# MECHANISM OF RESPIRATION

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## Objectives

- Explain how the intrapulmonary and intrapleural pressures vary during ventilation and relate these pressure changes to Boyle's law.
- Define the terms compliance and elasticity, and explain now these lung properties affect ventilation.
- Discuss the significance of surface tension in lung mechanics, explain how the law of Laplace applies to lung function and describe the role of pulmonary surfactant.



## Objectives (continued)

- Explain how inspiration and expiration are accomplished in unforced breathing and describe the accessory respiratory muscles used in forced breathing.



## Every Breath You Take

- Repeated 12 to 16 times per minute
- Has a tidal volume of 500 mL
- Has a portion (30%) which does not reach the alveoli (anatomic dead space)
- Has the remaining 70% reaching the alveolar zone



## Fun Facts

At rest, the body takes in and breathes out about 10 liters of air each minute.
The right lung is slightly larger than the left.
The highest recorded "sneeze speed" is 165 km per hour.
The surface area of the lungs is roughly the same size as a tennis court.
The capillaries in the lungs would extend 1,600 kilometers if placed end to end.
We lose half a liter of water a day through breathing. This is the water vapor we see when we breathe onto glass.
A person at rest usually breathes between 12 and 18 times a minute.
The breathing rate is faster in children and women than in men.



## Mechanism of respiration Concepts

- Deals with various forces and processes involved in ventilation.
- Achived by simple process: when the pressure in side the chest is decreased the air moves in ----inspiration and vice-versa.
- Thoracic cage : base is formed by the diaphragm, is made up of ribs, vertebrae, sternum, muscles and ligaments--- elastic
- So if the chest forcefully expands it will try to collapse and in reverse situation it will prevent collapse

## Concepts

- Lungs: elastic but unlike the chest wall they only have collapsing tendency, but no ability to expand on its own.
- Pleural and pleural cavity: two layers of pleura are attached in such a way due to capillary action of the fluid, so the layers are inseparable unless pneumo or hydrothorax. So the negative pressure is manintained



## Mechanics of breathing

- Two phases
  - Inspiration = inhalation of atmospheric air
  - Expiration = exhalation of atomospheric air
- Occur because of volume changes in thoracic cavity
  - Boyles Law => $P1 \times V1 = P2 \times V2$



## Lung Pressures

- Intrapleural pressure (IPP):
  - Pressure in the intrapleural space.
  - Pressure is negative, due to lack of air in the intrapleural space
  - due to elastic recoil lung wants to collapse, while chest wall expands and maintain the –ve pressure
  - during quite breathing IPP is -5 to -7.5 cm $H_2O$.
  - during forced inspiration may be up to -40cm $H_2O$
  - during forced expiration may be be up to +40 cm $H_2O$
  - when IPP decrease inspiration occurs and also helps venous return.
  - when IPP increase expiration occurs and no venous return and decrease in Cardiac output will occurs.



- Intrapulmonary pressure:
  - Intra-alveolar pressure (pressure in the alveoli).
  - the pressure is equal to the atmosphere, ie O at rest and no airflow.
  - during inspiration : -ve and expiration: +ve
- Transpulmonary pressure:
  - Pressure difference across the wall of the lung.
  - Intrapulmonary pressure – intrapleural pressure.
    - Keeps the lungs against the chest wall and prevents the collapse of the airways.

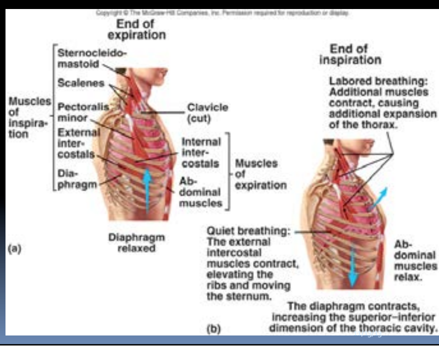


## Boyle’s Law

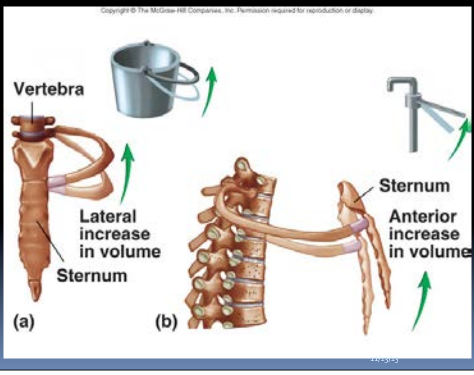
- Changes in intrapulmonary pressure occur as a result of changes in lung volume.
  - Pressure of gas is inversely proportional to its volume.
- Increase in lung volume decreases intrapulmonary pressure.
  - Air goes in.
- Decrease in lung volume, raises intrapulmonary pressure above atmosphere.
  - Air goes out.



## Thoracic Walls
## Muscles of Respiration



## Thoracic Volume






## Inspiration

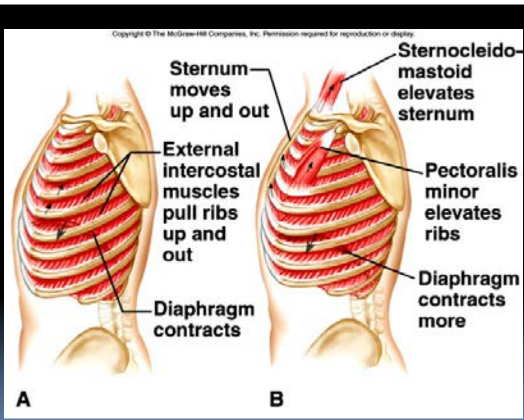
- Volume of thoracic cavity increases
  - *Decreases* internal gas pressure
  - contraction of the intercostal muscles which are situated between the ribs--lifting upwards and outwards.
  - There is also movement in the body as the DIAPHRAGM contracts, changing from a dome shape to a flatter sheet.



## Inspiration

- *Deep* inspiration requires
  - Scalenes
  - Sternocleidomastoid
  - Pectoralis minor
  - Erector spinae – extends the back








## Changes in Thoracic Volume

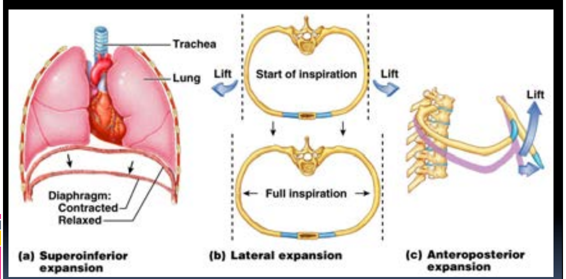
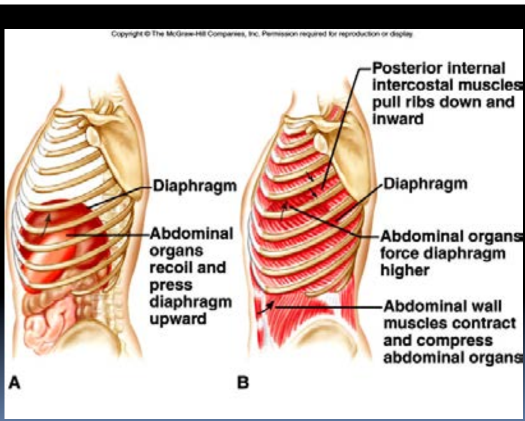



Figure 21.16a–c

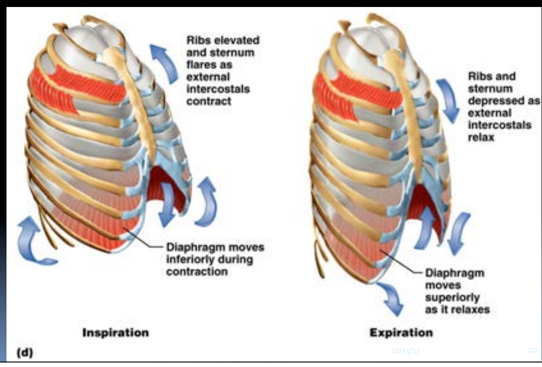


## Expiration

- Quiet expiration – chiefly a *passive process*
  - Inspiratory muscles relax
  - Diaphragm moves superiorly
  - Volume of thoracic cavity *decreases*
- Forced expiration – an active process
  - Produced by contraction of
    - Internal and external oblique muscles
    - Transverse abdominis muscles



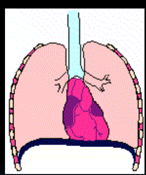

## Changes in Thoracic Volume




## PHYSIOLOGY OF RESPIRATION

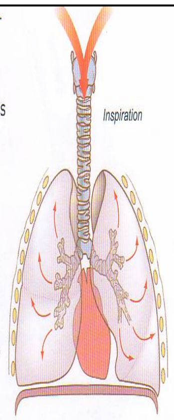
During inspiration, as these muscles contract, the thorax expands. Intrathoracic pressure decreases, drawing air into the tracheobronchial tree into the alveoli and expanding the lungs. Gas exchange takes place in the alveoli.

After inspiratory effort stops, the expiratory phase begins. The chest wall and the lungs recoil, the diaphragm relaxes and rises passively, air flows outward and the chest and abdomen return to their resting positions.



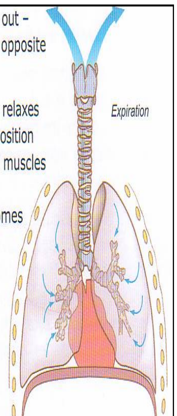
When we breathe in – **inspiration** – the following happens:

- our diaphragm pulls down
- our intercostal muscles contract
- air pressure is reduced
- air is sucked through the tubes into the lungs
- our chest expands.

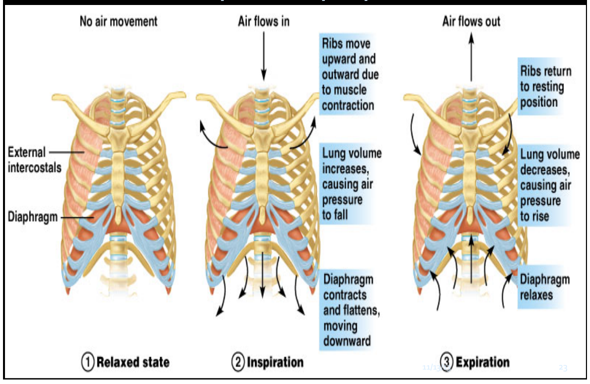



When we breathe out – **expiration** – the opposite happens:

- our diaphragm relaxes into its dome position
- our intercostal muscles relax
- our chest becomes smaller
- pressure increases on our lungs
- air is forced out.




## Respiratory Cycle




| What happens to me.....? | ACTION | |
|---|---|---|
| | When I breathe in | When I breathe out |
| RIB CAGE | | |
| DIAPHRAGM | | |
| CHEST | | |
| LUNGS | | |

## Quiet Inspiration

- Active process:
  - Contraction of diaphragm, increases thoracic volume vertically.
  - Contraction of parasternal and internal intercostals, increases thoracic volume laterally.
  - Increase in lung volume decreases pressure in alveoli, and air rushes in.
- Pressure changes:
  - Alveolar changes from 0 to –3 mm Hg.
  - Intrapleural changes from –4 to –6 mm Hg.
  - Transpulmonary pressure = +3 mm Hg.

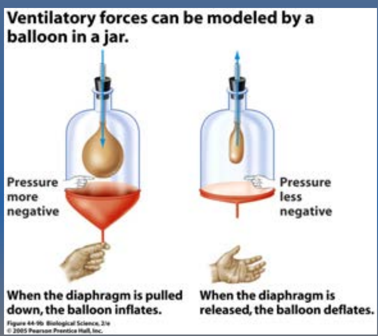


## Expiration

- Quiet expiration is a passive process.
  - After being stretched, lungs recoil.
  - Decrease in lung volume raises the pressure within alveoli above atmosphere, and pushes air out.
- Pressure changes:
  - Intrapulmonary pressure changes from –3 to +3 mm Hg.
  - Intrapleural pressure changes from –6 to –3 mm Hg.
  - Transpulmonary pressure = +6 mm Hg.

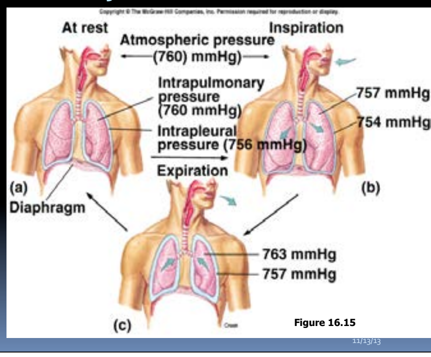


Here is an experiment that you can try.



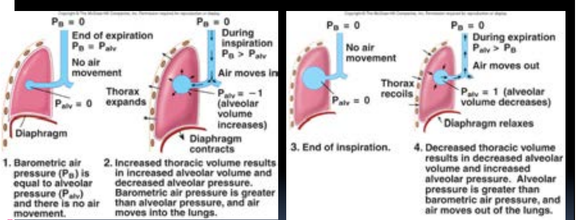



## Pressure change during Pulmonary Ventilation



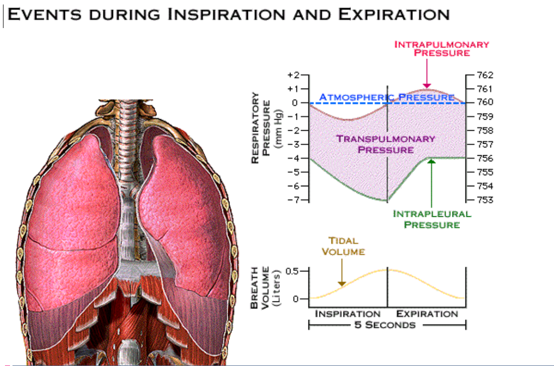



## Alveolar Pressure Changes

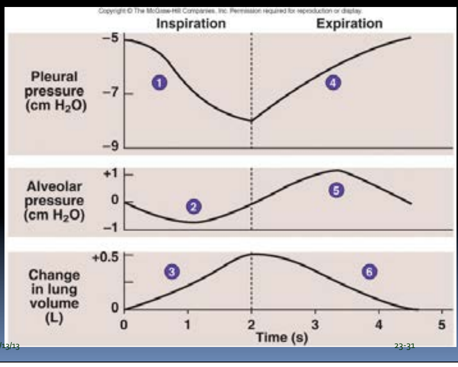





## Events during Inspiration and Expiration



## Normal Breathing Cycle




11/13/13 23-31

## Factors influencing ventilation

**1. Compliance:**

- Measure of the ease with which lungs and thorax expand
- is expressed as volume change per unit transpulmonary pressure change during inflation or deflation.
- normal value is 0.2 L/cm of $H_2O$.
- depends on lungs elastic properties and on the surfactant present in the alveoli.

11/13/13 32

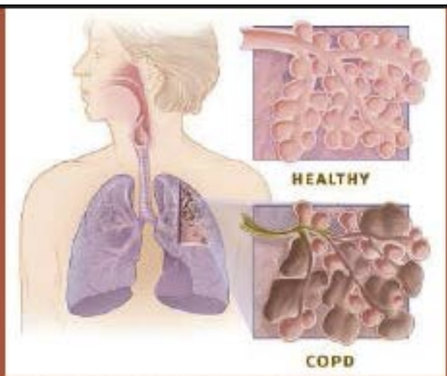
- The greater the compliance, the easier it is for a change in pressure to cause expansion
- A lower-than-normal compliance means the lungs and thorax are harder to expand
- related to lung and chest wall elasticity and surface tension
  - Conditions that decrease compliance
    - Pulmonary fibrosis
    - Pulmonary edema
    - Respiratory distress syndrome

11/13/13 33

## Lung Elasticity

- Depends upon elastic fibers in alveoli and smaller airways
- COPD loses alveolar wall and loses elasticity
- Increased compliance occurs because of COPD
- Decreased compliance occurs because of pneumonia, fibrosis, kyphosis, or decreased surfactant

11/13/13 34




http://www.nhlbi.nih.gov/health/public/lung/copd/images/diagram/healthy-vs-copd.jpg

11/13/13 35

## 2. Airway resistance

- It is the resistance the air encounters while passing through the airways.
- Depends on the diameter of the airway, length of the airway and viscosity of the air.
- Since the length of the airway and viscosity of air don't change, so Resistance depends on the diameter of the airway that is will decrease if the diameter of the airway is bigger

11/13/13 36

## 2. Airway resistance

- Airway resistance- Airway diameter affects resistance
  - Think of breathing through straw
  - Occurs because of friction between air and walls of airways
- Larger airways = little resistance
- Smaller airways = increased resistance
- Greatest resistance in healthy humans is in the trachea!!!!!
  - Think summative effect!

11/13/13 37

## Factors influencing airway resistance

- The lung volume:

1. higher the lung volume the lower the resistance
2. In high lung volume there is more elastic recoil of lungs so more negativity in the intrapleural pressure which will exerts radial traction on the bronchi– which will be more dilated.

- The tone of the respiratory smooth muscles

1. The tone determines the lumen size of the airway-tone increases airway becomes narrow and the pressure increases.

11/13/13 38

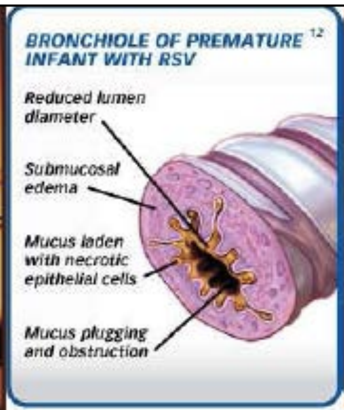
- Viscosity of the air: depends in the density of the air. In high altitude the airway resistance is low. Resistance increases in a polluted atmosphere.
- Breathing

1. During inspiration airway are stretched so the resistance decreases
2. during expiration the airway are compressed due to increase intrapulmonary pressure, so the resistance is high and even some airway can collapse completely.

◆ Expiratory flow rate can not be increased beyond certain level.

◆ during Br asthma expiration is difficult than inspi

11/13/13 39




- **Small inflamed airways become point of highest resistance!!**

http://www.synagis.com/hcp/images/g.jpg

11/13/13 40

## 3. Surface Tension

- Force exerted by fluid in alveoli to resist distension.
  - Lungs secrete and absorb fluid, leaving a very thin film of fluid.
  - This film of fluid causes surface tension.
- $H_2o$ molecules at the surface are attracted to other $H_2o$ molecules by attractive forces.
  - Force is directed inward, raising pressure in alveoli.

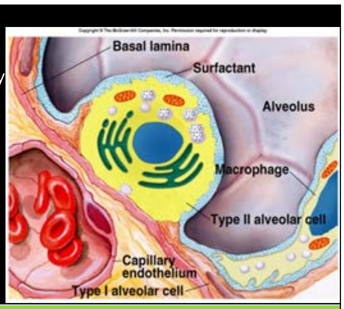
11/13/13 41

## Surfactant

Phospholipid produced by alveolar type II cells.

Lowers surface tension.
- Reduces attractive forces of hydrogen bonding by becoming interspersed between $H_2o$ molecules.

As alveoli radius decreases, surfactant's ability to lower surface tension increases.

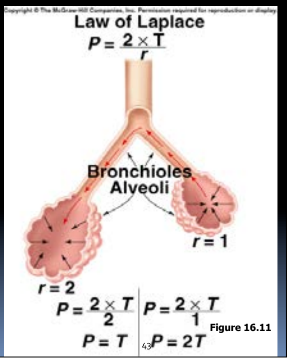



Lowering of the surface tension by the surfactant improves the distensibility of the lungs. Reducing the muscular effort required for breathing is the first function of the surfactant. Surfactant contributes to the stability of the architecture of the lung.

11/13/13 42

## Law of Laplace

- Pressure in alveoli is directly proportional to surface tension; and inversely proportional to radius of alveoli.
  - Pressure in smaller alveolus greater.
  - Allveoli don't all have the same diameter.
  - smaller alveoli would emty in to large alveoli till the entire lung work as a giant alveolus – which doesn't happen




43

- amount of the surfactant secreted by the cell is relatively constant.
- in larger alveoli the surfactant spreads over the large surface and so is less effective than smaller alveoli--- larger alveoli experience less reduction of the surface tension.
- so the surface tension in large alveoli is greater than smaller alveoli.
- but P = 2T/r and and surface tension increases with radius decrease and T/r is constant. Meaning presure in large alveoli is same with smaller alveoli. So small alveoli will not empty in to large alveoli---- arhciteture of lungs will be stable.

11/13/13 44