pressure inside the alveoli during respiratory cycle.

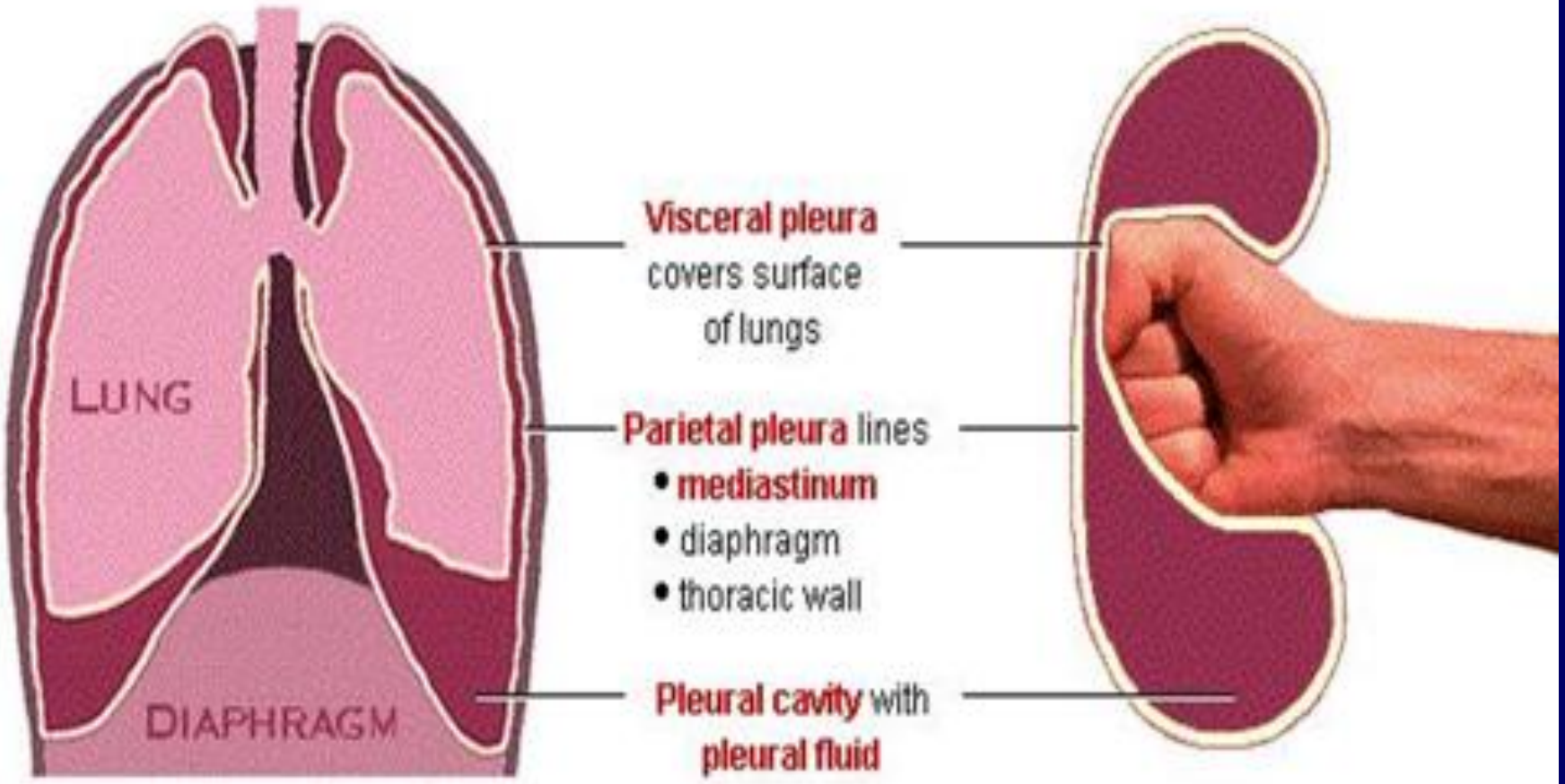
Value:	Inspiration	Expiration
Normal	- 1 mmHg	+1 mmHg
Forced	- 30 mmHg	+ 40 mmHg
Forced with closed glottis	- 80 mmHg (Muller maneuver)	+ 100 mmHg (Valsalva maneuver)
During respiratory pause = zero (atmospheric).		

Pleurae and the Lungs

Each lung is surrounded by two layers of **serous membrane** known as the **pleurae**.

The visceral and parietal pleurae are actually a continuation of the same membrane.

The relationship between the pleurae and the lungs can be demonstrated by pushing a fist into a water-filled balloon.



2. Intra pleural (intra thoracic) pressure (IPP):

- *Definition:* It is the pressure in the space between the two layers of pleura.
- *Value:*

	Inspiration	Expiration
Normal	- 6 mmHg	- 3 mmHg
Forced	- 12 mmHg	
Forced with closed glottis	- 30 mmHg (Muller maneuver)	+ 40 mmHg (Valsalva maneuver)

- ***Cause of negativity of IPP:***
  - It is due to **continuous tendency of the lung to recoil against continuous tendency of the chest wall to expand.** So, the two opposing forces cause negativity in pleural sac.

The recoil tendency of lung

- At end of normal expiration when respiratory muscles are relaxed the volume of lung and thorax = 2.5 liters, But the relaxation volume of the lungs = 1 liter.

So, the lung is distended from 1L to 2.5L and has tendency to recoil.

This **recoil tendency** is caused by:

1) Stretched elastic fibers of the lung (1/3 recoil tendency).

2) Surface tension of the fluid lining the alveoli (2/3 recoil tendency).

The expansion tendency of chest wall

At end of normal expiration when respiratory muscles are relaxed the volume of lung and thorax = 2.5 liters.

But the relaxation volume of chest = 5 liters So, the chest is compressed from 5L to 2.5L and has tendency to expand.

This **expansion tendency** is caused by:

Elasticity of muscles, tendons and tissue of chest.

Functions of IPP 1) It helps venous and lymph return against gravity.

2) It causes continuous **Expansion** of the lungs.

3) It is a measure of lung **Elasticity.**

e.g. when elasticity in emphysema decrease so, recoil decreases and negativity of IPP decreases as well (become more +ve).

Lung surfactant

- **Definition:** It is lipoprotein mixture containing phospholipid , dipalmitoyl lecithin.
- **Secreted from:** type II alveolar cells
- **Functions**:

1. **Decrease the Surface tension of the fluid lining the alveoli** Surfactant forms a layer between alveolar fluids & air inside alveoli So, prevent air – water interface. (make it air – surfactant interface). This leads to decrease surface tension from 20 to 3 mmHg.

2. Decrease muscular effort during inspiration Surfactant causes easy gradual expansion during inspiration.

Surfactant also prevents rapid expiration and collapse.

3. Safety factor against pulmonary edema

Surfactant decreases surface tension causing dry alveoli & prevents formation of pulmonary edema. (As increased surface tension in alveoli leads to filtration of fluid from blood)

4. Stabilization of alveolar size

It is Less concentrated in Large alveoli so, prevent their rupture, while it is more concentrated in small alveoli so, prevent their collapse.

Factors affecting surfactant formation

- Surfactant formation starts from **24th weeks** of intrauterine life.
- Surfactant formation completes at **35th weeks**.
- Surfactant formation needs **Cortisol** & **Thyroxin**

Hyaline membrane diseases (Infantile respiratory distress syndrome)

- Decrease surfactant formation in **newly born** leading to failure of lung expansion & alveolar collapse & pulmonary edema & respiratory failure then death.
- It occurs in premature babies due to (low cortisol & low thyroxin) & infant of diabetic mother due to (high insulin).
- **Diagnosis**: decreased ratio between **lecithin / sphingomylin** in amniotic fluid (< 1).
- Normally = 1 at 24 weeks & = 2 at 35 weeks of pregnancy.
- **Treatment:**

a) Artificial respiration and cortisone & thyroxin.

b) Artificial surfactant.


THank you