Physiology of Respiration

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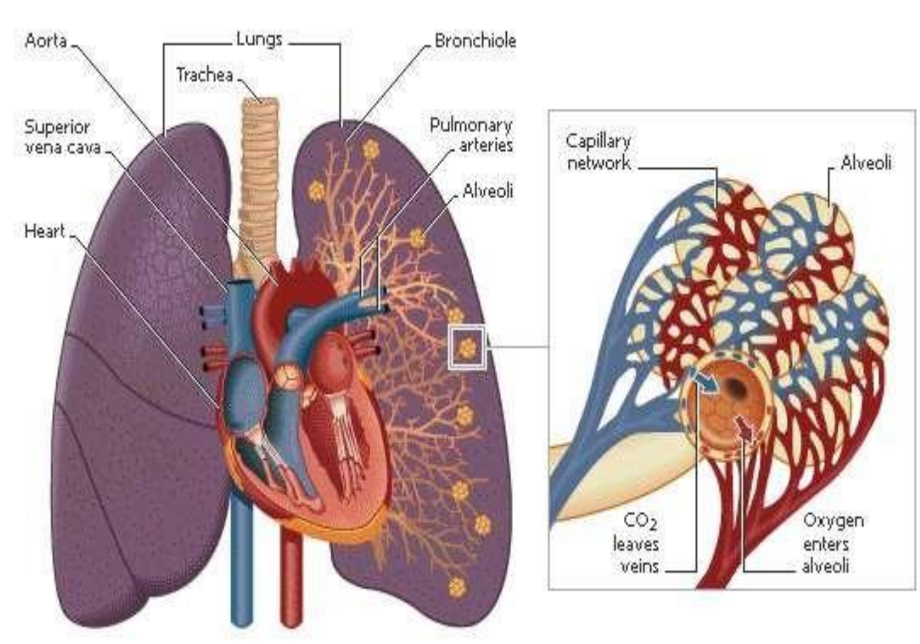
Physiological Anatomy of Respiratory System

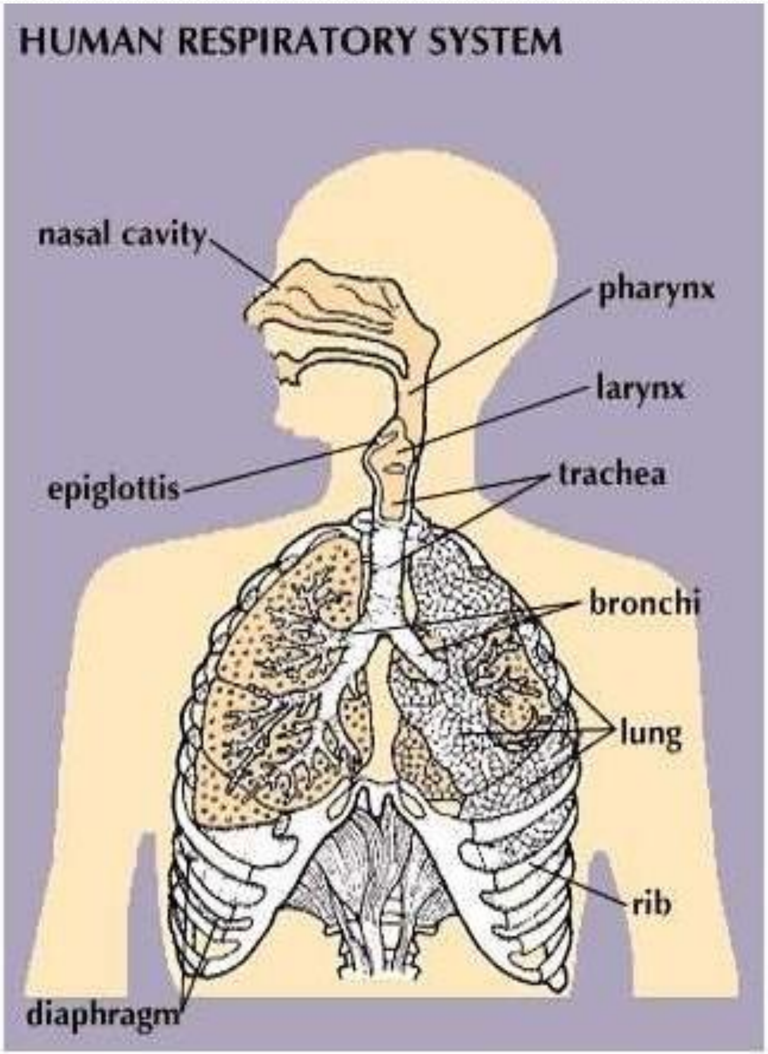
- The respiratory system consists of the **nasal cavity**, **pharynx**, **larynx**, **trachea**, **bronchi**, and **lungs,** which is whole together called as the Respiratory Tract..

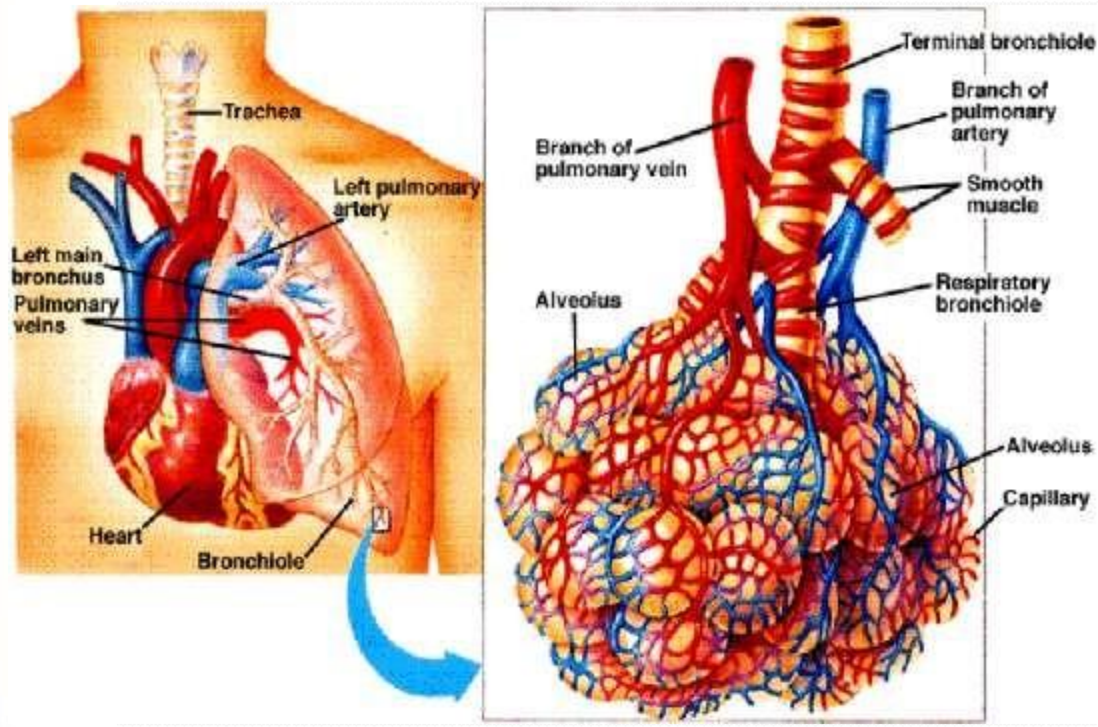
- Upper respiratory tract refers to: Nasal cavity, pharynx, and associated structures.

- Lower respiratory tract refers to: Larynx, trachea, bronchi, and lungs.



The Lungs & Alveoli

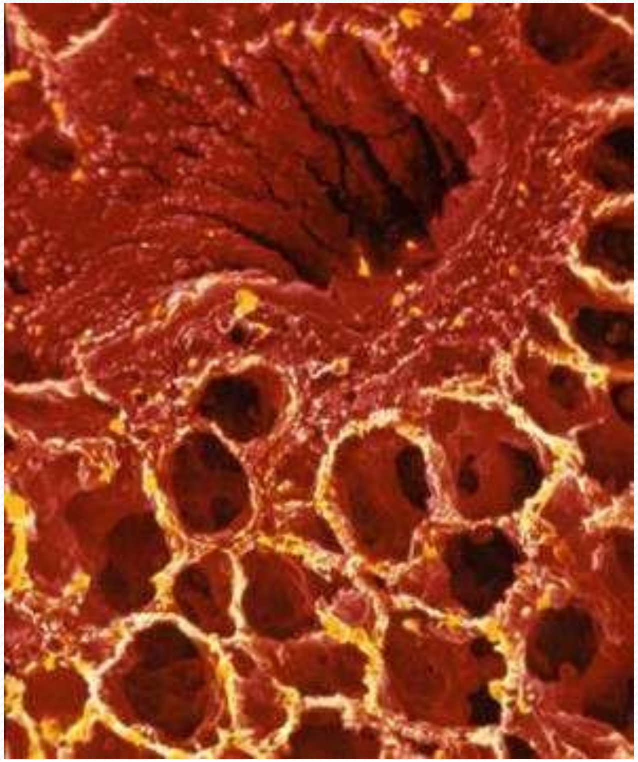

HUMAN RESPIRATORY SYSTEM
nasal cavity
pharynx
larynx
epiglottis
trachea
bronchi
lung
rib
diaphragm


Trachea
Left pulmonary artery
Left main bronchus
Pulmonary veins
Heart
Bronchiole
Terminal bronchiole
Branch of pulmonary artery
Branch of pulmonary vein
Smooth muscle
Alveolus
Respiratory bronchiole
Alveolus
Capillary

Respiratory Unit

Respiratory unit is the terminal portion of the Respiratory Tract. It includes:

- 1. Respiratory Bronchioles
- 2. Alveolar Ducts
- 3. Antrum
- 4. Alveolar Sacs
- 5. Alveoli (Human beings have a thin layer of about 700 million alveoli within their lungs. Which is crucial for respiration, exchanging O2 & CO2 with the surrounding blood capillaries.)



Microscopic Structure of Alveoli

Respiratory Functions:

- 1. Pulmonary Ventilation
- 2. Diffusion of O_2 and CO_2 between the alveoli and the blood.
- 3. Transportation of O_2 & CO_2 in the blood & body fluids to & fro from the body's tissue cells.
- 4. Regulation of Ventilation.

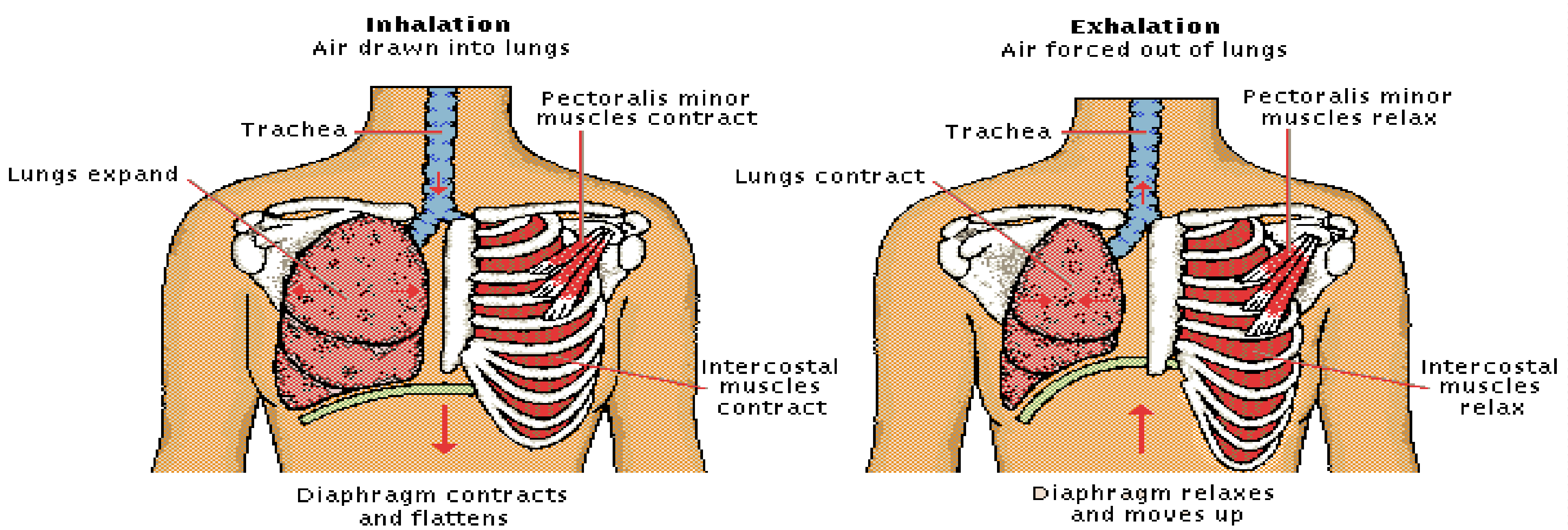
Non- Respiratory Functions:

- 1. Olfaction, Vocalization
- 2. Defense Mechanism
- 3. Anticoagulant Function
- 4. Regulation of Body Temperature & Acid Base Balance
- 5. Maintenance of Water balance
- 6. Secretion of ACE Angiotensin Converting Enzyme

Mechanism of Respiration

- Muscles of Respiration

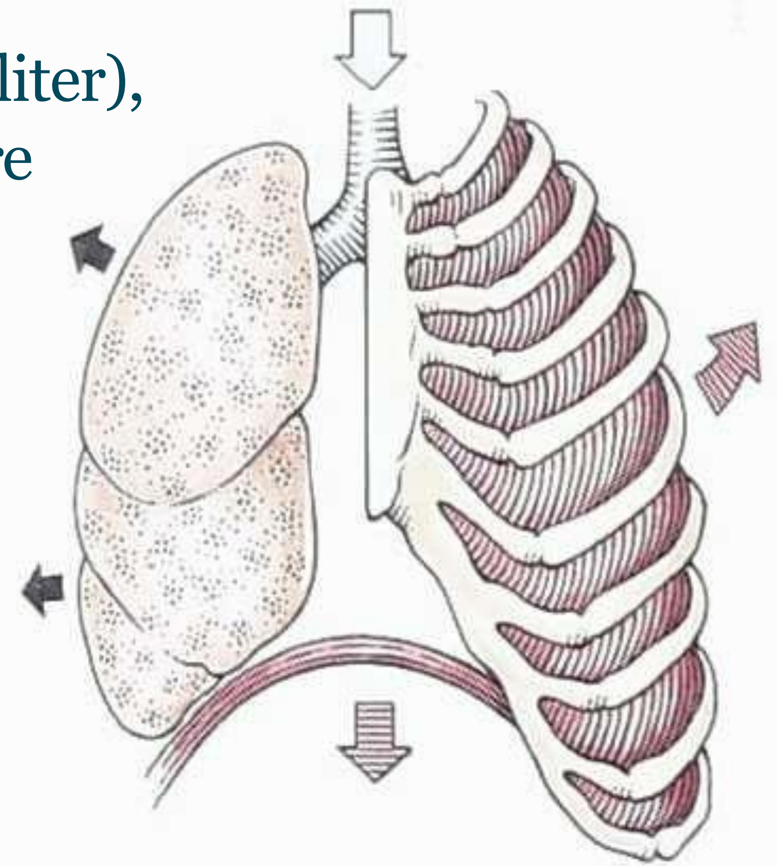
i. Primary Inspiratory: Diaphragm
ii. Accessory Inspiratory: Sternomastoid, Scaleni, Anterior Serrati, Elevators of Scapula & Pectorals
iii. Primary Expiratory: Internal Intercostal muscles
iv. Accessory expiratory: Abdominal muscles Inspiration



Inspiration

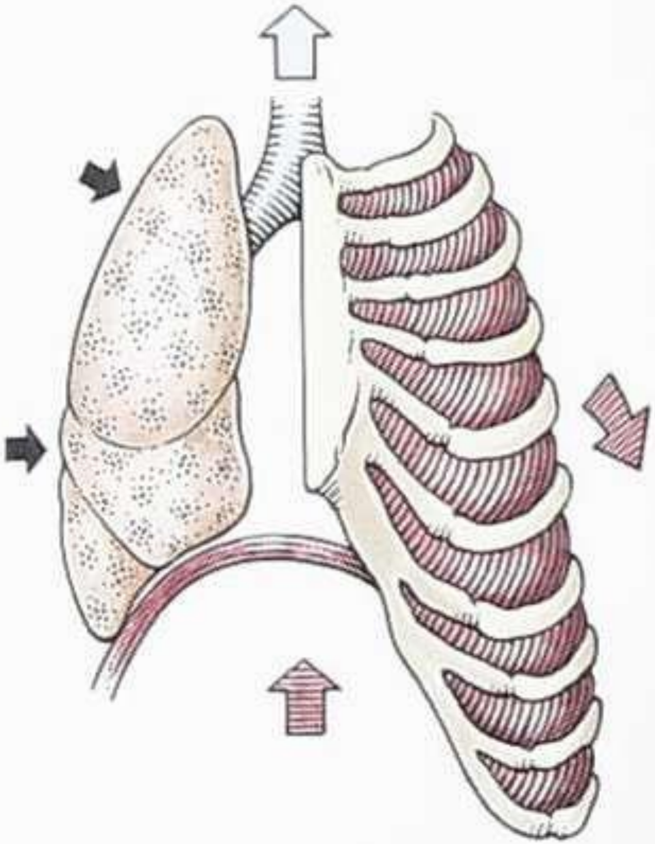
- 1. Diaphragm muscle contracts, increasing thoracic cavity size in the superior-inferior dimension
- 2. External intercostal muscles contract, expanding lateral & anterior-posterior dimension
- 3. INCREASED volume (about 0.5 liter), DECREASED pulmonary pressure (-1 mm Hg), air rushes into lungs to fill alveoli
- Deep/forced inspirations – as during exercise and pulmonary disease

  * scalenes, sternocleidomastoid, pectorals are used for more volume expansion of thorax.



Expiration

- 1. Quiet expiration (exhalation) - simple elasticity of the lungs DECREASES volume INCREASED pulmonary pressure -> movement of air out of the lungs
- 2. Forced expiration - contraction of abdominal wall muscles (i.e. obliques & transversus abdominus) further DECREASES volume beyond relaxed point ----> further INCREASE in pulmonary pressure ---> more air moves out.



Movements of Thoracic Cage

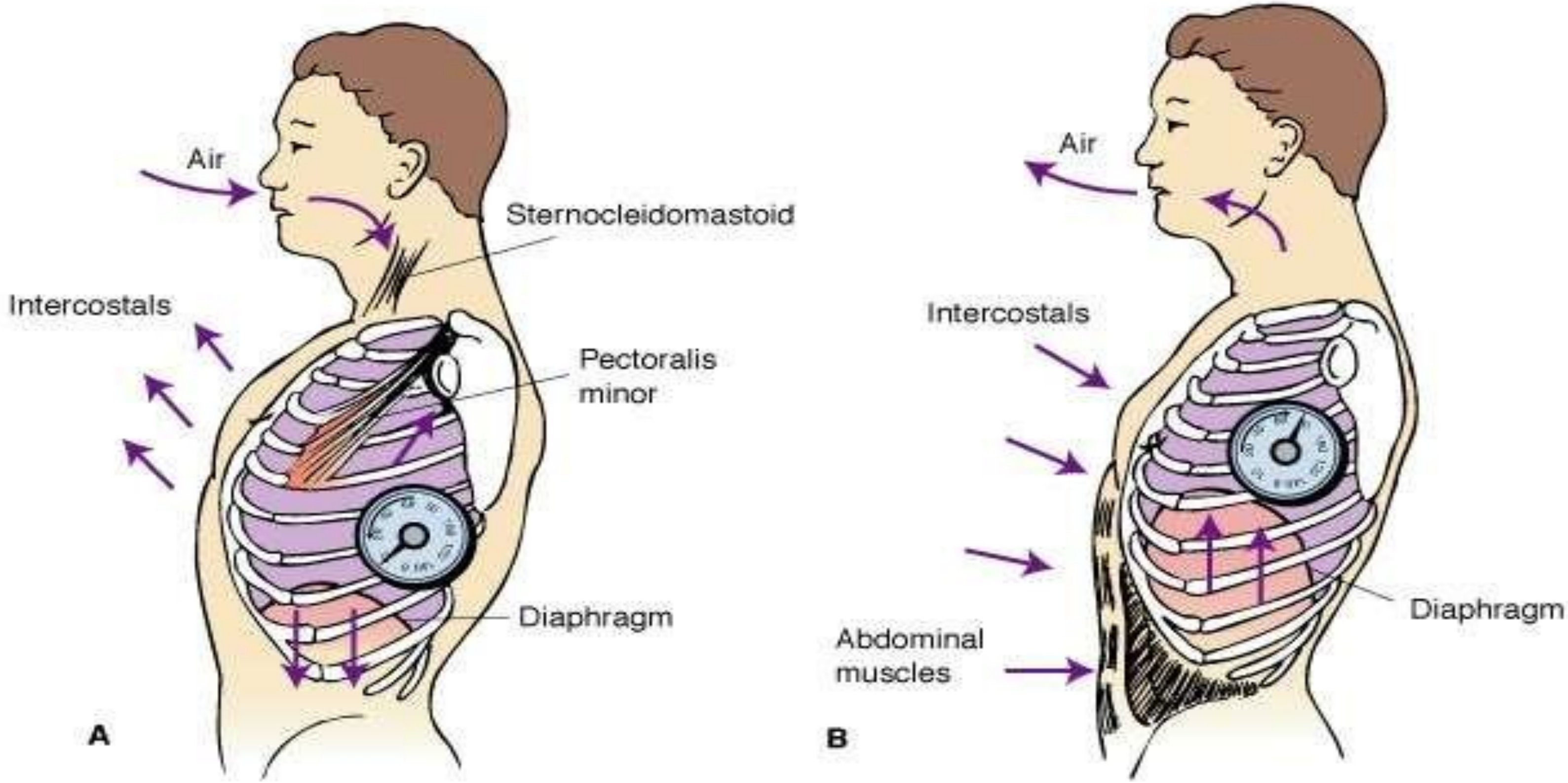


Figure 20-2 Ventilation and thoracic pressure changes. (A) Inspiration. (B) Expiration.

Movements of Lungs

Factors holding lungs AGAINST the thorax wall:

i. Surface tension holding the "visceral" and "parietal" pleura together.

ii. Intrapulmonary pressure is ALWAYS slightly greater than intrapleural pressure by 4 mm Hg.

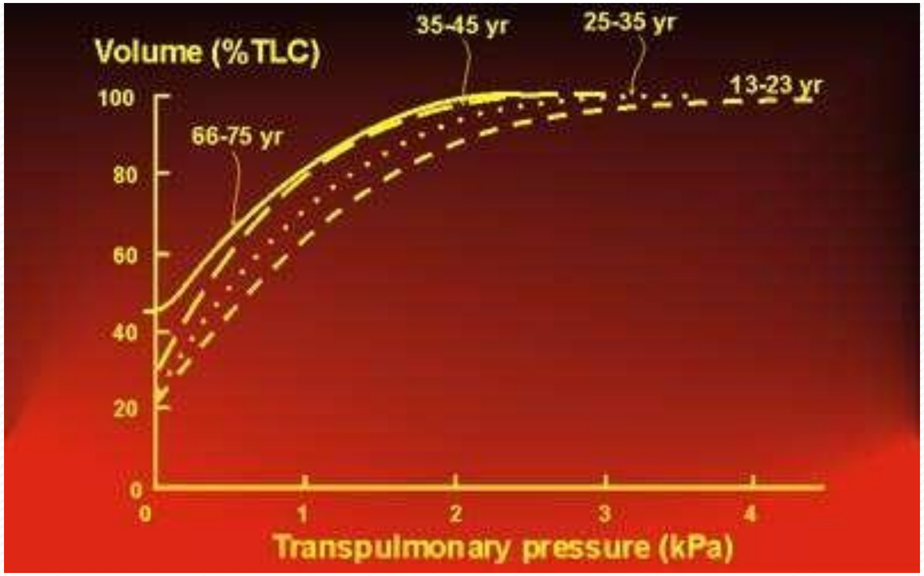
iii. Atmospheric pressure acting on the lungs.

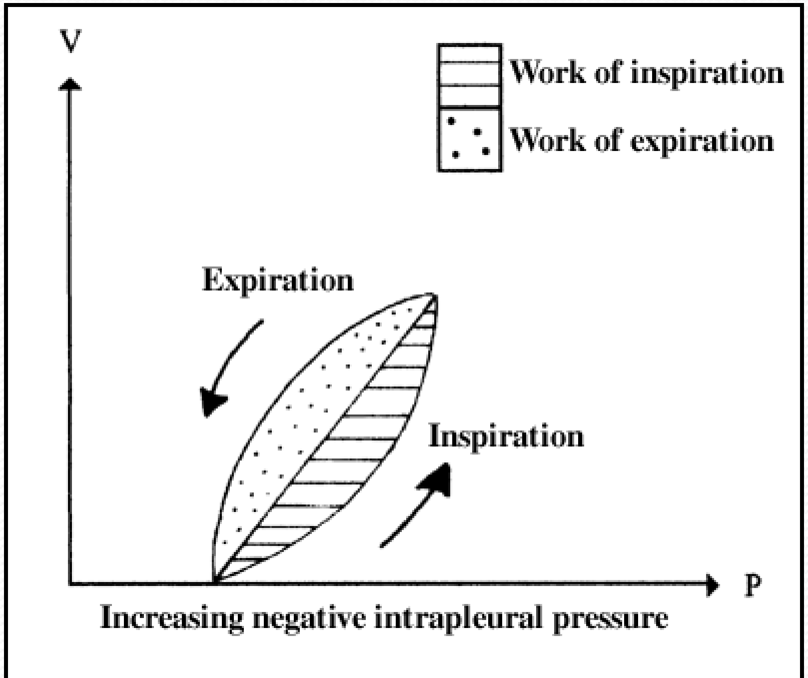
a)Atelectasis (collapsed lung) - hole in pleural "balloon" causes equalization of pressure and collapse of the lung.

b)Pneumothorax - abnormal air in the intrapleural space, can lead to collapsed lung.

Factors facilitating lung movement AWAY from thorax wall

i. Elasticity of lungs allows them to assume smallest shape for given pressure conditions.
ii. Fluid film on alveoli allows them to assume smallest shape for given pressure conditions.





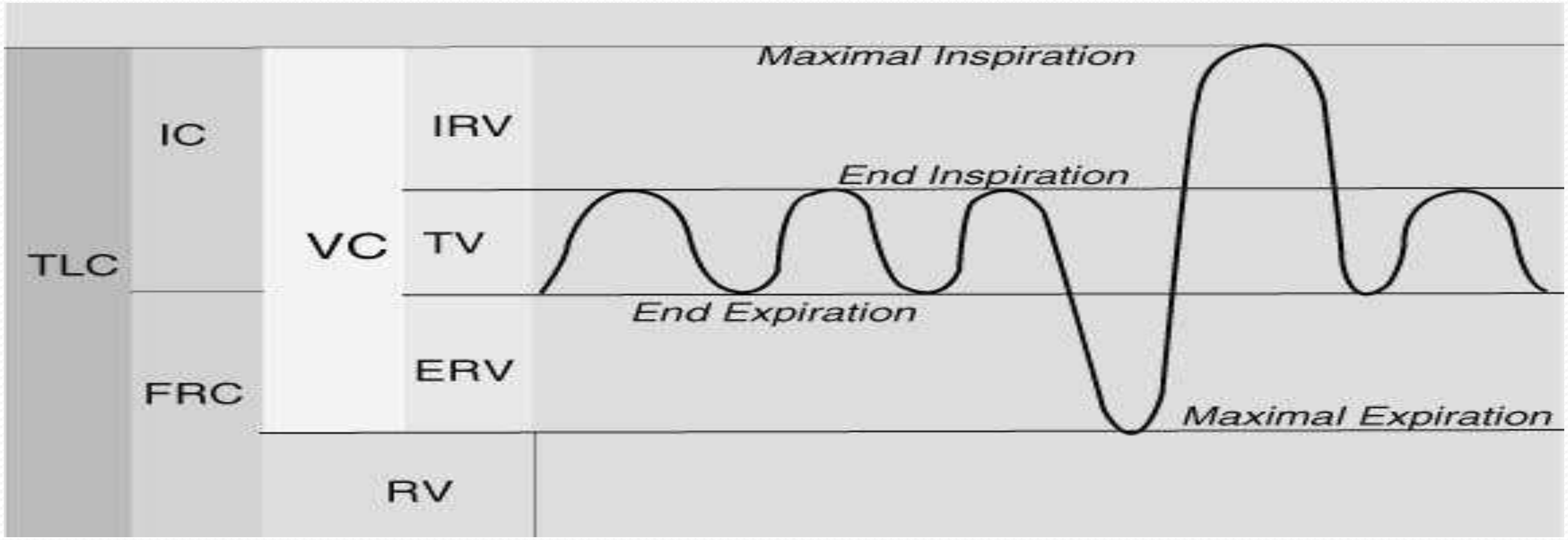
Pulmonary Volumes, Capacities & Function Tests

A. Respiratory Volumes

- 1. Tidal volume (TV) - normal volume moving in/out (0.5 L).
- 2. Inspiratory reserve volume (IRV) - volume inhaled AFTER normal tidal volume when asked to take deepest possible breath (2.1-3.2 L).
- 3. Expiratory reserve volume (ERV) - volume exhaled AFTER normal tidal volume when asked to force out all air possible (1.- 2.0 L).
- 4. Residual volume (RV) - air that remains in lungs even after totally forced exhalation (1.2 L).

B. RespiratoryCapacities

- 1. Inspiratory capacity (IC) = TV + IRV (MAXIMUM volume of air that can be inhaled).
- 2. Functional residual capacity (FRC) ERV + RV (all non-tidal volume expiration).
- 3. Vital capacity (VC) = TV + IRV + ERV (TOTAL volume of air that can be moved).
- 4. Total lung capacity (TLC) = TV + IRV + ERV + RV (the SUM of all volumes; about 6.0 L).

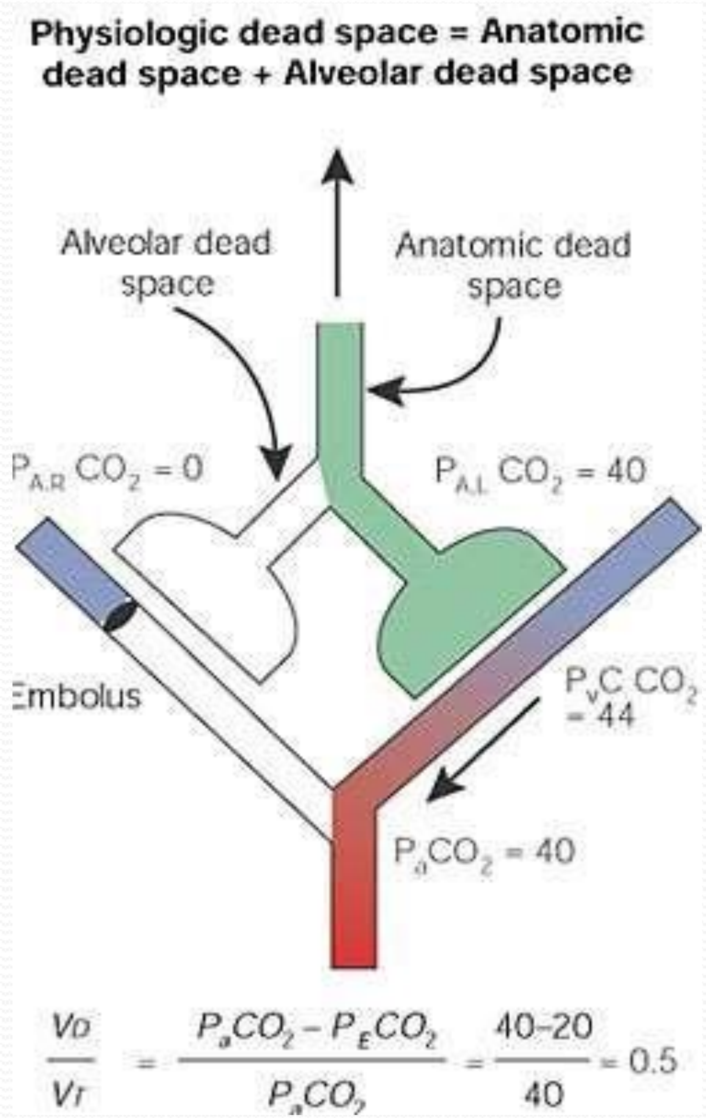


C. Dead Space

- 1. Anatomical dead space – all areas where gas exchange does not occur

- 2. Alveolar dead space – non-functional alveoli.

- 3. Total dead space – Anatomical + Alveolar.



D. Pulmonary FunctionTests

- 1. Spirometer - measures volume changes during breathing.

  a. Obstructive pulmonary disease - increased resistance to air flow (bronchitis or asthma).

  b. Restrictive disorders - decrease in Total Lung Capacity (TB or polio) .

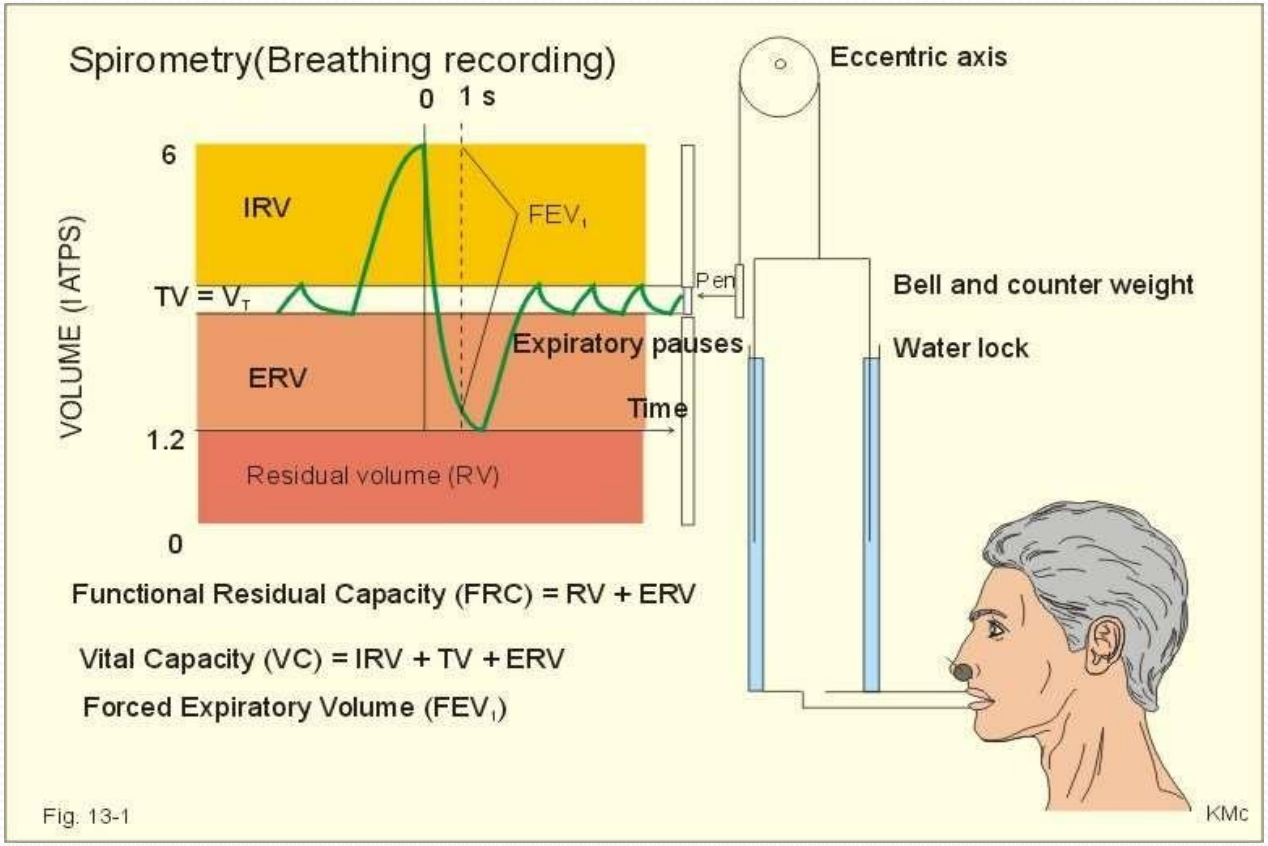
- 2. Minute respiratory volume (MRV) - total volume flowing in & out in 1minute (resting rate = 6 L per minute).
- 3. Forced vital capacity (FVC) - total volume exhaled after forceful exhalation of a deep breath.
- 4. Forced expiratory volume (FEV) - FEV volume measured in 1second intervals (FEV_1...).

E. Alveolar Retention Rate

- AVR = Breath Rate X (TV - Dead space)

  (Normal) AVR = 12 /minute X (500 ml – 150 ml)

  (Normal) AVR = 4.2 L/min



Spirometer

Transport of Gases

- Transport of O_2:

  i) As simple solution (3% i.e. 0.3ml/100ml)

  ii) In Combination with Hb (97%)

- Transport of CO_2:

  i) As dissolved form (7%)

  ii) As carbonic Acid (Negligible)

  iii) As Bicarbonate (63%)

  iv) As Carbamino Compounds (30%)

A. Effects of Temperature

1. HIGHER Temperature ⟶ Decreased Affinity (right)
2. LOWER Temperature ⟶ Increased Affinity (left)

B. Effects of pH (Acidity)

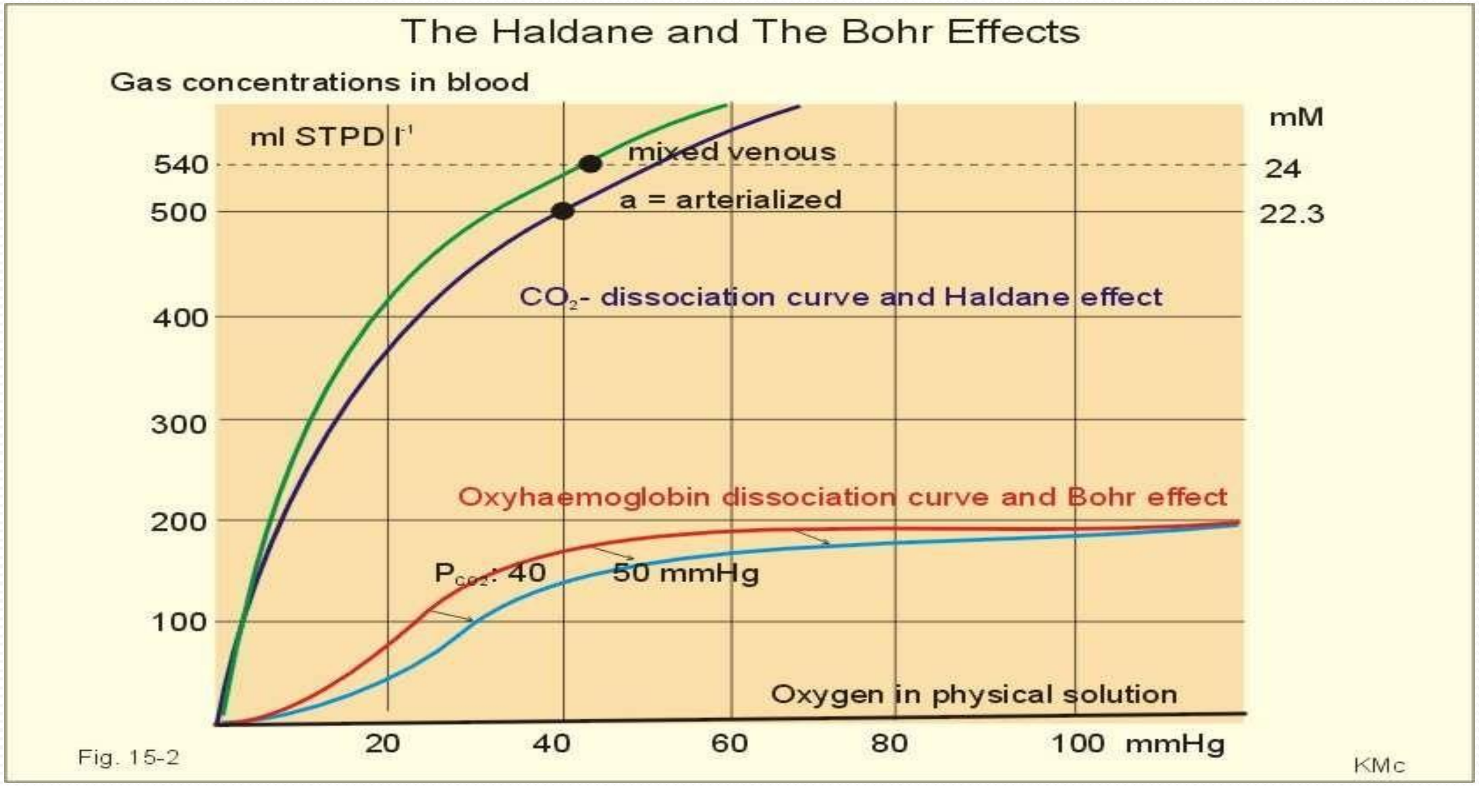
1. HIGHER pH ⟶ Increased Affinity (left)
2. LOWER pH ⟶ Decreased Affinity (right) "Bohr Effect“

C. Effects of Diphosphoglycerate (DPG)

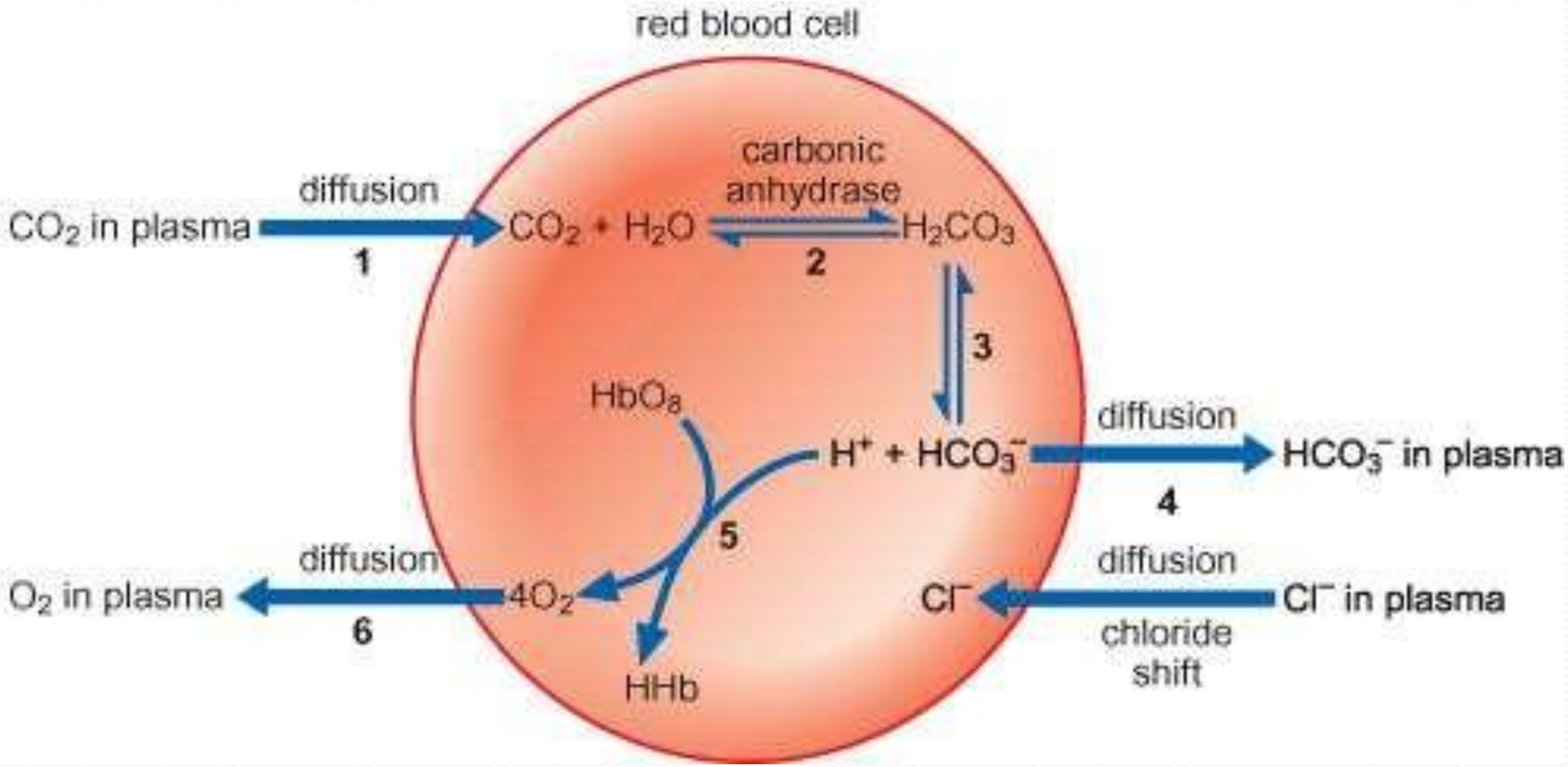
1. DPG - produced by anaerobic processes in RBCs
2. HIGHER DPG > Decreased Affinity (right)
3. Thyroxine, testosterone, epinephrine, NE - increase RBC metabolism and DPG production, cause RIGHT shift.

Carbon Dioxide Dissociation Curve

- Bohr Effect - Formation of Bicarbonate (through Carbonic Acid) leads to LOWER pH (H+ increase), and more unloading of Ox to tissues. Since Hb "buffers" to H^+, the actual pH of blood does not change much.



Chloride Shift - Chloride ions move in opposite direction of the entering/leaving Bicarbonate, to prevent osmotic problems with RBCs



Exchange of Gases

A. External Respiration (Air & Lungs)

1. Partial Pressure Gradients & Solubilities

a. Oxygen: alveolar (104 mm) ---> blood (40 mm)

b. Carbon Dioxide: blood (45 mm) ----> alveolar (40 mm) (carbon dioxide much more soluble than oxygen)

2. Alveolar Membrane Thickness (0.5-1.0 micron) very easy for gas to diffuse across alveoli

b. Edema - increases thickness, decreases diffusion

3. Total Alveolar Surface Area for Exchange

a. Total surface area healthy lung = 145 sq. Meters

b. Emphysema - decreases total alveolar surface area

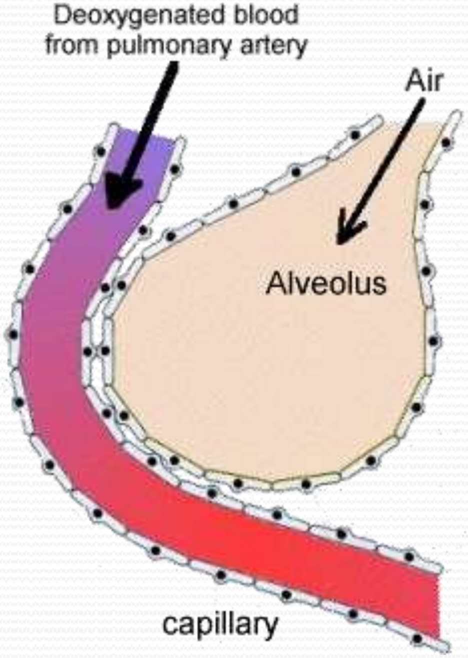
4. Ventilation-Blood Flow Coupling

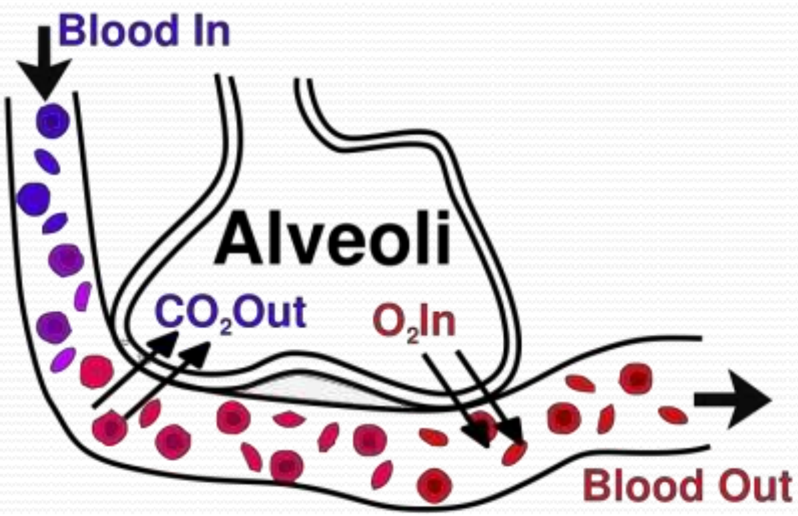
Low O2 in alveolus → vasoconstriction

High O2 in alveolus → vasodilation

High CO2 in alveolus → dilate bronchioles

Low CO2 in alveolus → constrict bronchioles





B. Internal respiration (Blood & Tissues)

- 1. Oxygen: blood (104 mm) ⟶ tissues (40 mm)
- 2. Carbon Dioxide: tissues (>45 mm) ⟶ blood (40 mm)

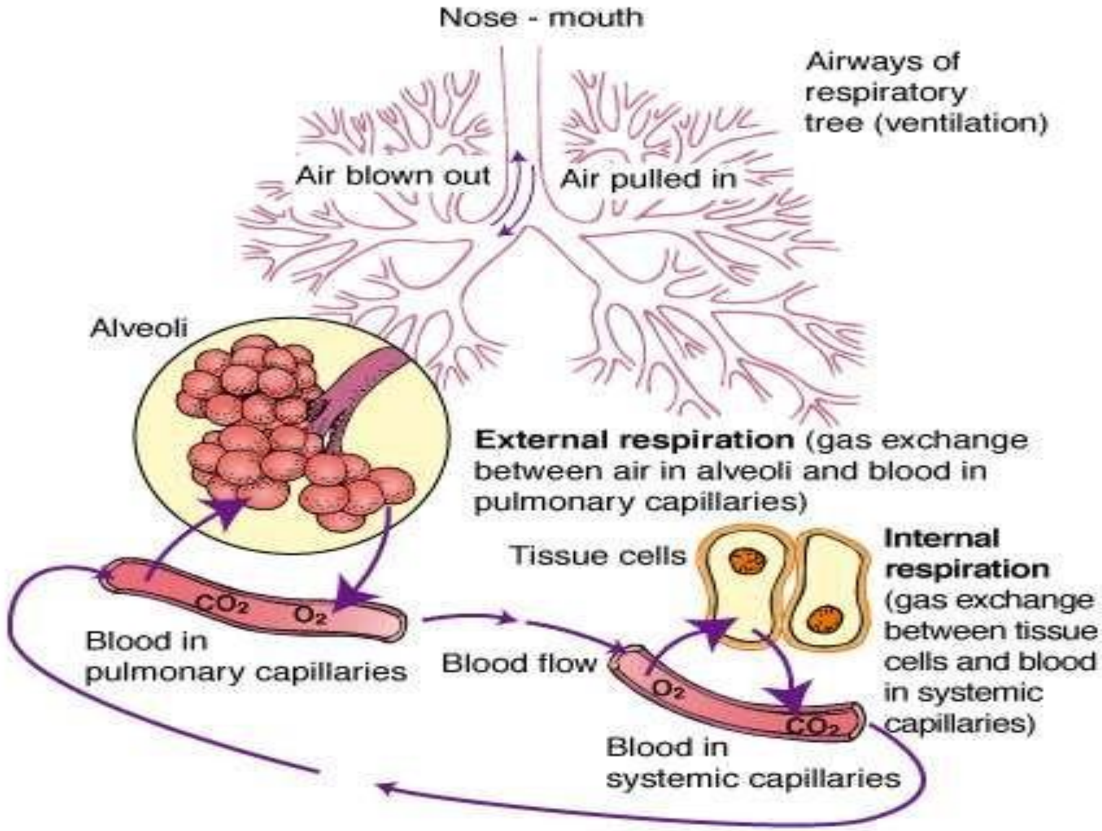


Figure 20-1 External and internal respiration.

Regulation of Respiration

Nervous Mechanism

A. Medullary Respiratory

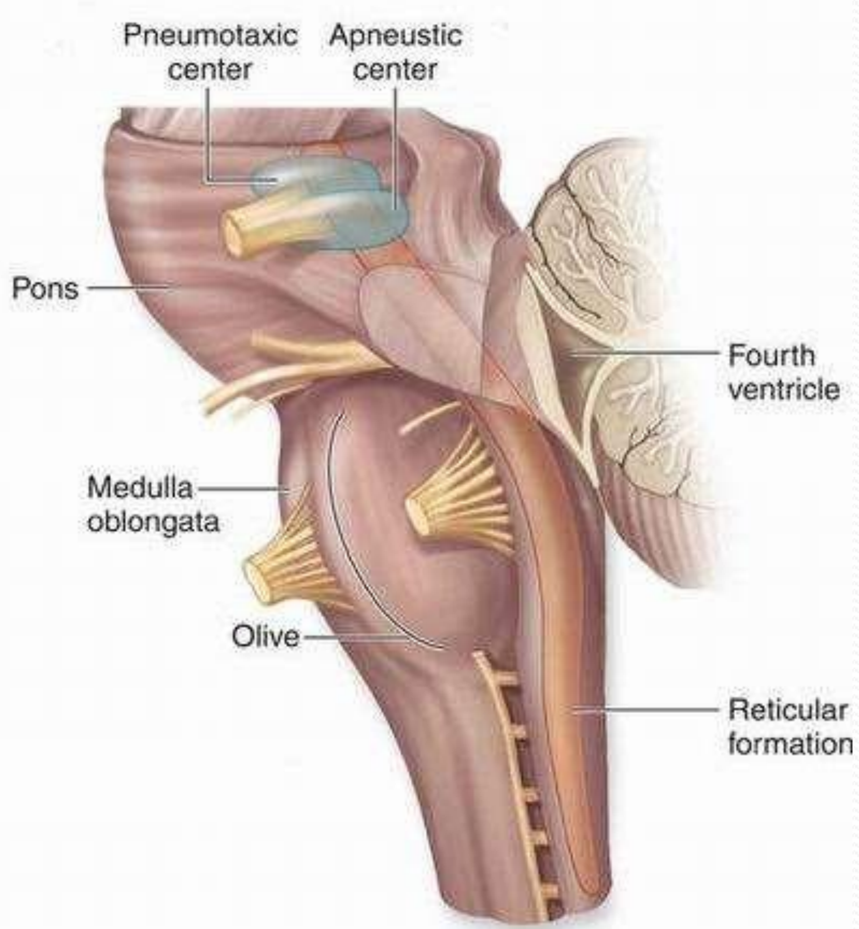
- **Inspiratory Center (Dorsal Resp Group - rhythmic breathing**
- Phrenic nerve, Intercostal nerves , diaphragm +external intercostals

- **Expiratory Center (Ventral Resp Group - forced expiration)**
- Phrenic nerve, Intercostal nerves, Internal intercostals + abdominals (expiration)

1. Eupnea - normal resting breath rate (12/minute)
2. Drug overdose - causes suppression of Inspiratory Center

B. Pons Respiratory Centre

- 1. Pneumotaxic center - slightly inhibits medulla, causes shorter, shallower, quicker breaths
- 2. Apneustic center – stimulates the medulla, causes longer, deeper, slower breaths



C. Control of Rate & Depth of Breathing

- 1. Breathing rate - stimulation/inhibition of medulla.
- 2. Breathing depth - activation of inspiration muscles.
- 3. Hering-Breuer Reflex - stretch of visceral pleura that lungs have expanded (vagal nerve).

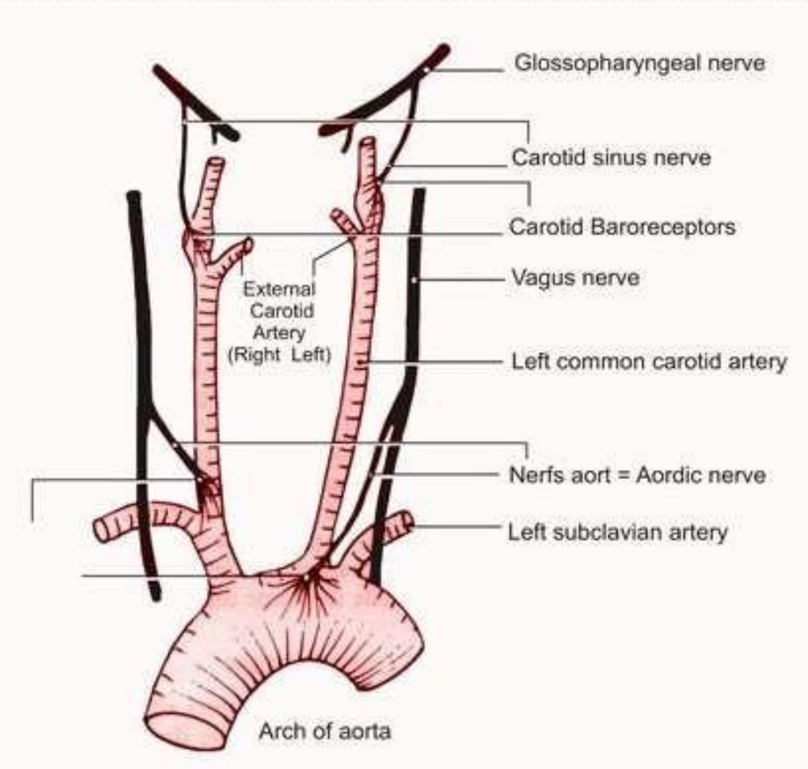
D. Hypothalamic Control - emotion + pain to the medulla

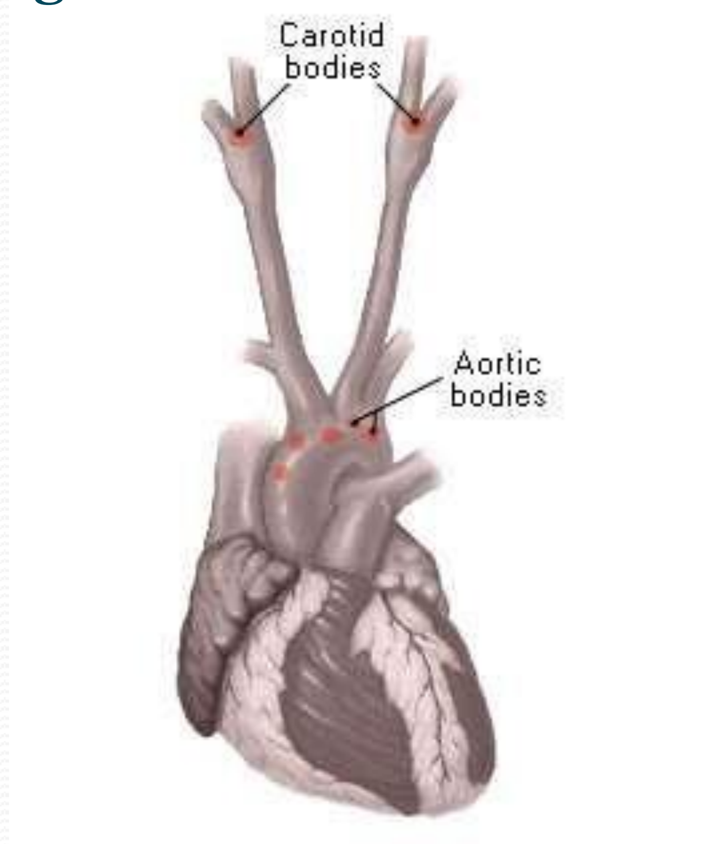
E. Cortex Controls (Voluntary Breathing) - can override medulla as during singing and talking

Chemical Mechanism

A. Chemoreceptors

- 1. Central chemoreceptors - located in the medulla
- 2. Peripheral chemoreceptors - large vessels of neck





B. Overview of Chemical Effects

Chemical	Breathing Effect
Increased CO2 (more H^+)	Increase
Decreased CO2 (less H^+)	Decrease
Slight decrease in O2	Effects CO_2 system
Large decrease in O2	Increases ventilation
Decreased pH (more H+)	Increase
Increased pH (less H+)	Decrease

EXERCISE EFFECT ON THE RESPIRATORY SYSTEM

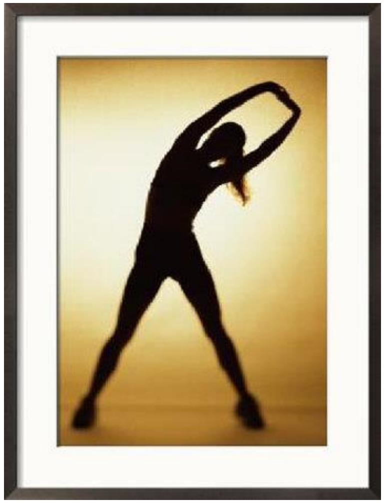
Exercise is the most influential physiological stress on breathing

How is increased ventilation accomplished?

Exercise increases the breaths/minute
Exercise increases the amount of air in each breath (tidal volume)

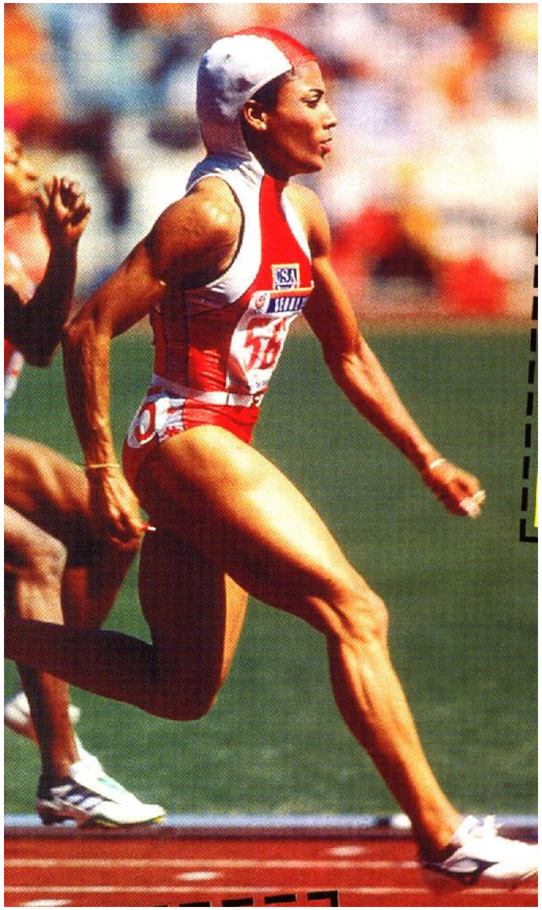
During light exercise

Ventilation increases linearly with oxygen uptake and carbon dioxide production
This increase in ventilation is accomplished more by increased ***tidal volume*** (breathing deeper in and out)



During higher exercise levels

Ventilation is increased more by
increased breathing frequency
This will keep the blood saturated with oxygen because the blood is in the alveoli capillaries long enough for complete diffusion of gases



Steady rate (moderate) exercise

sufficient oxygen is supplied to muscles
due to increased oxygen up take, there is little, or no, build up of lactic acid in the muscles
some lactate will be produced and removed by the blood stream
lactic acid is neutralized in the blood (this reaction produces carbon dioxide as a by-product)
increased carbon dioxide in the blood will stimulate increased ventilation
Increased ventilation is accomplished by ***both increased tidal volume and frequency***

How does pulmonary ventilation (breathing) increase during exercise?

1. During light exercise (walking)?

   <u>By increasing the tidal volume (breathing deeper)</u>

2. During intense exercise (sprinting)?

   <u>By increasing the frequency of breathing</u>

3. During steady state exercise (jogging)?

   <u>By increasing both the tidal volume and the frequency of breathing</u>

The Energy Cost of Breathing

At rest and with light exercise

the energy cost of breathing is minimal (4% of energy)

During intense exercise

the energy use may increase from 10-20% of total energy expenditure

In a person with respiratory disease

the work of breathing itself during exercise may be exhausting

The Effects of Cigarette Smoking

Cigarette smoking has little long term effect on athletes *in the initial years*

Cigarettes have *an immediate effect* of airway resistance (as much as 3 times) that lasts about 35 minutes after only 15 puffs

This could be significant in vigorous exercise

Ventilation Adaptations with Training

Minimal exercise

ventilation during exercise improves with training (due to increased alveolar ventilation and increased tissue oxygen uptake)

Sub-maximal exercise

The percentage of energy used in breathing is reduced after training only 4 weeks

Tidal volume increases

Breathing frequency is reduced

This results in increased amounts of oxygen extracted from inspired air

Untrained expired air contains 18% oxygen

Trained expired air contains 14-15% oxygen

This is important because

It reduces the fatiguing of the ventilation musculature
The freed up oxygen (not used by the respiratory muscles) can be available for the exercising muscle