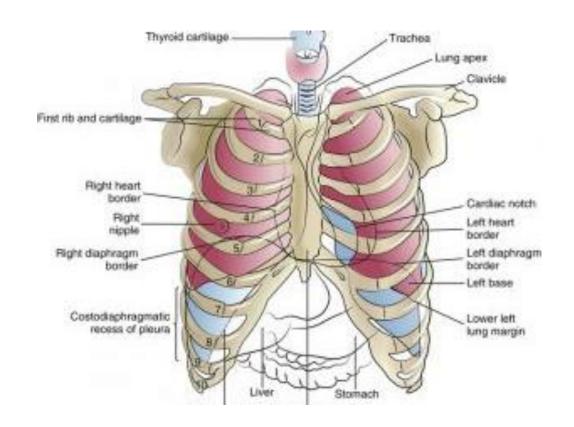
Pulmonary volume and capacities

DR. Arwa Rawashdeh

Quit normal breathing

Inspiration (negative pressure)
Expiration (positive pressure)

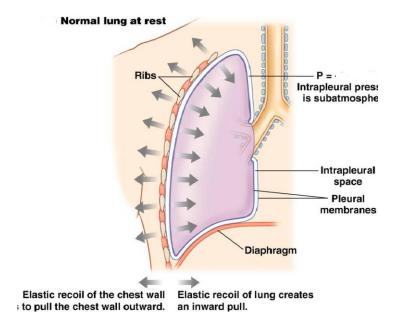
Intrapleural or intrathoracic pressure: is always negative due to dynamic harmonious antagonism between the chest wall and the lung



Mechanics of breathing

Intra plural pressure

- -5 to -7 normal
- Less negative emphysema
- Zero at birth and stab wound without valve
- Positive stab wound tension pneumothorax with valve and Valsalva maneuver



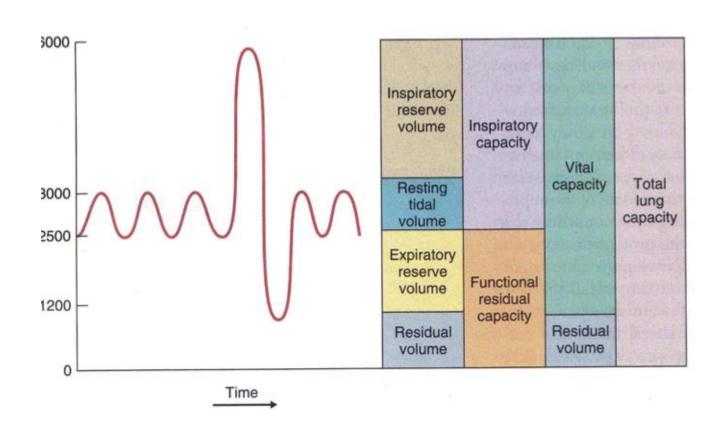
There are three types of pressure:

- 1. Intrapulmonary a.k.a. intra-alveolar. [can be positive or negative]
- 2. Intrapleural a.k.a. intrathoracic. [always negative]
- 3. Transthpulmonary a.k.a. transmural: the difference between 1 and 2 [always positive]

- The greater resistance is found in the bronchi.
 - That's why asthma can be dangerous —> bronchial hyperreactivity —>
 constriction of bronchi (which already have more resistance than bronchioles)
 —> decreased radius —> increased resistance —> air cannot exit —>
 obstructive lung disease.
- Quesiton: is the problem worse during inspiration or expiration?
 - Answer: -expiration, because the airways are narrower -> less radius -> more resistance.
 - so, during expiration, I cannot get the air out (Obstructive lung disease)

	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) • I mean the intrapulmonary (intra-alvelolar pressure) pressure decreased to -1 cmH2O assuming that the atmospheric pressure is zero.	 I mean the intrapulmonary (intra-alvelolar pressure) pressure increased to +1 cmH2O assuming that the atmospheric pressure is zero.
Muscles	Diaphragm: decends. external intercostals: > elevate ribs> increase transverse diameter. > evert ribs> increases AP diameter.	- passive- -diaphragm ascends, lungs shrink by their elastic recoil.
Accessory muscles for forced	Forced inspiration: • Sternocleidomastoid. • serratous anterior • scalene muscles	Forced expiration (voluntary "musical instuments", obstruvtive [COPD], restrictive [fibrosis]): • internal intercostals • abdominal muscles "abdominal recti" [abdominal breathing]
Effect on murmurs	Inspiration accentuates the right-sided murmurs 💝	Expiration accentuates the murmurs of left side of the heart

Lung volume and capacities



- Volume: one thing
- Capacity: more than one volume

Spirometer cannot measure

- RV
- FRV
- TLC

Residual volume

RV: the air that remains in the lung after maximal forced expiration

Physiological significance

Maintain aeration of blood

Prevent sudden flotation of blood gases

Clinical significance

Obstructive lung disease (emphysema)

RV/TLC>30% emphysema normal =21%

Forensic significance

Autopsy

Stillborn lung will sink in water

Child homicide lung float on water

VC= IRV+TV+ERV=4600mL

Male=2.5L/m2

Female = 2L/m2

Note

FVC (Timed) 3600mL and VC (not timed) 4600mL

Low vital capacity

Physiological

Pregnancy

Recumbent position

Pathological

Chest wall

Deformities in the bone (kyphosis, lordosis.....)

Neuromuscular (muscular dystrophy, myasthenia gravis......)

Lung Obstructive (emphysema) and restrictive (pulmonary fibrosis)

Heart (congestive heart failure) pulmonary edema

Abdomen Hepatomegaly splenomegaly and ascities

FEV1/FVC ratio

FEV1/FVC ratio: 4L/5L=80%

FEV1: the amount of air quickly and forcibly exhale in 1 second

FVC: the amount of air quickly and forcibly exhale after maximum inhalation (timed)

- low FEV1/FVC ratio —> obstructive lung disease
- low TLC -> restrictive lung disease.
- high FEV1/FVC ratio —> restrictive lung disease
- low FEV1/FVC ratio and low TLC —> mixed lung disease (obstructive + restrictive)

Alveolar ventilation

External respiration

Pulmonary Ventilation

Alveolar ventilation

Perfusion

Diffusion

Internal respiration

Mitochondria

FIO2 PAO2 PaO2 SaO2 Carbmino-Hgb PVO2 CO2 breathout

Alveolar capillary membrane diffusion (blood gas barrier)

- Very thin Fibrosis = 1 micron
- Huge surface area Pneumonectomy "Restrictive"
- 6 layers

Fluid lining the alveoli Pneumonia (consolidation)

Alveolar epithelium Emphysema

Epithelial basement membrane fibrosis

Fluid in interstitial space capillary endothelium pulmonary edema

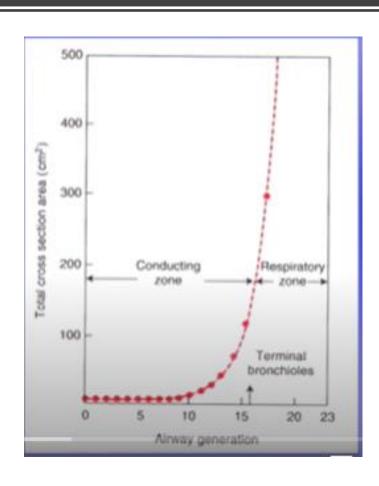
Endothelium basement membrane

Simple diffusion no ATP

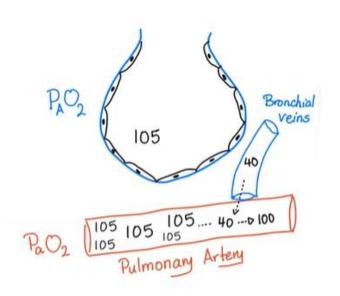
• CO2 out

Narrow range of PH 7- 7.7

Cross sectional area of the airways

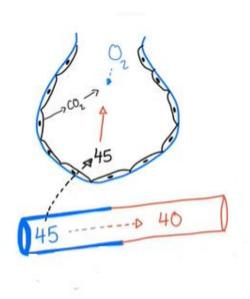






$$P_{A}O_{2} = \left[\left(P_{b} - P_{H_{2}O} \right) \times F_{i}O_{2} \right] - \left[\frac{P_{a}CO_{2}}{0.8} \right]$$

$$P_{i}O_{2} \qquad P_{A}CO_{2}$$



A-a gradient normal extrinsic restrictive lung disease A-a gradient abnormal intrinsic restrictive lung disease

Patient A "normal"

- RR = 12/min, TV = 500 mL
 - Pulmonary ventilation = respiratory rate x tidal volume = 12 x 500 = 6 liters.
 - Alveolar ventilation = respiratory rate x (tidal volume dead space) = 12 x (500-150) = 12x350 = 4.2 liters.

Patient B "increased respiratory rate"

- RR = 30/min, TV = 200 mL
 - Pulmonary ventilation = respiratory rate x tidal volume = 30 x 200 = 6 liters.
 - Alveolar ventilation = respiratory rate x (tidal volume dead space) = 30 x (200 150) = 1.5 liters.

Patient C "increased tidal volume"

- RR = 6/min. TV=1,000 mL
 - Pulmonary ventilation = respiratory rate x tidal volume = 6 x 1000 = 6 liters.
 - Alveolar ventilation = respiratory rate x (tidal volume dead space) = 6 x (1000-150)= 5.1 liters.

Therefore, increasing the tidal volume is a better way to achieve more alveolar ventilation than increasing the respiratory rate.

- But, everything is good within limits.
 - if you increase the tidal volume too much —> the alveoli will expand tremendously (inspiration) and then collapse (expiration)...This big difference, repeated over and over again, can lead to INFLAMMATION!