RESPIRATION

Dr.Abeer Shoman

objectives

ILOS:

1- Definition & general functions of respiratory system.

2- Functions of upper and lower air ways.

3- Mechanism of inspiration & expiration (respiratory cycle).

4- Changes in different respiratory pressures during respiratory cycle.

Respiration involves all the processes by means of which atmospheric oxygen (O2) is used by living cells for the oxidation of organic food substances and carbon dioxide (CO2) is removed from the body.

Functions of Respiratory System:

The Respiratory Function:

It means the conduction of O_2 from the atmosphere to the tissues and CO_2 from the tissues to the atmosphere *(**which is the main function of respiratory system**)*.

Respiration includes 3 main processes:

1- External respiration:

It means the conduction of O_2 from the atmosphere to the blood in the lungs and CO_2 from the blood to the atmosphere. **It consists of**:

- **Pulmonary ventilation**: which means air entry (i.e. inspiration) and exit (i.e. expiration) through the respiratory passages
- **Pulmonary perfusion***:* which means the blood flow through the pulmonary capillaries/minute. This is **equal** to the cardiac output.
- **Pulmonary gas exchange (by diffusion)** exchange of gases between venous blood in the pulmonary capillaries and air in the lung alveoli through the pulmonary membrane.
- **Blood transport of gases: (O_2 & CO_2),** will be discussed later.

2- Internal respiration:

It means capillary gas exchange between the capillary blood and the tissues. The cells take O_2 and give CO_2.

Non-respiratory Functions of Respiratory System:

1. Regulation of body temperature and water balance:

- Warming cold inspired air.
- Loss of water vapour in expired air: About 400 ml of metabolic water

2. Regulation of blood pH:

CO_2 gas is acidic. When it dissolves in water → carbonic acid (H_2CO_3)

- Hypoventilation → ↑ CO_2 in the blood → ↑ H_2CO_3 → acidosis.
- Hyperventilation → ↓ CO_2 in the blood (due to excessive loss from the lungs) → ↓ H_2C0_3 → alkalosis.

3. Vocalization:

Expired air moves the vocal cords for sound production during speaking.

4. Excretory function:

Excretion of volatile oils and toxic substances from the lungs through expired air e.g alcohol and ether (anaesthetic substance).

The Respiratory Apparatus

It is formed of:

1. The airways or passages.
2. The lungs.
3. The bony chest wall and the respiratory muscles.
4. The respiratory centres and the peripheral nerves supplying the respiratory muscles.

The Air Passages (i.e. air ways):

It means the air ways from the external nares to the alveoli. They are **divided into:**

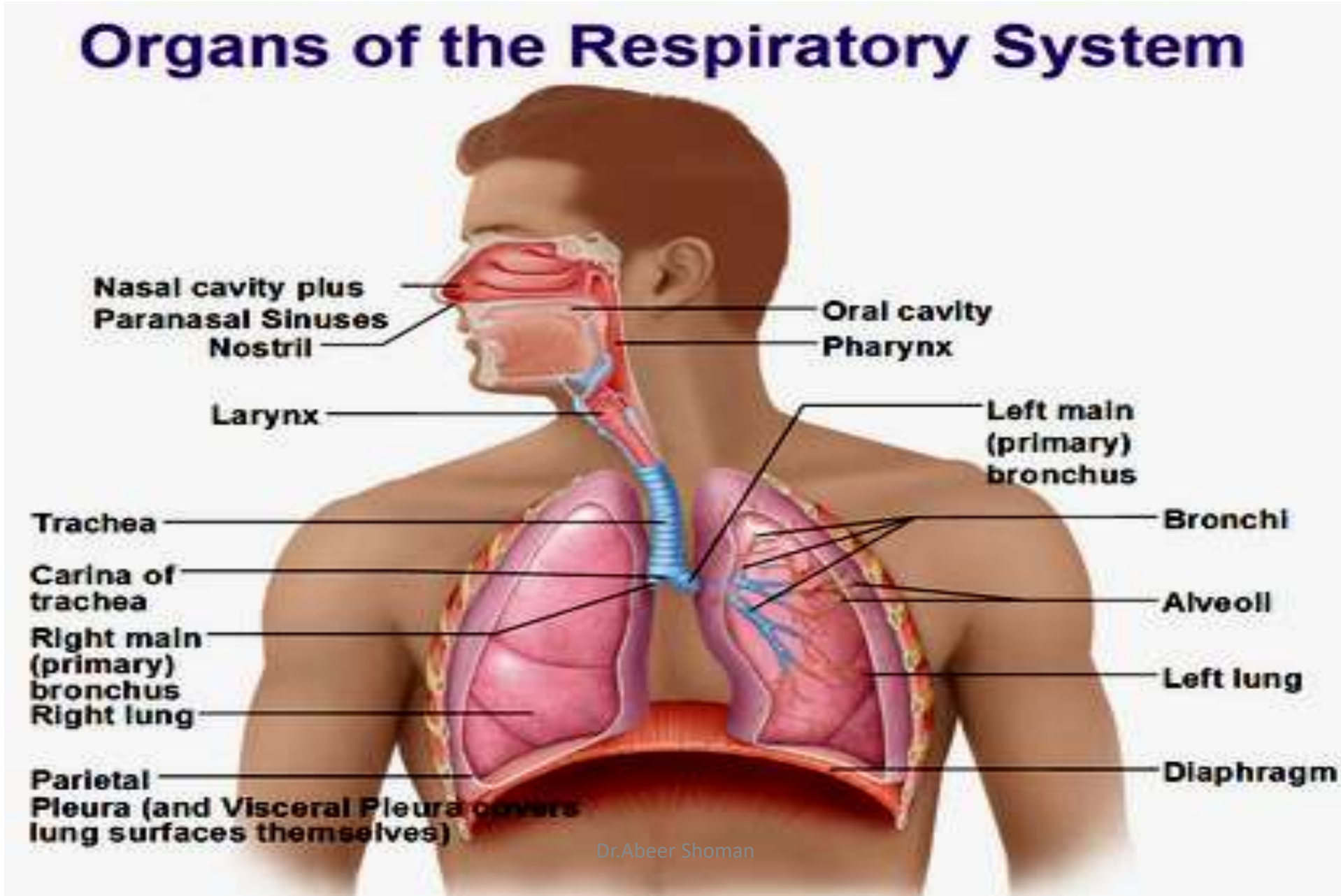
1] Conducting part (i.e. Upper airways):

- **Conduct** the inspired air **from** the atmosphere **to** the terminal bronchioles

It include; the nose → pharynx → larynx → trachea → bronchi → bronchioles → terminal bronchioles.

The wall is **thick**, **does not** allow any gas exchange between the air filling the lumen of these passages and blood in the capillaries of the wall.

Organs of the Respiratory System



Functions of the Conducting Part:

* **The nose:** smell sensation, warming cold air, protection through; trapping foreign particles
* **The larynx:** vocalization (i.e. voice production).
* **Trachea and bronchi:**

1. Air conditioning through: warming cold inspired air and humidifying dry air by contacting the mucosal capillaries.
2. Trapping foreign particles by mucus lining the wall.
3. Expelling foreign particles by :

    a) The movement of cilia of epithelial lining in an upward direction.

    b) The cough reflex.

2] The Respiratory unit (i.e. Lower airways):

- It includes: the respiratory bronchioles → alveolar sacs → alveoli.
- Its wall is thin allowing easy gas exchange and is lined with flat endothelial cells.

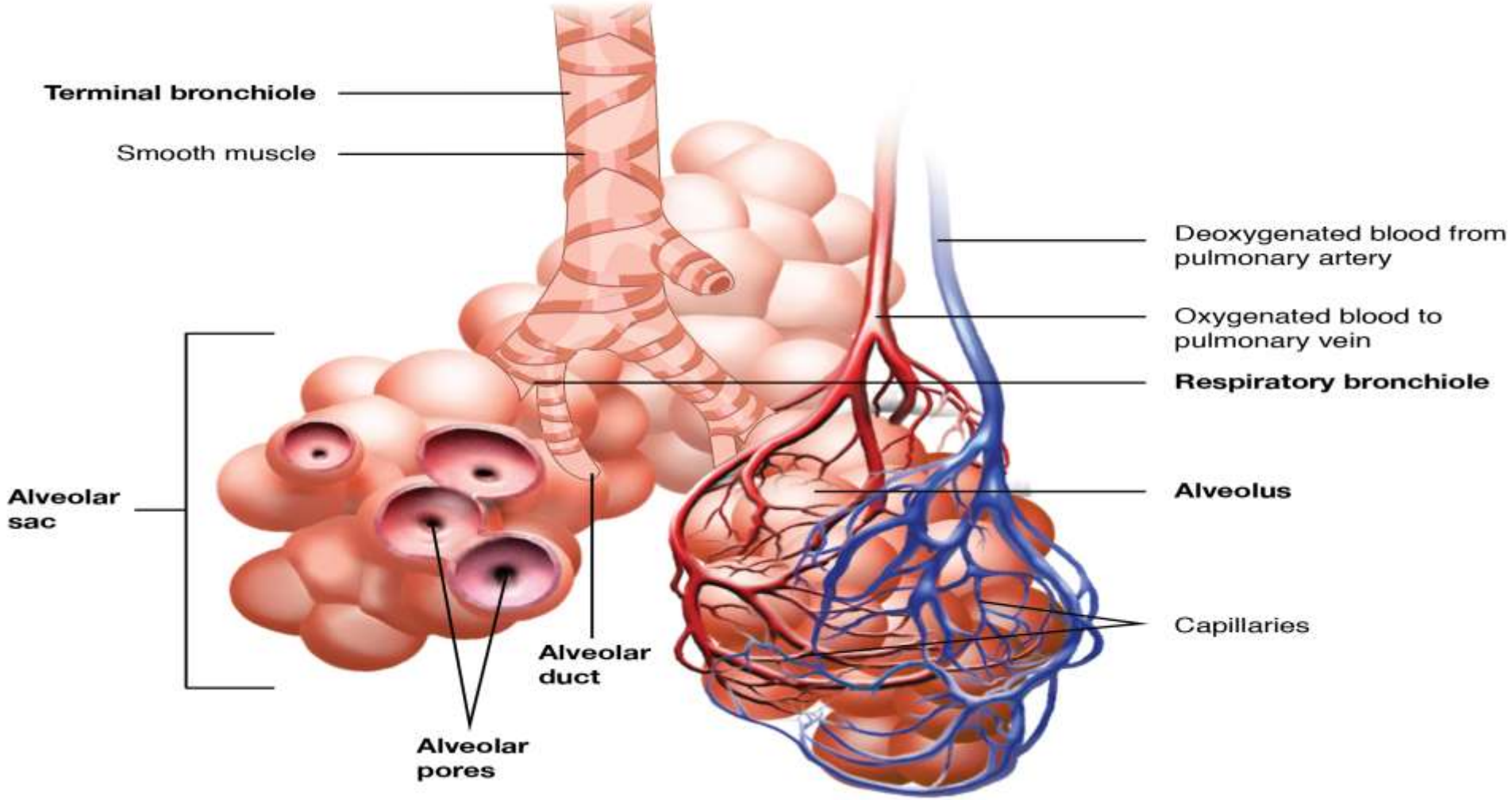
Functions:

1. Gas exchange.
2. Protection through engulfing large foreign particles by the pulmonary macrophages in its wall e.g dust cells.

N.B.

- **Bronchopulmonary segments:** are wedge-shaped areas within the lung (smaller than a lobe) formed of discrete anatomical and functional units supplied by an individual bronchus, artery, and vein.

Respiratory unit



Normal Breathing (Eupnea)

The Respiratory Cycle:

- The normal respiratory rate in adults is about 12 – 16 cycles/minute.
- **Each Respiratory Cycle consists of 3 phases:**
  1. Inspiration.
  2. Expiration (longer than inspiration).
  3. Expiratory pause; it **disappears** when respiratory rate in **increased** (i.e. tachypnea).
- **Respiratory rate is increased in the following conditions:**
  1. New borne infants (~ 40 cycles/ min.
  2. Physical activity (i.e. muscular exercise).
  3. Emotions or stress.

Mechanism of Respiration:

- A peripheral mechanism affecting the lungs and thoracic wall.
- A central mechanism through the activity of the respiratory centres.

Functional anatomy of the lungs and pleura:

- The two lungs are spongy like visceral organs that fill the thoracic cavity.
- Each lung is covered by a thin adherent serous membrane;

1) The visceral pleura which

2) The parietal pleura.

- The space between the visceral and the parietal pleura is a closed space called the pleural cavity , thin fluid film that acts as a lubricant between the visceral and the parietal pleura during the respiratory movements.
- The visceral pleura follows the movements of the lungs, while the parietal pleura moves with the chest wall.

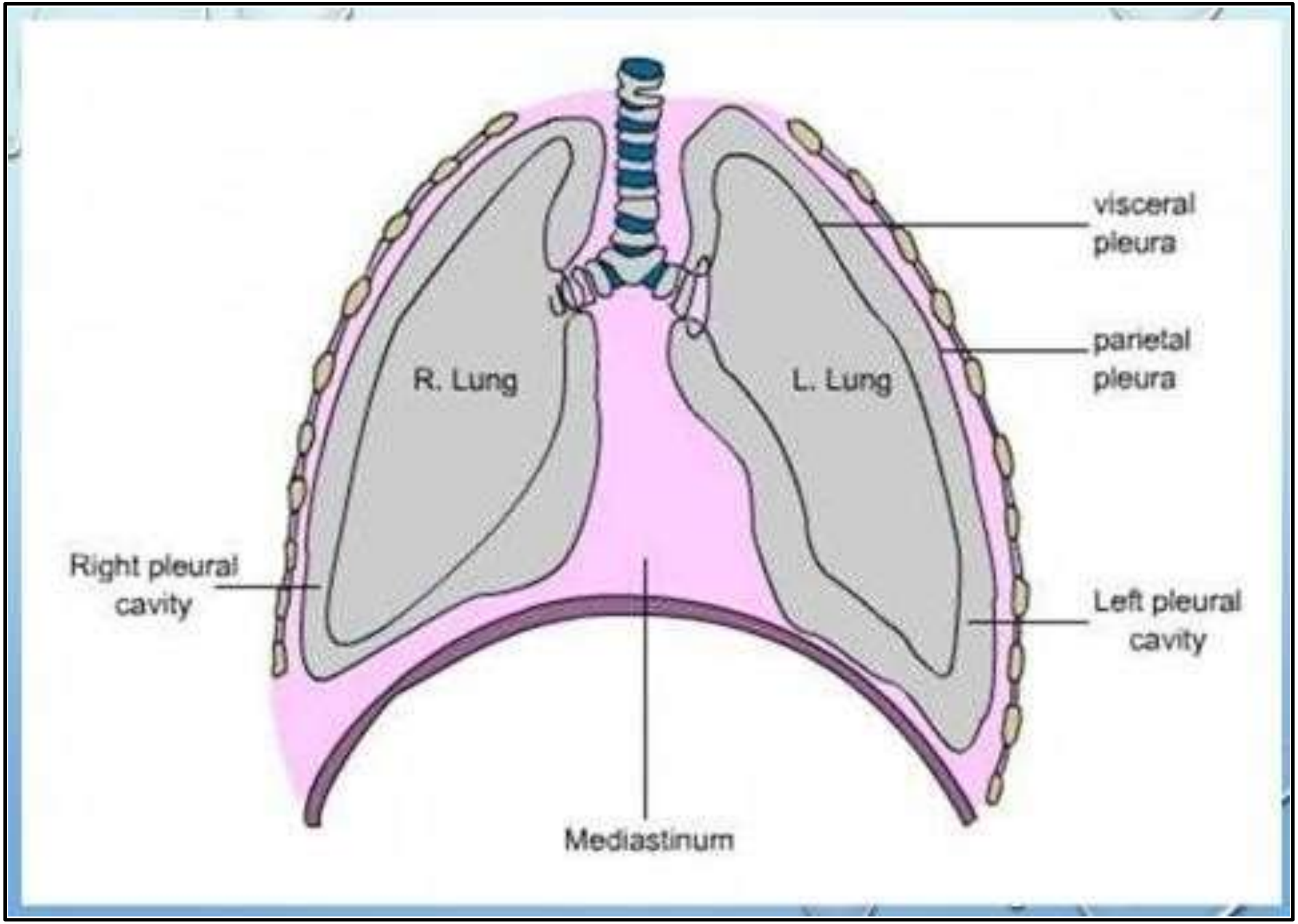
Development of intrapleural pressure (IPP):

At birth:

- The lungs nearly fill the chest cavity and no space is left between the visceral and the parietal pleura. The **intrapleural pressure** is nearly **zero** i.e. atmospheric.

After birth:

- The first forceful inspiration of life expands the chest wall more than the lungs.
- The rate of growth of chest wall is faster than that of the lungs creating a (–ve) pressure inside the pleural cavity.
- The negative IPP creates a **suction force** on the lungs → causing partial expansion of the lungs which try to recoil → expansion of pleural cavity → maintaining the –ve IPP around (-3 mmHg) under resting condition.
- **So, the main cause of the –ve IPP is** the mutual traction between the elastic recoil of the lungs **against** the rigid chest wall.
- **The net effect of this –ve IPP,** under normal resting condition is the **lungs are partially distended** and the **chest wall is partially collapsed**.


visceral pleura
parietal pleura
R. Lung
L. Lung
Right pleural cavity
Left pleural cavity
Mediastinum

Functions of the negative intrapleural pressure:

the IPP is the same as the intra thoracic pressure i.e negative or subatmospheric under normal condition. **This helps the following:**

(1) Venous return to the heart.

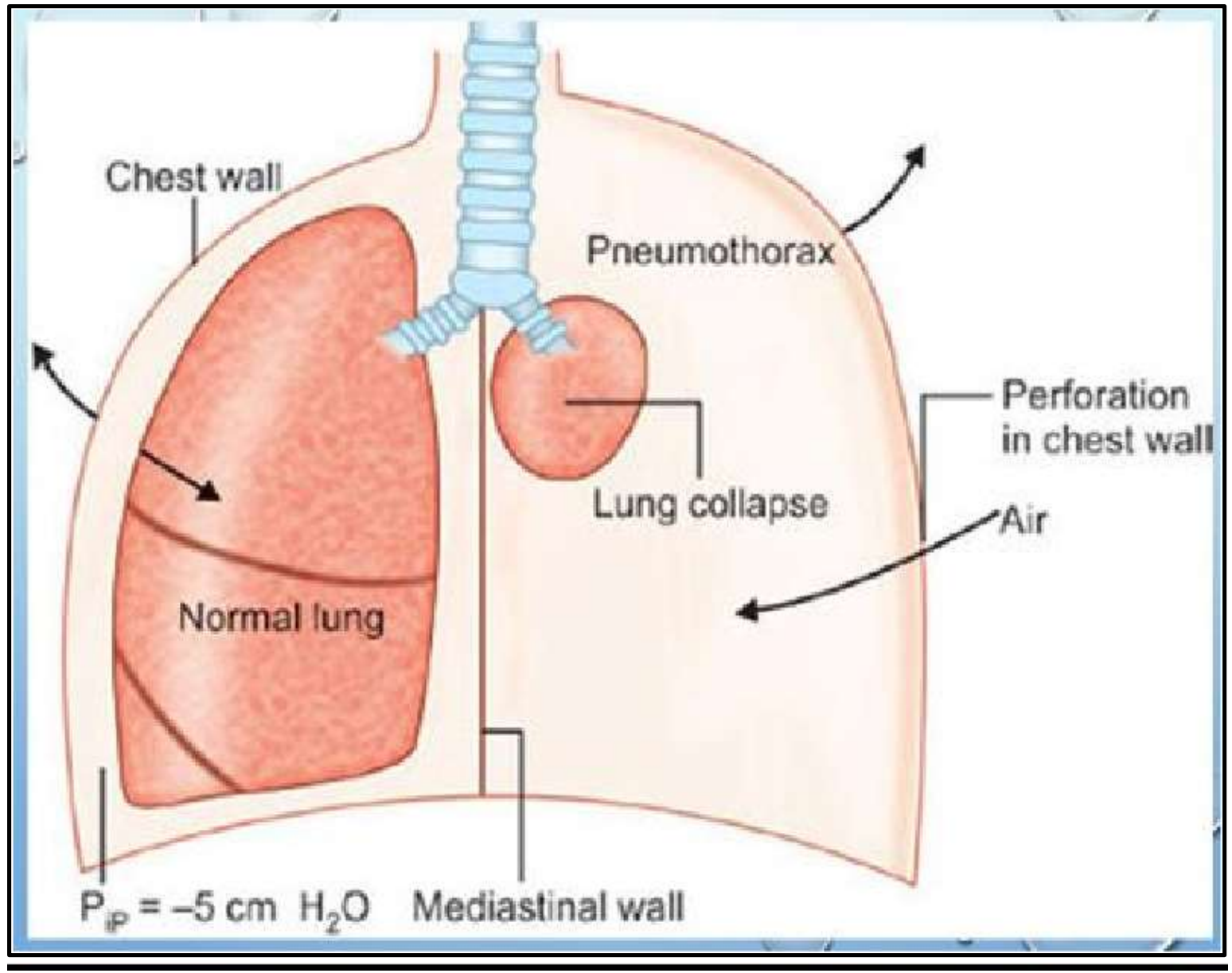
(2) Lymph flow through the thoracic duct to the venous system and finally to the heart.

(3) Expansion of the lungs during inspiration and acts as a brake during expiration

preventing sudden lung collapse so, expiration becomes gradual.

Clinical Significance:

In case of ↑ IPP (i.e. becomes less negative or atmospheric) **due to** ↓ lung Elasticity (as in **emphysema**) or air entry into the pleural cavity (as in **pneumothorax**) → ↓ suction force of IPP → ↓ traction on chest wall and lungs → complete collapse of the lungs and expansion of chest wall (i.e. **Barrel chest**).


Chest wall
Pneumothorax
Perforation
in chest wall
Lung collapse
Air
Normal lung
P_{IP} = –5 cm H_2O
Mediastinal wall

The Peripheral Mechanism of Respiration:

I. Inspiration:

Inspiration is active (i.e. produced by contraction of the inspiratory muscles) which are:

a)The Diaphragm;

- It is a skeletal muscle supplied by the phrenic nerve and is responsible for 75% of inspiration.
- When it contracts → ↑ the longitudinal diameter of the chest cavity.
- Paralysis of the diaphragm is not fatal and can be compensated by excessive work of the intercostal muscles if healthy.

b)The External Intercostal Muscles:

- Its fibers run downwards and forwards from the lower border of a rib above to the upper border of a rib below.
- They are supplied by the intercostal spinal nerves.

When they contract they elevate the ribs → ↑ both the anteroposterior and transverse diameters of chest cavity (Bucket handle mechanism).

Mechanism of Normal Inspiration:

1.Contraction of inspiratory muscles → ↑ all diameters of chest cavity (anteroposterior, transverse, and longitudinal) → expansion of chest wall → → ↓ IPP (i.e. becomes more negative; from - 3 mmHg at the beginning of inspiration to - 6 mmHg at its end.

2.Increased negativity of IPP causes → more suction on the lungs → distension of the lungs → ↑ volume of lung alveoli without air entry → ↓ pressure inside lung alveoli (i.e. ↓ intra-alveolar pressure from atmospheric pressure (zero) at the beginning of inspiration to - 2 mm Hg at its end).

3.Decreased intra-alveolar pressure causes → rush of air (about 500 ml inspired air) from the atmosphere to the distended alveoli (lower pressure) → the alveolar pressure increases again to the atmospheric pressure at the end of inspiration.

Mechanism of Forced Inspiration:

Requires the contraction of Accessory Muscles of Inspiration including;

- The sternomastoid, the levator scapulae, the scalene muscles, the serratus anterior, and the

serratus posterior.

- They contract during forced inspiration to produce more elevation of ribs and more expansion of chest cavity. They **do not work** under **normal resting respiration** (eupnea).

I. Expiration:

Expiration is **passive** (i.e. no muscular contraction necessary for normal expiration).

Mechanism of Normal Expiration:

1. Relaxation of inspiratory muscles and depression of chest wall → ↓ all diameters of chest cavity.
2. Recoil of the distended lungs.
3. Both (1) and (2) → squeeze the alveoli → ↑ the alveolar pressure 2 mmHg above the atmospheric pressure → expulsion of equal volume of air (500 ml expired air) during normal expiration.

N.B.

The contracting inspiratory muscles **relax** gradually to act as a **brake** preventing sudden expiration.

Mechanism of Forced Expiration:

Forced expiration is **active** due to contraction of expiratory muscles which are:

a) **The Abdominal Wall Muscles:**

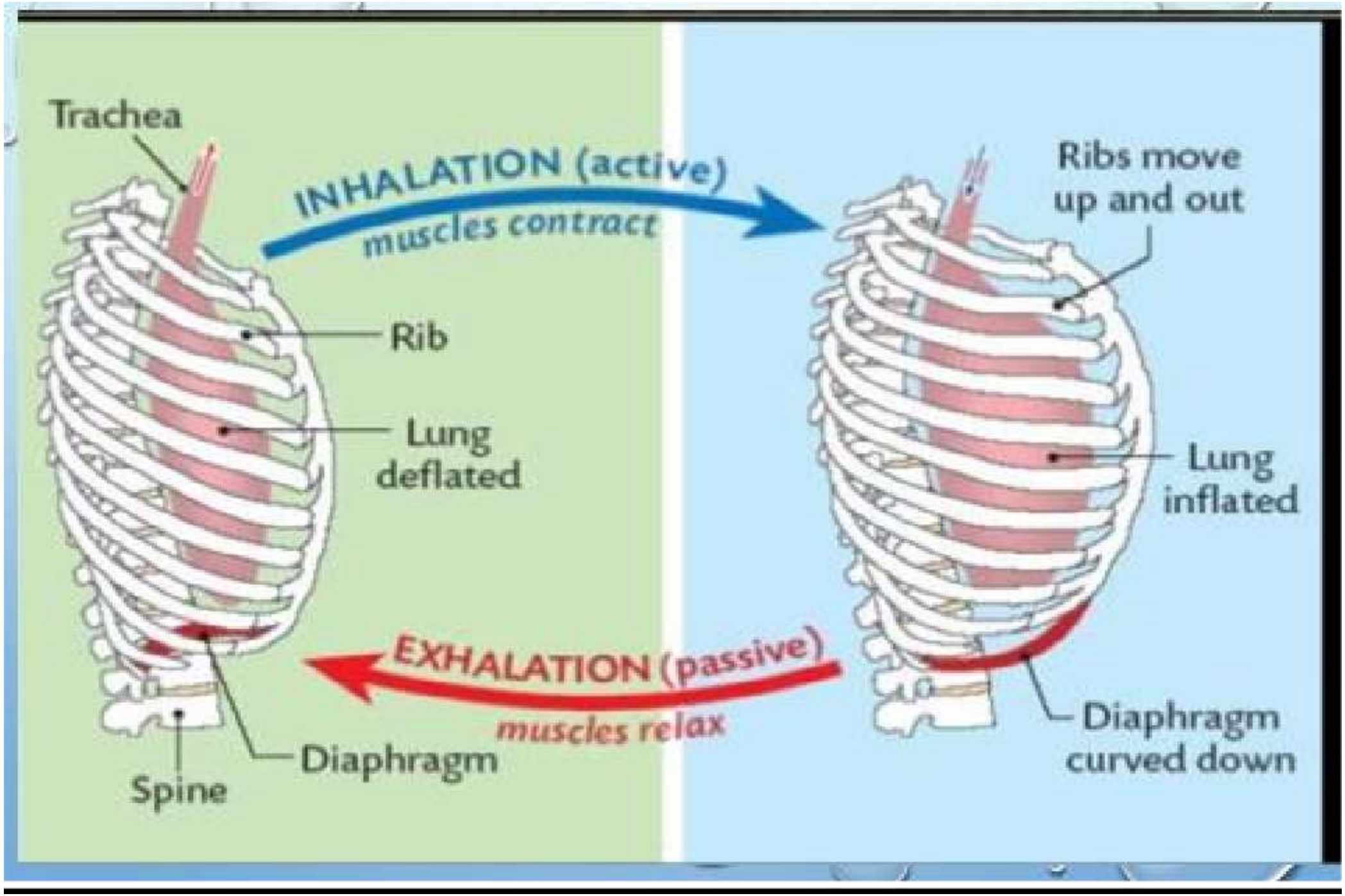
Contraction of the abdominal wall muscles → ↑ intraabdominal pressure → more elevation of the diaphragm and depression of ribs to squeeze the lungs → forceful expiration.

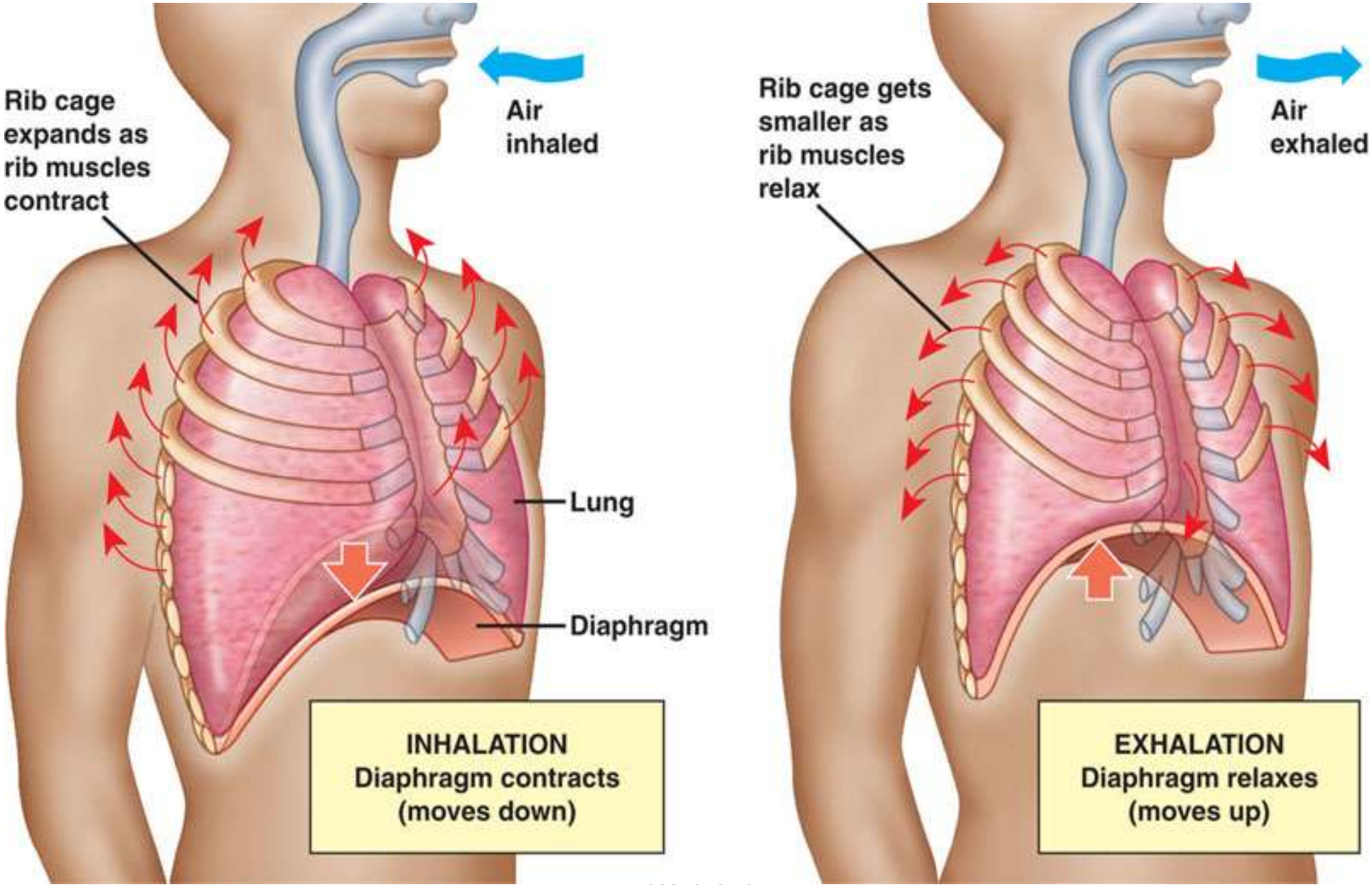
a) **The internal intercostal muscle:**

- Its fibers run from the upper border of a rib below upward and forwards to the lower border of a rib above.
- Supplied by intercostal spinal nerves.
- Its contraction → depression of ribs → forceful collapse of chest cavity → forced expiration.

Forceful expiration occurs in:

1. Hyperventilation as during muscular exercise.
2. Air way obstruction as in bronchial asthma.
3. During coughing and sneezing reflexes to get rid of the foreign particles.


Trachea
INHALATION (active)
muscles contract
Ribs move
up and out
Rib
Lung
deflated
Lung
inflated
EXHALATION (passive)
muscles relax
Diaphragm
Spine
Diaphragm
curved down


Rib cage expands as rib muscles contract
Air inhaled
Lung
Diaphragm
INHALATION
Diaphragm contracts
(moves down)
Rib cage gets smaller as rib muscles relax
Air exhaled
EXHALATION
Diaphragm relaxes
(moves up)

Pressure Changes during Respiratory Cycle

Changes in the intrapleural (or pleural) pressure (IPP):

- **In the midthoracic position** (i.e. at the end of normal expiration), the IPP is **negative** (i.e. subatmospheric) and is about **(-3 mmHg)**. This is **due to** the mutual traction between the elastic recoil of the lung against the rigid chest wall (as mentioned before).
- **At the end of normal inspiration (eupnea)** → the IPP decreases to **(-6 mmHg)** **due to** expansion of the chest and pleural cavities, however, (expansion of chest is **greater than** distension of the lungs creating **more negativity** in pleural cavity).
- **At the end of normal expiration (midthoracic position)** → the IPP increases again to normal resting level **(-3 mmHg)** **due to** collapse of chest cavity.
- **During forced expiration with closed glottis** (i.e. Valsalva manoeuvre) → the IPP increases up to about **(+40 mmHg)**.
- **Forced inspiration with the glottis closed** → more negative IPP reaching about **(-30 mmHg)**.

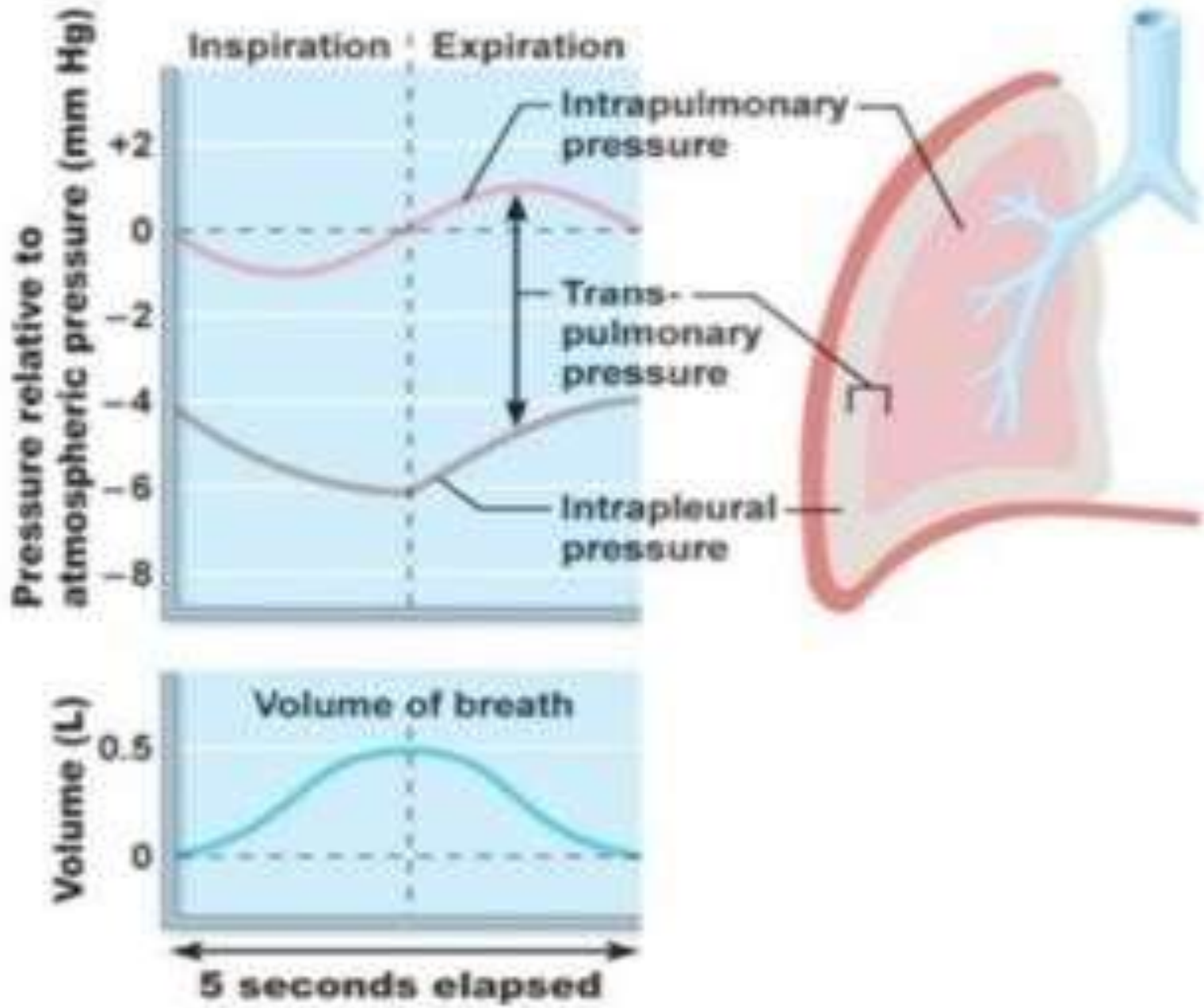
Changes in the intra-alveolar (or alveolar) pressure:

- In midthoracic position (i.e. at the end of normal expiration), it is **atmospheric** (i.e. **zero**).
- At the beginning of inspiration → it decreases to **-2 mmHg due to** expansion of the lungs before air entry. Then, it becomes **atmospheric** again at the end of inspiration when inspired air (500 ml) fills the distended alveoli.
- At the beginning of expiration → it increases to **+2 mmHg due to** elastic recoil of the lungs before expulsion of air. Then, it becomes **atmospheric** (zero) again at the end of expiration due to expulsion of expired air (500 ml).

Intrapulmonary pressure. Pressure inside lung decreases as lung volume increases during inspiration; pressure increases during expiration.

Intrapleural pressure. Pleural cavity pressure becomes more negative as chest wall expands during inspiration. Returns to initial value as chest wall recoils.

Volume of breath. During each breath, the pressure gradients move 0.5 liter of air into and out of the lungs.




Thank
you!