Mechanism of Respiration

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PROGRAMME OUTCOMES

- PO1- Demonstrate comprehensive knowledge and application of the Trisutra concept to explore root causes, identify clinical manifestations of disease to treat ailments and maintain healthy status.
- PO2- Demonstrate knowledge and skills in Ayurveda, acquired through integration of multidisciplinary perspectives and keen observation of clinical and practical experiences.

COURSE OUTCOMES

- CO1- Explain all basic principles & concepts of Kriya Sharir along with essentials of contemporary human physiology and biochemistry related to all organ systems.

- **Teaching learning methods-** lecture with power point presentation
  Domain- Cognitive/comprehension
  Must to know / desirable to know / Nice to know- Must to know
  Millers pyramid- Knows how(applied knowledge)
  Bloom taxonomy- Understand

Mechanism of Respiration

- Ventilation

  The exchange of air between the lungs and the atmosphere so that oxygen can be exchanged for carbon dioxide in the alveoli (the tiny air sacs in the lungs).

- Eupnoea

  Rhythmic breathing at rest. It consists of Inspiration and Expiration.

- INSPIRATION

- An active process
- Size of thoracic cavity is increased by appropriate muscles
- The parietal pleura follows the expanding chest wall
- The visceral pleura also follow the parietal pleura and thus the elastic lungs also follow the thoracic expansion passively.
- The expansion of lungs is associated with fall in pressure in the lung parenchyma and atmospheric air is thereby drawn into the depth of lungs

- EXPIRATION

- A passive process
- At the end of the inspiration, muscles relax and the elastic recoil of the thoracic wall and lungs cause passive expiration

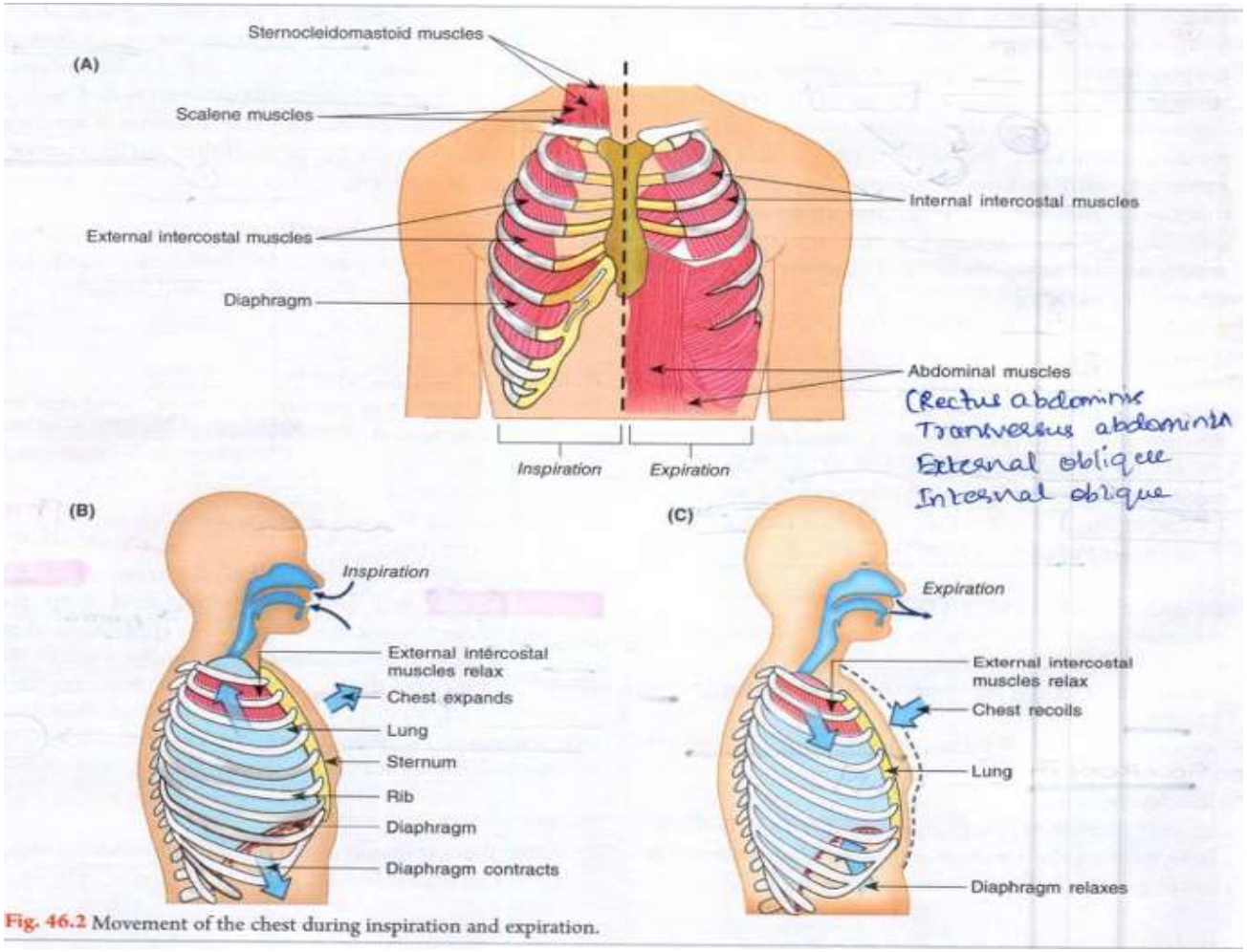


Fig. 46.2 Movement of the chest during inspiration and expiration.

Mechanism of Inspiration

It is an active process. During inspiration the thorax is enlarged by-

- Movements of ribs outwards and upwards(Rib movements)
- Descent of diaphragm(Diaphragmatic movements)

RIB MOVEMENTS

a) In quite respiration, the first pair of ribs moves but little. In hyperpnoea their movements bring about increase in AP diameter of upper thorax.

b) The 2^{nd} to 6^{th} ribs slope obliquely downwards and forward from their joints with the spinal column.

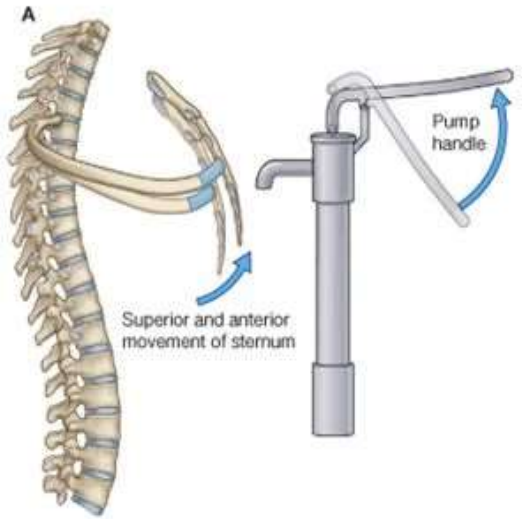
On inspiration the ribs move upwards(pump handle movements) to assume a more horizontal position due to contraction of external intercoastal muscles(T1 & T2) to increase in AP diameter of the chest. Moreover by virtue of their curved(bowed) mid part, cause an increase in transverse diameter of the thorax.

RESPIRATORY MOVEMENTS
B- MOVEMENTS OF RIBS

PUMP HANDLE MOVEMENT

Elevation of ribs

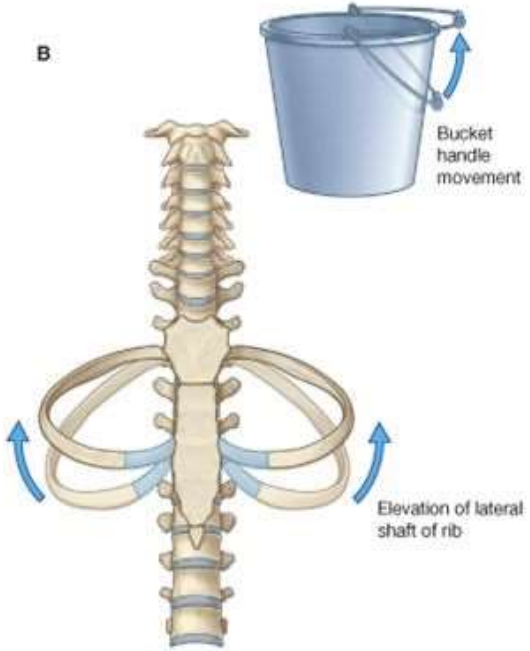
Increase in antero-posterior diameter of thoracic cavity



BUCKET HANDLE MOVEMENT

Elevation of ribs

Increase in lateral diameter of thoracic cavity



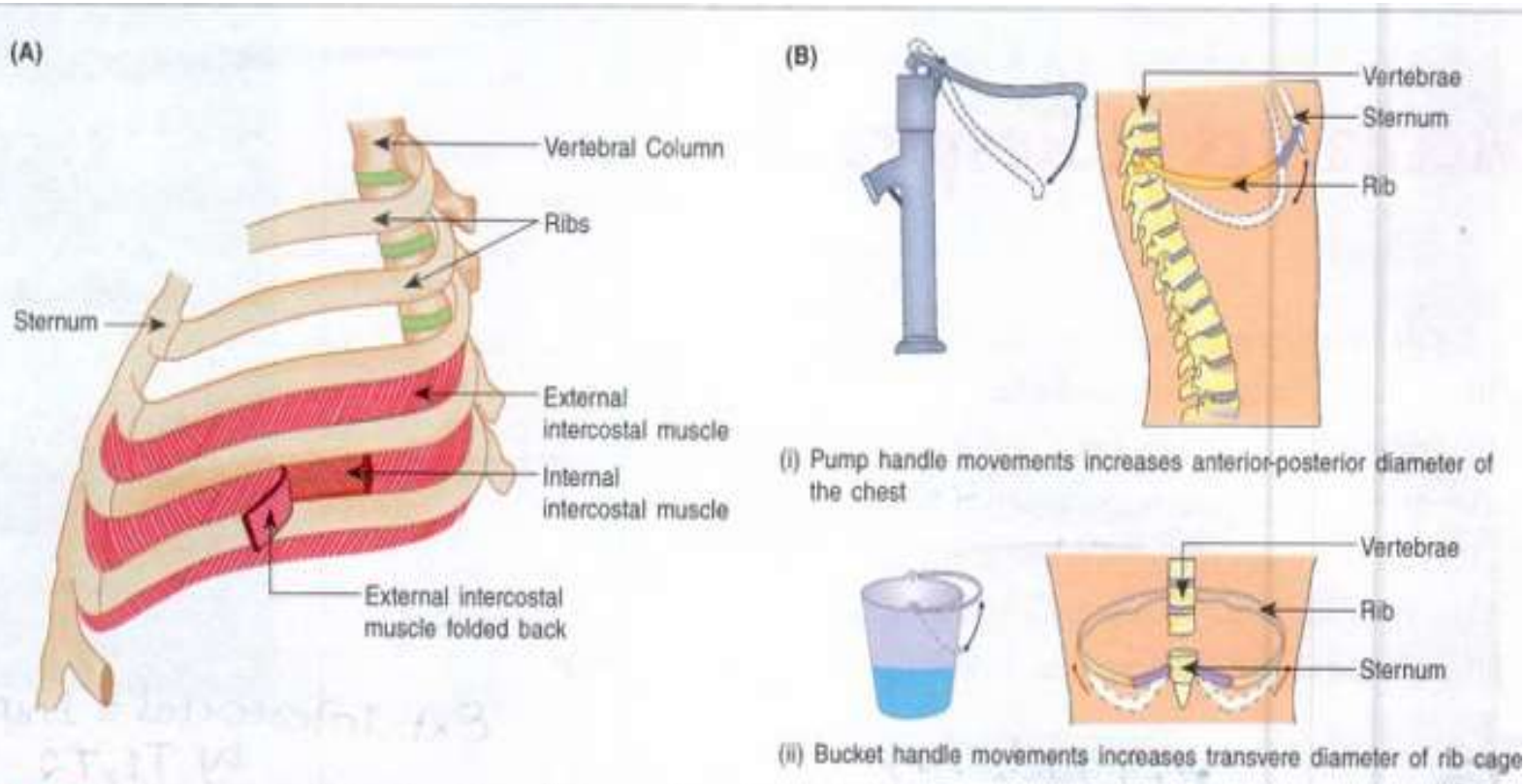


Fig. 46.1 (A) Arrangement of external and internal intercostal muscles on ribs. (B) Rib movements during inspiration

c) The lower ribs(7^{th} to 10^{th}) also swing outwards and upwards(bucket handle movement)in inspiration to cause increase in the transverse diameter of the thorax

DIAPHRAGMATIC MOVEMENTS

a) Diaphragm consists of muscle fibres which converge on the dome shaped central tendinous portion. Muscle fibres arise from the-

1. Xiphisternum
2. Inner surface of the lower ribs
3. Lumbar vertebrae

b) During inspiration as a result of discharge in phrenic nerves(C3,C4,C5), muscle fibres contract and draw the central tendon downwards by 1.5cm in eupnoea and by 7cm in deep inspiration. This causes an increase in the vertical diameter of the thoracic cage

c) For each 1 cm descent, 200-300ml of air sucked in, therefore these movements account for as much as 75% of tidal volume(approx.500ml)

Clinical Significance of inspiratory movements

- Either the diaphragm or external intercoastal muscles alone can maintain adequate ventilation at rest.
- In pts with B/L phrenic nerve palsy but intact intercoastal muscles innervation, respiration is somewhat laboured but adequate to maintain life.
- Transection of spinal cord above C3 spinal segment is fatal without artificial respiration but transection below C5 is not because it leaves the phrenic nerve(C3,C4,C5), that innervates diaphragm, intact.

Position of diaphragm and postural relationship

- Sitting- most comfortable posture because diaphragm is down due to gravity and not pressing over abdominal contents. This also increases the reserve volume of lungs.
- Standing- Abdominal muscles contract to cause intra abdominal pressure to increase, resulting in ascent of diaphragm.
- Lying- the most uncomfortable posture.

Accessory muscles of inspiration

- Scalene and sternocleidomastoid muscles play little role in breathing at rest, but become active in voluntary static effort and help to elevate thoracic cage during deep inspiration.
- Intrinsic muscles of larynx i.e. abductor muscles of vocal cords, contracts early in inspiratory phase pulling the vocal cords apart and opening the glottis. It is supplied by recurrent laryngeal nerve, a branch of vagus nerve. Their paralysis leads to inspiratory stridor i.e. inspiration with a loud sound

Mechanism of Expiration

- In quite breathing, expiration is the passive process, but during forced expiration for e.g voluntary expiratory efforts, during exercise, bronchial asthma etc. The muscles of expiration contract, which include:

a) Anterior abdominal muscles(abdominal recti, transverse abdominis, internal and external oblique muscles)

b) Internal intercoastal muscles

- Contraction of ant. Abdominal wall muscles increases intra abdominal pressure and draws the lower ribs down and medially, thereby pushing the diaphragm upwards, thus aiding in expiration.
- Internal intercoastal muscles pass obliquely downwards and posteriorly from rib to rib, on contraction they pull the upper ribs down so that the ribs acquire the original position

- **RESPIRATORY PRESSURES**

Two types of pressures are exerted in the thoracic cavity and lungs during process of respiration:

1. Intrapleural pressure or intrathoracic pressure
2. Intraalveolar pressure or intrapulmonary pressure.

- **INTRAPLEURAL PRESSURE**

Definition

Intrapleural pressure is the pressure existing in pleural cavity, that is, in between the visceral and parietal layers of pleura. It is exerted by the suction of the fluid that lines the pleural cavity. It is also called intrathoracic pressure since it is exerted in the whole of thoracic cavity.

- **Normal Values**

  Respiratory pressures are always expressed in relation to atmospheric pressure, which is 760 mm Hg. Under physiological conditions, the intrapleural pressure is always negative.

  Normal values are:

1. At the end of normal inspiration:

   –6 mm Hg (760 – 6 = 754 mm Hg)

2. At the end of normal expiration:

   –2 mm Hg (760 – 2 = 758 mm Hg)

3. At the end of forced inspiration:

   –30 mm Hg

4. At the end of forced inspiration with closed glottis

   (Müller maneuver): –70 mm Hg

5. At the end of forced expiration with closed glottis (Valsalva maneuver): +50 mm Hg.

Cause for Negativity of Intrapleural Pressure

- Pleural cavity is always lined by a thin layer of fluid that is secreted by the visceral layer of pleura. This fluid is constantly pumped from the pleural cavity into the lymphatic vessels. Pumping of fluid creates the negative pressure in the pleural cavity.
- Intrapleural pressure becomes positive in Valsalva maneuver and in some pathological conditions such as pneumothorax, hydrothorax, hemothorax and pyothorax.

Significance of Intrapleural Pressure

Throughout the respiratory cycle intrapleural pressure remains lower than intra-alveolar pressure. This keeps the lungs always inflated.

Intrapleural pressure has two important functions:

1. It prevents the collapsing tendency of lungs

2. Because of the negative pressure in thoracic region, larger veins and venacava are enlarged, i.e. dilated. Also, the negative pressure acts like suction pump and pulls the venous blood from lower part of body towards the heart against gravity. Thus, the intrapleural pressure is responsible for venous return. So, it is called the **respiratory pump for venous return**

- **INTRA-ALVEOLAR PRESSURE**

Definition

Intraalveolar pressure is the pressure existing in the alveoli of the lungs. It is also known as **intrapulmonary pressure.**

Normal Values

Normally, intraalveolar pressure is equal to the atmospheric pressure, which is 760 mm Hg. It becomes negative during inspiration and positive during expiration

- Normal values are:

  1. During normal inspiration:

  –1 mm Hg (760 – 1 = 759 mm Hg)

  2. During normal expiration:

  +1 mm Hg (760 + 1 = 761 mm Hg)

  3. At the end of inspiration and expiration: Equal to atmospheric pressure (760 mm Hg)

  4. During forced inspiration with closed glottis (Müller maneuver): –80 mm Hg

  5. During forced expiration with closed glottis (Valsalva maneuver): +100 mm Hg.

- **Significance of Intra-alveolar Pressure**

  1. Intra-alveolar pressure causes flow of air in and out of alveoli. During inspiration, the intra alveolar pressure becomes negative, so the atmospheric air enters the alveoli. During expiration, intra alveolar pressure becomes positive. So, air is expelled out of alveoli.

- Intra alveolar pressure also helps in exchange of gases between the alveolar air and the blood.
- **Transpulmonary Pressure**

  Transpulmonary pressure is the pressure difference between intra-alveolar pressure and intrapleural pressure. It is the measure of elastic forces in lungs, which is responsible for collapsing tendency of lungs.
- **COMPLIANCE**

  Compliance is the ability of the lungs and thorax to expand or it is the **expansibility of lungs and thorax. It** is defined as the change in volume per unit change in the pressure.

  Change in volume of lung

  Change in airway pressure

  It is expressed as litre/cmH_2o

- **Significance of Determining Compliance**

  Determination of compliance is useful as it is the measure of stiffness of lungs. Stiffer the lungs, less is the compliance.
- **NORMAL VALUES**

  Compliance is expressed by two ways:

  1. In relation to intraalveolar pressure
  2. In relation to intrapleural pressure.
- **Compliance in Relation to Intra-alveolar Pressure**

  Compliance is the volume increase in lungs per unit increase in the intraalveolar pressure.

  1. Compliance of lungs and thorax together:

  130 mL/1 cm H_2O pressure

  2. Compliance of lungs alone:

  220 mL/1 cm H_2O pressure.

- **Compliance in Relation to Intrapleural Pressure**

  Compliance is the volume increase in lungs per unit decrease in the intrapleural pressure.

  1. Compliance of lungs and thorax together:

  100 mL/1 cm H_2O pressure

  2. Compliance of lungs alone:

  200 mL/1 cm H_2O pressure.

  Thus, if lungs could be removed from thorax, the expansibility (compliance) of lungs alone will be doubled. It is because of the absence of inertia and restriction exerted by the structures of thoracic cage, which interfere with expansion of lungs.

- **Specific Compliance**

  The term specific compliance is introduced to assess the stiffness of lung tissues more accurately.

- It is the compliance divided by functional residual capacity.

- Specific compliance

  of lungs = Compliance of lungs

  Functional residual capacity

  Functional residual capacity is the volume of air present in lungs at the end of normal expiration.

- **TYPES OF COMPLIANCE**

  Compliance is of two types:

  1. Static compliance

  2. Dynamic compliance.

  1. Static Compliance

  Static compliance is the compliance measured under **static conditions, i.e. by measuring pressure and** volume when breathing does not take place. Static compliance is the pressure required to overcome the elastic resistance of respiratory system for a given tidal volume under zero flow (static) condition.

- **2. Dynamic Compliance**

  Dynamic compliance is the compliance measured during **dynamic conditions, i.e. during breathing.**

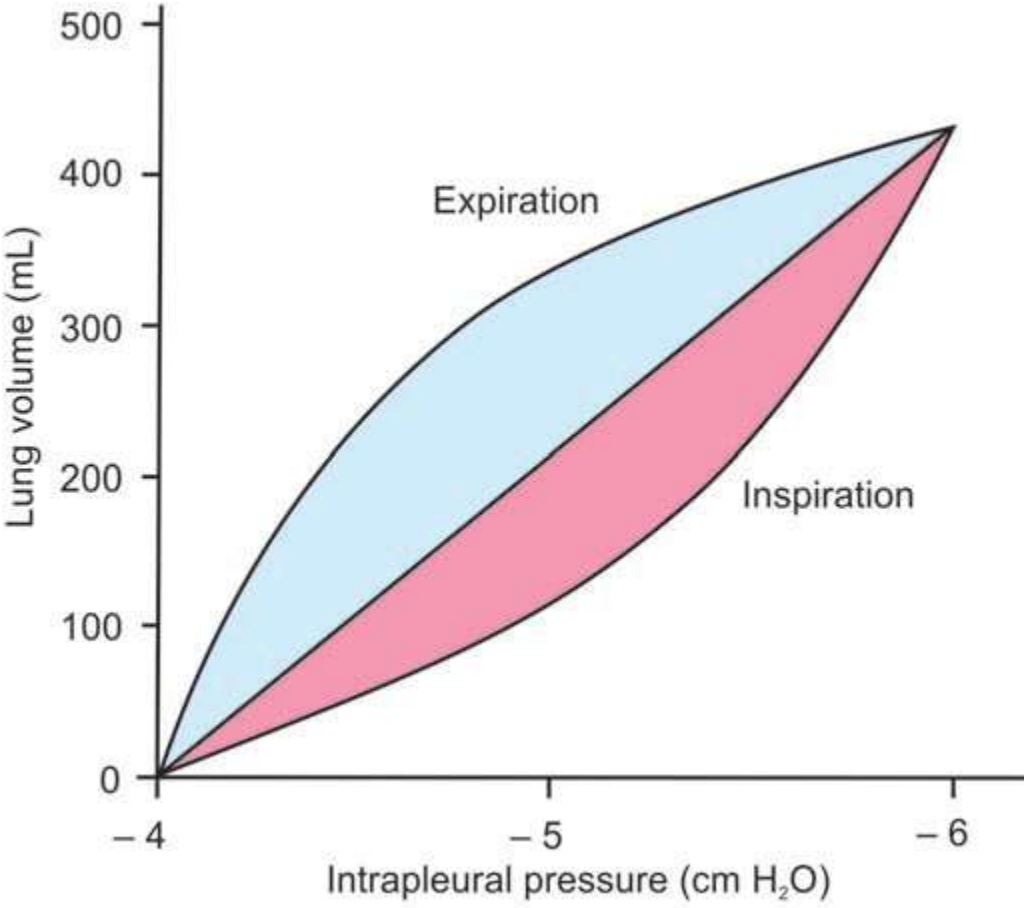
- **Static Compliance Vs Dynamic Compliance**

  In healthy subjects, there is little difference between static and dynamic compliance. In patients with stiff lungs, the dynamic compliance decreases while little change occurs in the static compliance.

- **Measurement of Compliance**

1. To measure the static compliance, the subject is asked to inspire air periodically at regular steps from a spirometer.
2. In each step, a known volume of air is inspired.
3. At the end of each step, intrapleural pressure is measured by means of an **esophageal balloon.**
4. Then, the air is expired in steps until the volume returns to original preinspiratory level. Intrapleural pressure is measured at the end of each step.

Pressure-volume curve



- Values of volume and pressure are plotted to obtain a curve, which is called **pressure-volume curve. From** this curve compliance can be calculated. This curve also shows the difference in inspiration and expiration.

- **APPLIED PHYSIOLOGY**

  Increase in Compliance

  Compliance increases due to loss of elastic property of lung tissues, which occurs both in physiological and pathological conditions:

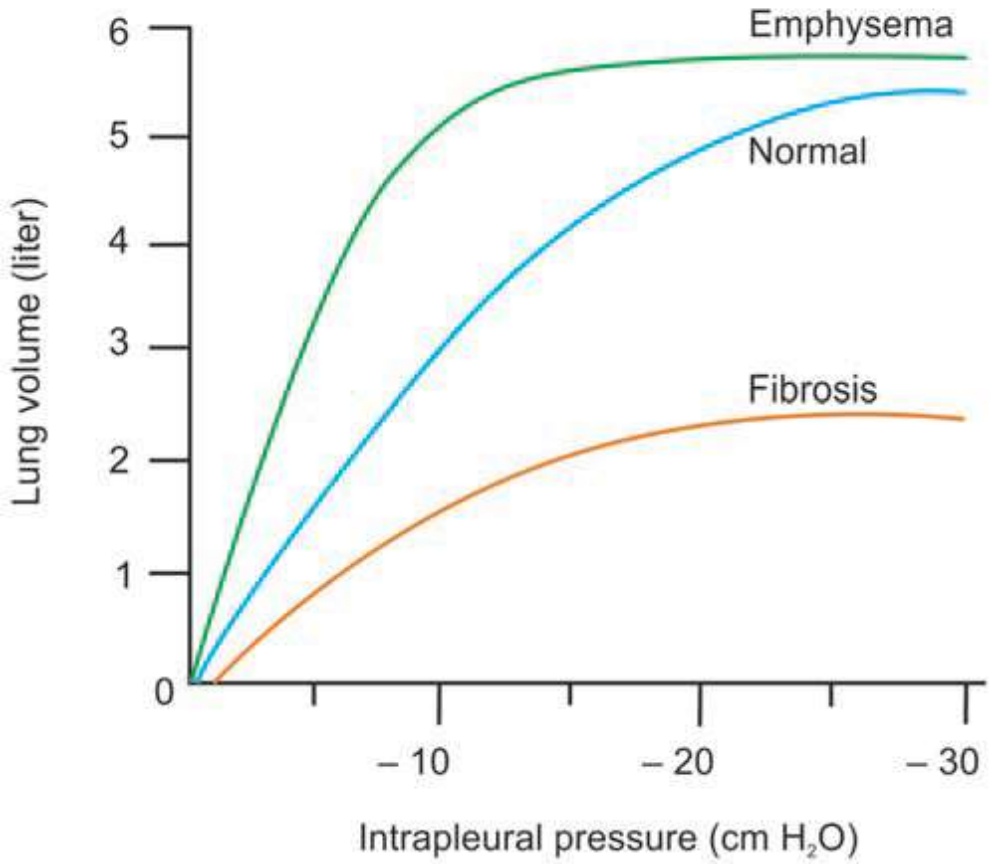
  1. Physiological condition: Old age
  2. Pathological condition: Emphysema

- **Decrease in Compliance**

  Compliance decreases in several pathological conditions such as:

  1. Deformities of thorax like kyphosis and scoliosis
  2. Fibrotic pleurisy (inflammation of pleura resulting in fibrosis)
  3. Paralysis of respiratory muscles
  4. Pleural effusion
  5. Abnormal thorax such as pneumothorax, hydrothorax, hemothorax and pyothorax

Variation in lung compliance



Alveolar Surface Tension

- The lungs and chest wall are elastic structures. The lungs are stretched when they are expanded at birth and at the end of resting expiration their tendency to recoil from the chest wall is just balanced by the tedency of chest wall to recoil in opposite direction.
- Therefore, if the chest wall is opened the lungs lose their elasticity, the chest expands and becomes barrel shaped.
- The tendency of the lungs to recoil back from the chest wall is governed by 2 forces:

I. Recoil of elastic tissues of the lungs and

II. Surface tension within the alveoli, due to the fact that alveoli contain a very thin film of fluid lining their inner side

- Surface Tension is due to the intermolecular attraction between surface molecules and thus it tries to reduce surface area and collapses the lungs.
- Hence this surface tension must be reduced especially during expiration. Otherwise, the lungs will collapse. Moreover, this force opposes the expansion of the lungs during inspiration.
- According to the Law of Laplace in spherical structure like alveoli, outer surface or distending pressure equals two times the surface tension divided by the radius,
- $P = \frac{2T}{R}$

Where,

P = distending pressure

T = tension on the walls due to surface tension

R = radius

- Surface tension(T) is inward force. Therefore. With **P** constant if **T** is not reduced during expiration, surface tension may overcome the distending pressure and then lungs will collapse. But in the lungs, with reduction of radius, there is reduction of surface tension by surface tension lowering agent called **Surfactant.**

SURFACTANT

Surfactant is a **surface acting material or agent that is** responsible for lowering the surface tension of a fluid.

Surfactant that lines the epithelium of the alveoli in lungs is known as **pulmonary surfactant and it decreases the surface tension on the alveolar membrane.**

Source of secretion of pulmonary surfactant

Pulmonary surfactant is secreted by two types of cells:

1. **Type II alveolar epithelial cells in the lungs, which** are called surfactant secreting alveolar cells or pneumocytes. Characteristic feature of these cells is the presence of microvilli on their alveolar surface.

2. **Clara cells, which are situated in the bronchioles.**

These cells are also called bronchiolar exocrine cells.

- **Chemical composition of surfactant**

  Surfactant is a **lipoprotein complex formed by lipids** especially phospholipids, proteins and ions.

  1. Phospholipids*:* Phospholipids *form about 75%* of the surfactant. Major phospholipid present in the surfactant is **dipalmitoylphosphatidylcholine** (DPPC).

  2. Other lipids*:* Other lipid substances of surfactant are triglycerides and phosphatidylglycerol (PG).

  3. Proteins: Proteins of the surfactant are called specific surfactant proteins. There are four main surfactant proteins, called SPA, SPB, SPC and SPD. SPA and SPD are hydrophilic, while SPB and SPC are hydrophobic. Surfactant proteins are vital components of surfactant and the surfactant becomes inactive in the absence of proteins.

  4. Ions: Ions present in the surfactant are mostly calcium ions.

- **Functions of surfactant**

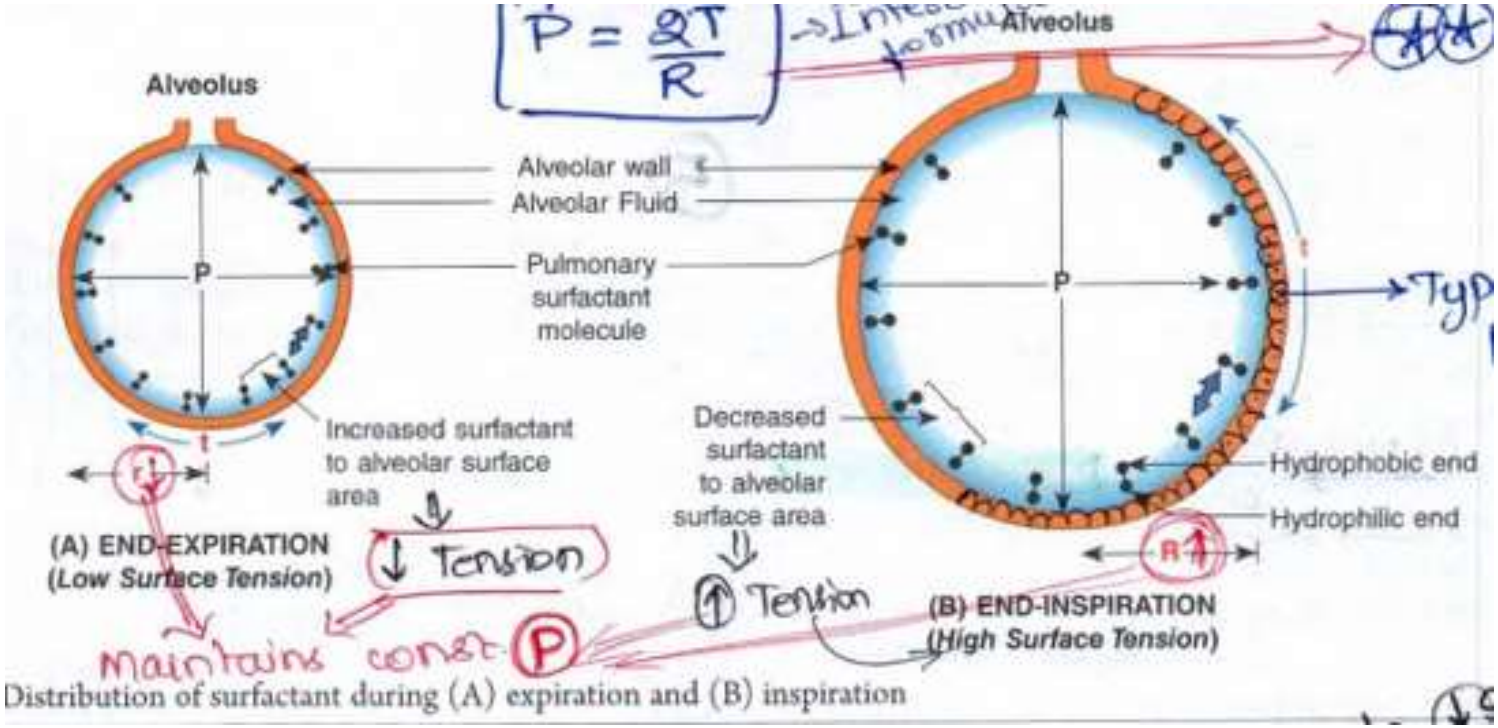
1. Surfactant reduces the **surface tension in the** alveoli of lungs and prevents **collapsing tendency** of lungs.

I. There is a layer of fluid and air lining the alveoli which causes surface tension.

II. Surfactant reduces the surface tension by forming a layer between the fluid lining the alveoli and alveolar air. **Hydrophilic** portion of surfactant dissolves in water and lines the alveoli. Other portion is **hydrophobic and it is** directed towards the alveolar air. This surface of the phospholipid along with other portion spreads over the alveoli and reduces the surface tension. SPB and SPC play active role in this process.

III. Thus it prevents development of surface tension between the fluid and air.

IV. Surface tension between between the fluid and the air is 7-24 times more than that between the surfactant and the air.



Distribution of surfactant during (A) expiration and (B) inspiration

V. Surface Tension is inversely proportional to the concentration of surfactant per unit area. Surfactant molecules are spread apart as alveolar size increases during inspiration but come closer during expiration thereby adjusting surface tension during breathing. Its action is more effective during expiration than during inspiration.

2. Surfactant is responsible for stabilization of the alveoli, which is necessary to withstand the collapsing tendency.

3. It plays an important role in the inflation of lungs after birth.

I. In fetus, the secretion of surfactant begins after the 3rd month. Until birth, the lungs are solid and not expanded.

II. Soon after birth, the first breath starts because of the stimulation of respiratory centers by hypoxia and hypercapnea.

III. Although the respiratory movements are attempted by the infant, the lungs tend to collapse repeatedly. And, the presence of surfactant in the alveoli prevents the lungs from collapsing.

3.Surface tension keeps the alveoli dry and thus helps in exchange of gases.

Normal pulmonary capillary hydrostatic pressure is 8-10mmHg and oncotic pressure is 25mmHg. This produces an inwardly directed pressure gradient of about 15-17mmHg. Therefore, low pulmonary hydrostatic pressure helps to pull fluid from alveoli into pulmonary capillaries and keeps the alveolar surface free of fluid.

4. Surfactant help to prevent pulmonary oedema by reducing the surface tension.

If surfactant was not present, the unopposed surface tension in the alveoli would produce a 20mmHg force, which can draw out fluid from the blood capillaries into the alveoli(i.e. transudation of fluid) producing pulmonary oedema. Surfactant prevents this phenomenon by lowering the surface tension.

- **5.** Another important function of surfactant is its role in defense within the lungs against infection and inflammation. Hydrophilic proteins SPA and SPD destroy the bacteria and viruses by means of opsonization. These two proteins also control the formation of inflammatory mediators.
- **Factors affecting surfactant**
- **Surfactant decreases due to:**

I. Long term inhalation of 10%O2(as occurs during cardiac surgery)
II. Occlusion of main bronchus
III. Occlusion of one pulmonary artery
IV. Cigarette smoking
V. Cutting both the vagi(vagal innervation is important for the maturation of the surfactant system.)

- **Surfactant increases due to:**

I. Thyroid hormones, increase the size and number of inclusion bodies in type II alveolar lining epithelial cells
II. Glucocorticoids, accelerate the maturation of surfactant
III. Absence of GM-CSF (Granulocyte-macrophage colony-stimulating factor plays a crucial role in regulating surfactant metabolism and its deficiency can lead to surfactant accumulation.)

- **Applied physiology**

1.Hyalin membrane disease or infant respiratory distress syndrome(IRDS)

- It is a serious disease of newborn infants due to deficiency of surfactant, or it develops in infants born before their surfactant system becomes functional.(Surfactant is always present by 35 weeks of gestation). Normally after birth, the infant makes several strong inspiratory movements and the lungs expand. Surfactant keeps them from collapsing again.
- Prolonged immaturity of epithelial Na+ channels,results in defi ciency of surfactant. Therefore,the surface tension in the lungs of these infants is very high, and there are many areas in which alveoli are collapsed(Atelectasis). Also pulmonary oedema occurs and infants die of pulmonary insufficiency.
- IRDS is also called hyaline membrane disease due to formation of hyaline(a translucent) membrane from albuminous intrapulmonary fluid in the walls of the alveoli and respiratory bronchioles.

2.In addition, the deficiency of surfactant increases the susceptibility for bacterial and viral infections.

WORK OF BREATHING

It is the work done by respiratory muscles during breathing to overcome the resistance in thorax and respiratory tract.

- **WORK DONE BY RESPIRATORY MUSCLES**

During respiratory processes, inspiration is active process and the expiration is a passive process. So, during quiet breathing, respiratory muscles perform the work only during inspiration and not during expiration.

- **UTILIZATION OF ENERGY**

During the work of breathing, the energy is utilized to overcome three types of resistance:

1. Airway resistance
2. Elastic resistance of lungs and thorax
3. Non elastic viscous resistance.

1. Airway Resistance

Airway resistance is the resistance offered to the passage of air through respiratory tract. Resistance increases during bronchiolar constriction, which increases the work done by the muscles during breathing. Work done to overcome the airway resistance is called airway resistance work.

Airway resistance is inversely related to total cross-sectional area of the respiratory passage. This greatly increases from 2.5cm2 in the trachea to11,800cm2 in the alveoli. Therefore, airflow resistance is high in conducting zone whereas low in the respiratory zone.

Airway resistance is high during expiration as diameter of airways decreases.

2. Elastic Resistance of Lungs and Thorax

Energy is required to expand lungs and thorax against the elastic force. Work done to overcome this elastic resistance is called compliance work.

3. Non-elastic Viscous Resistance

Energy is also required to overcome the viscosity of lung tissues and tissues of thoracic cage. Work done to overcome this viscous resistance is called **tissue resistance work.**

Distribution of various forms of resistances, when expressed as % of total resistance, is:

1.Elastic resistance: 65%

2. Non-Elastic resistance: 35%

(i) Viscous resistance: 7%

(ii) Airway resistance:28%

- Factors affecting total work done

  1. increases markedly during exercise(physiological)

  2. causes of pathological increase:

I. Emphysema
II. Bronchial Asthma
III. Congestive heart failure with dyspnoea
IV. Orthopnoea

Assessment

1.Draw well labelled diagram:

(i) Change in intra-pleural and intra-pulmonary pressure with respiratory cycle

(ii) Pressure-volume relationship of lungs alone

(iii) Distribution of surfactant during inspiration and expiration

2.Write short notes on:

(i) Major muscles involved in respiration and role of each.

(ii) Factors affecting intra-pulmonary and intra-pleural pressure

(iii) Surfactant and its actions. Explain the factors responsible for its maturation

(iv) Hyaline membrane disease

(v) Advantage of residual volume to the lungs

(vi) Factors affecting compliance of lungs

(ix) Factors affecting total dead space

(x) V/P ratio and its physio-clinical significance

(xi) Factors affecting VIP ratio

(xii) Factors affecting diffusion capacity of lungs

(xiii) Factors determining alveolar ventilation

(xiv) Airway resistance and factors affecting it

What will happen:

(i) If bilateral paralysis of diaphragm muscle occurs

(ii) if intrapulmonary pressure increases

(iii) if intrapleural pressure is increased

(iv) if lungs lose their elasticity

(v) if airway resistance is increased?

MCQs

1.Eupnoea means:

(a)Rhythmic normal breathing at rest

(b) Cessation of breathing

(c) Difficulty in breathing

(d) Consciousness of breathing at rest

2. For each 1 cm descent of diaphragm, the amount of air sucked into the lungs is:

(a) 100-200 mL (b)200-300 mL (c) 300-400 mL (d) 400-500 mL

3. Tendency of the lungs to recoil from the chest wall is balanced by the tendency of chest wall to recoil in the opposite direction at:

(a)End expiratory position

(c) End inspiratory position

(b) Maximum expiratory position

(d) Maximum inspiratory position

4. The most important factor that tends to collapse the lungs is the:

(a) lntrapleural fluid pressure

(b)Surface tension of the alveolar fluid

(c) Total intrapleural pressure

(d) Tension in the intercostal muscles

5. Surfactant causes:

(a)Increased compliance of lungs

(b) Secreted by bronchus

(b) Decreased compliance of lungs

(d) Absent at birth

6. Bilateral phrenic nerve paralysis results in:

(a) Death

(b) Is fatal without artificial respiration

(c) adequate ventilation to maintain life

(d) Normal respiration

THANKS