بسم الله الرحمن الرحيم

# Physiology Team 436
# Respiratory Block

# REVIEW FILE

أنت تحمل علماً بين ثناياهُ شفاء العالمين

فـ توكّل على الله ولا تعجز،وتذكر قوله تعالى:

(إِنَّا لَا نُضِيعُ أَجْرَ مَنْ أَحْسَنَ عَمَلًا )


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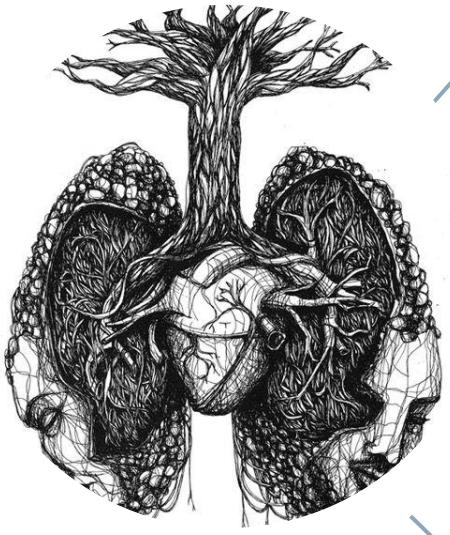
دعواتكم لنا بالتوفيق

This work is done by students , so if there are any mistakes please inform us .

# Lecture #1
## Functions and Organization of the Respiratory System



consists of:

- Passages
- Centers
- Muscles

Main function :

- Provide oxygen to tissues.
- Remove CO2 .

Other Functions:

- Gas exchange (respiratory function)
- Phonation
- Pulmonary defense
- Angiotensin
- Regulating( acid-base)
- Secretion( surfactant).. Will be discussed latter

Pulmonary defense

- macrophage
- Alpha-1 antitrypsin
- Ig A

# Respiratory Passages (Airways)

## Conductive Zone

### Structures:

Starts from nose to the end of terminal bronchioles.

### Function:

- Help in warming, humidification, filtration of inspired air.
- Contains the olfactory receptors for smell sensation.
- Conducts the sound during speech
- Protective function by cough and sneezing reflex.

## Respiratory Zone (Respiratory unit)

### Structures:

Respiratory bronchioles

Alveolar ducts

Alveolar sacs

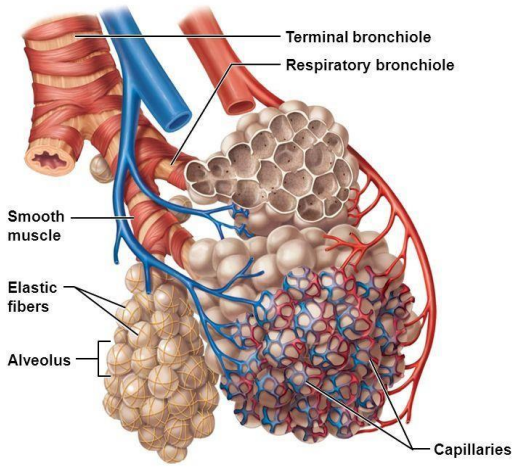
Alveoli

Atrium

### Function:

Gas exchange

Figure 22.9a Alveoli and the respiratory membrane.

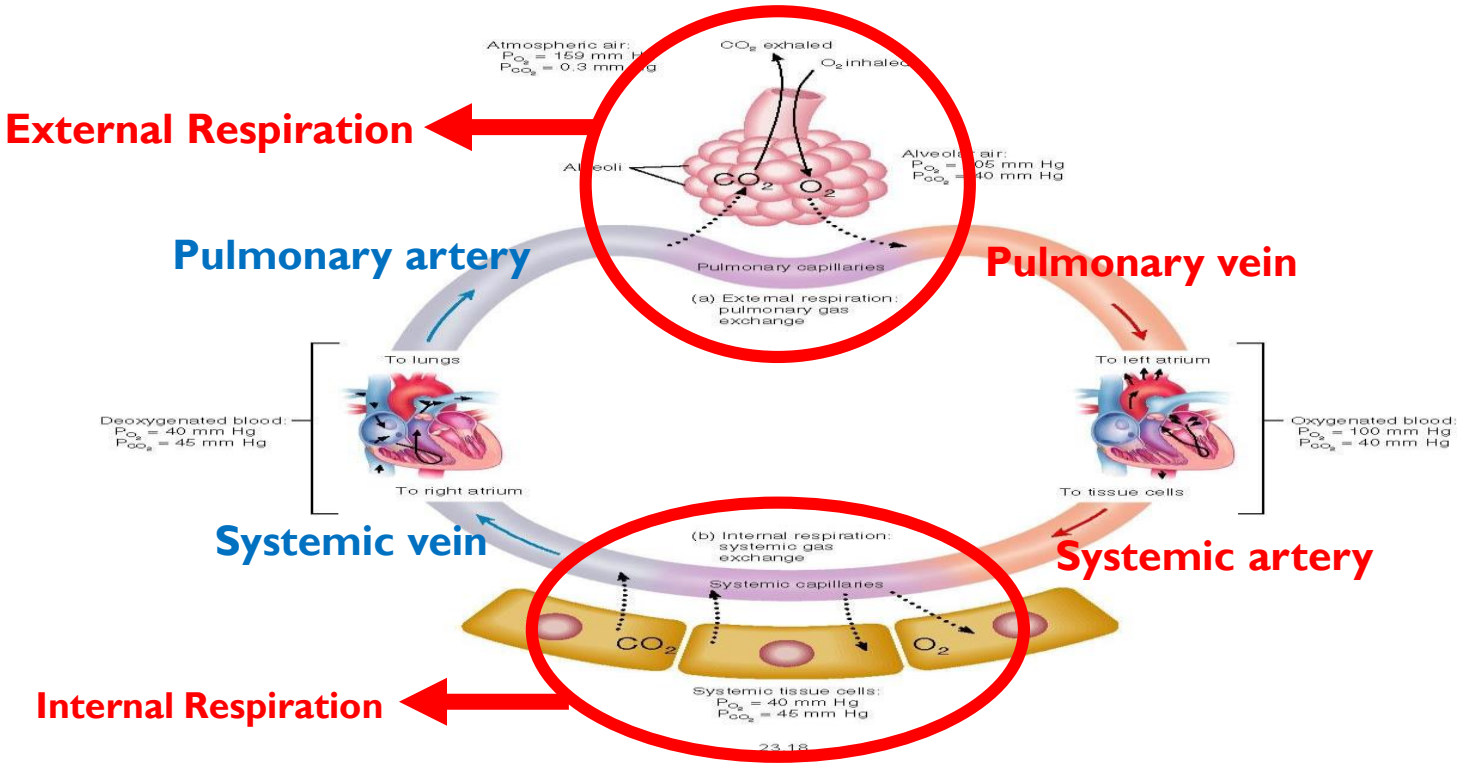



(a) Diagrammatic view of capillary-alveoli relationships

# External & Internal Respiration

- As we know there is Internal respiration and external respiration which provide O2 to all the body and remove the CO2.
- External respiration : is the gas exchange at the alveolar level or the lung level.
- Internal respiration : is the gas exchange at the cell level or tissue level.

| Pulmonary ventilation | Diffusion | Transport |
|---|---|---|
| Inward and outward movement of air between lung and atmosphere air. | When gases exchange during diffusion of O2 and CO2 between the alveoli and the pulmonary capillary blood. | Transport of O2 and CO2 in blood and body fluids to and from the cells. |



# Lining Cells of The Alveoli

**Type I Alveolar Cells**

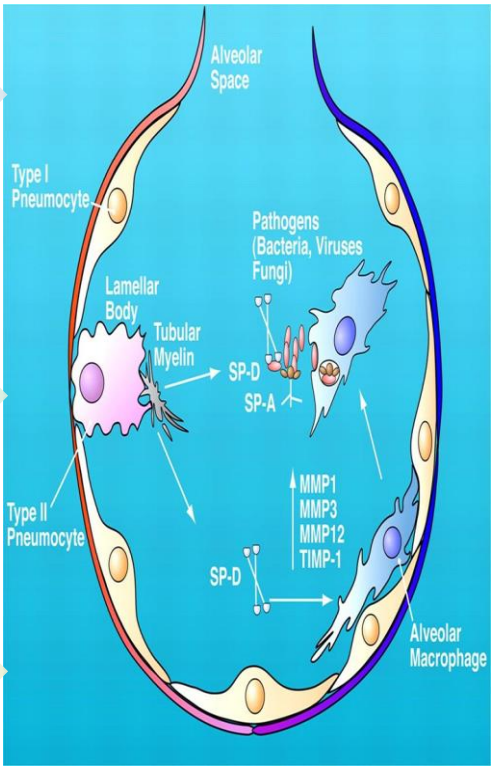
- Also called Type I pneumocyte.
- Participates in the respiratory (pulmonary) membrane.
- High percentage of surface area.
- Surrounded by capillaries and participates in gas exchange.

**Type II Alveolar Cells**

- Also called Type II pneumocyte.
- Secretes surfactant.

**Alveolar Macrophages**

- Engulfs the foreign bodies that reach the alveoli.

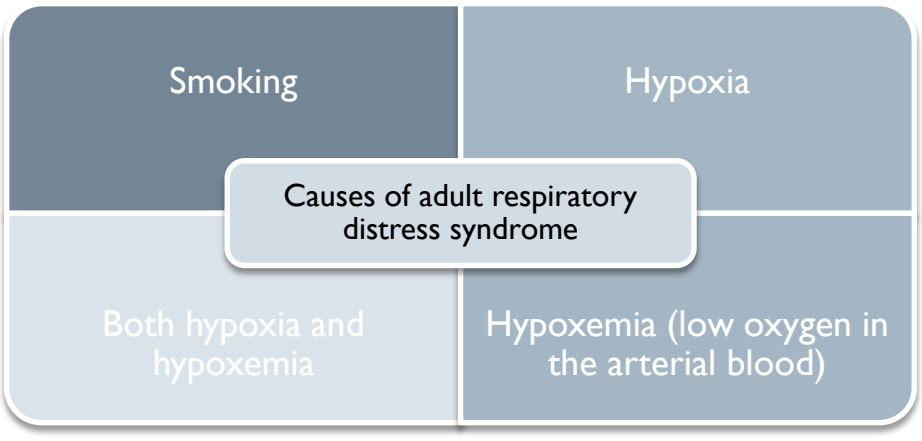



# Surface Tension

$H_2O$ molecules at the surface are attracted to each other by attractive forces that resist distension called **surface tension.**
It is the attractive force between adjacent water droplets.
Surface tension increases (water droplets stick to each other) in contact with air.
Surface tension tends to oppose alveoli expansion.
Increased surface tension will constrict the alveoli.
In respiratory functions we decrease the surface tension of water molecules .
Pulmonary surfactant reduces surface tension.

# Surfactant (Important)

- Surfactant is a complex substance containing phospholipids and a number of apoproteins.
- It lines the inside alveoli, which separates the air found in the lumen from water droplets on cells.
- It is secreted by Type II alveolar cells. The earliest detection from fetal alveoli (of surfactant) begins between the (surfactant secretion starts at the) $6^{th}$-$7^{th}$ month ($24^{th}$-$28^{th}$ week) but this could be delayed in others to week 35 of intrauterine life.
- At week 35 surfactant is mature and begins to function.
- Babies born before week 35 can be considered premature (before full maturation of surfactant) have immature surfactant, so the baby is vulnerable to alveolar collapse (RDS).
- Full term pregnancy: 38 ∓2 weeks.
- Premature babies: born between weeks 28 – 35 (approximately/no exact number).
- Importance of surfactant is to reduces surface tension throughout the lung, prevents alveolar collapse, decreases airway resistance to inflation and decreases the work of breathing and increases surface area for normal breathing.



# Respiratory Distress Syndrome

## In Adult

Smoking in adult, hypoxia or hypoxemia (low oxygen in the arterial blood) or both, decrease the secretion of surfactant and cause adult respiratory distress syndrome. All causes of adult respiratory distress syndrome decrease the secretion of surfactant, or destruct surfactant, which causes the syndrome.

## In Neonatal

known as: Hyaline Membrane Disease.

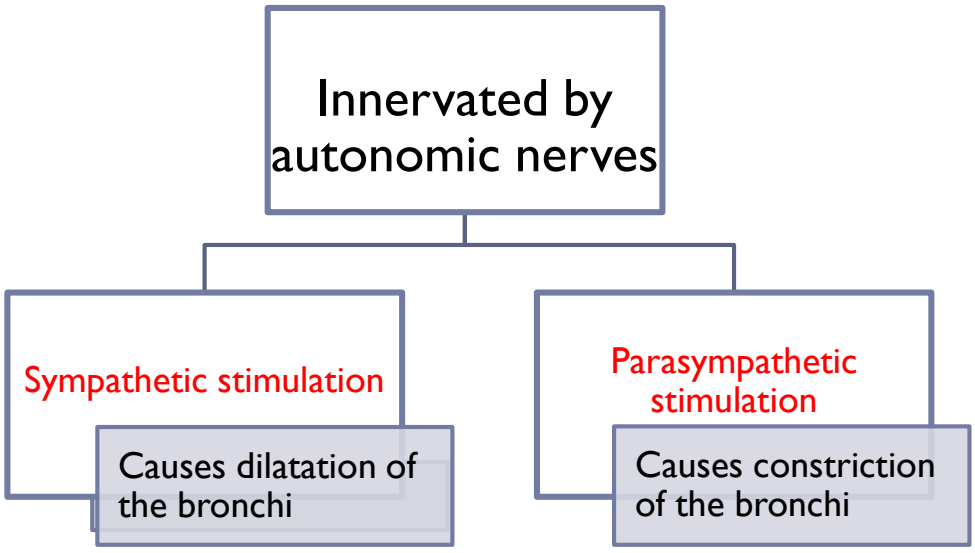
Deficiency in premature babies causes respiratory distress syndrome of the new born (RDS) which is a hyaline membrane disease.

Here the surfactant is lacking.

In the developing fetus. Infants born before week 24 will *never* have surfactant. Without surfactant, small alveoli have increased surface tension and increased pressures, and will collapse (**atelectasis**). Collapsed alveoli are not ventilated and therefore, cannot participate in gas exchange.

# Innervations of Lungs and Bronchi

Locally secreted factors: Histamine, slow reacting substances of anaphylaxis (SRSA) by mast cells, due to allergy (as in patients with asthma) often cause bronchiolar constriction and increased airway resistance.



# Lecture#2

## (Mechanics of Pulmonary Ventilation)

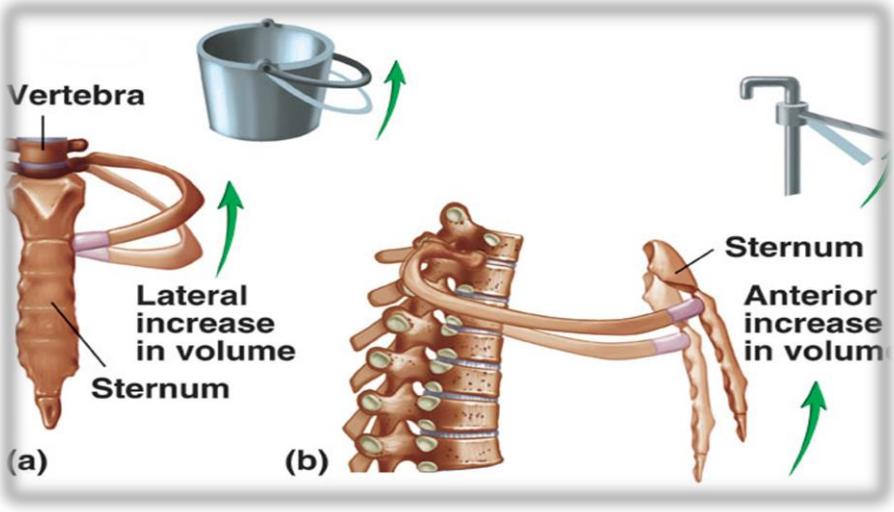
| Lungs are expanded and contracted during breathing: | | |
|---|---|---|
| | **Expansion of the lungs (in inspiration )** | **Contraction of the lungs (in expiration)** |
| *Diaphragm* | Downward movement of the diaphragm will lengthen the chest cavity (vertically). | Upward movement of the diaphragm will shorten the chest cavity (vertically). |
| *Ribs* | By elevation of the ribs, the anteroposterior diameter of chest cavity will increase. | By depression of the ribs will decrease the anteroposterior diameter. |

### Elevation of the ribs will lead to two things:

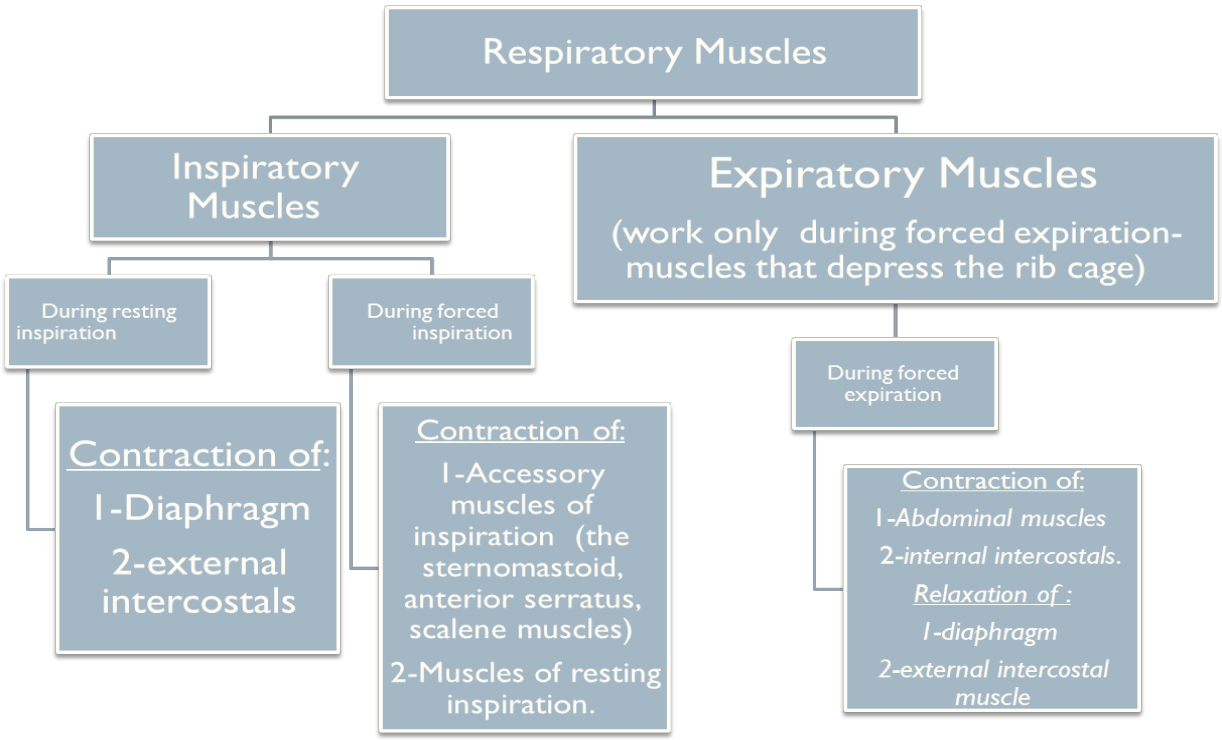
1. Increasing anteroposterior diameter of chest cavity. (b)
2. Increasing horizontal diameter of the chest cavity. (a)

### The chest has three diameters:

1. Vertical diameter.
2. Transverse diameter.
3. Anteroposterior diameter.



# Respiratory Muscles




- The diaphragm will increase the longitudinal diameter.
- The external intercostals will increase the transverse diameter and pull the ribs outward.
- Accessory muscles will increase the anteroposterior diameter and elevates the sternum and clavicle.
- Inspiration and expiration could be either resting or forced.
- Resting and forced (deep) inspiration is active. It requires muscle contraction and consumes energy.
- Resting expiration is passive while forced (deep) expiration is active. (forced expiration - muscles depress the rib cage)
- There are no muscles for resting expiration, It is a passive process that depends on the recoil tendency of the lung.

# Respiratory Muscles

**INSPIRATORY MUSCLES:**
All the muscles that expand (elevate) the chest cage are classified as muscles of inspiration **(FORCED "DEEP" AND "RELAXED")**

**EXPIRATORY MUSCLES:**
All the muscles that depress the chest cage are classified as muscles of expiration **(FORCED ONLY)***

**RELAXED INSPIRATION:** During resting breath. Contraction of the :

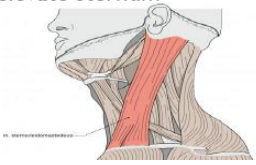
- **Diaphragm muscle.** - Goes down to expand the chest cage. Increase the vertical dimension.
- **External intercostals** – Moves the ribs outward. Increase the transverse dimension.

**DEEP INSPIRATION** During exercise : (relaxed inspiration muscles + Accessory muscles) During deep forceful inhalation accessory muscles of inspiration participate to increase size of thoracic cavity: "Are Bellow"

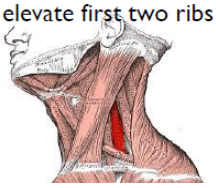
**DEEP EXPIRATION:**
-Thorax expiration during forceful breathing is active process. -Muscles of exhalation increase pressure in abdomen and thorax :

- **Abdominal muscles**
- **Internal intercostals** – Moves the ribs inward. Decrease the transverse dimension.

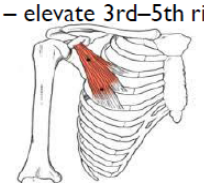
**A. Sternocleidomastoid**
– elevate sternum



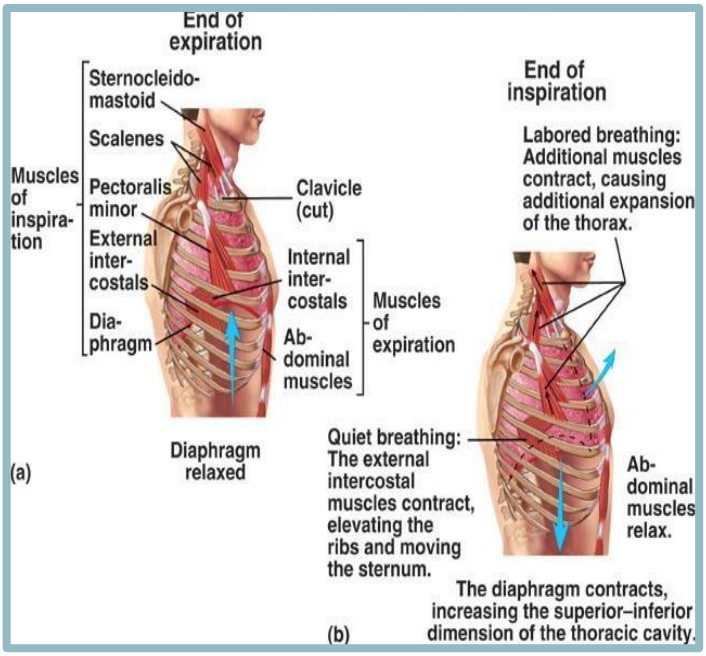
**B. Scalenes**
– elevate first two ribs

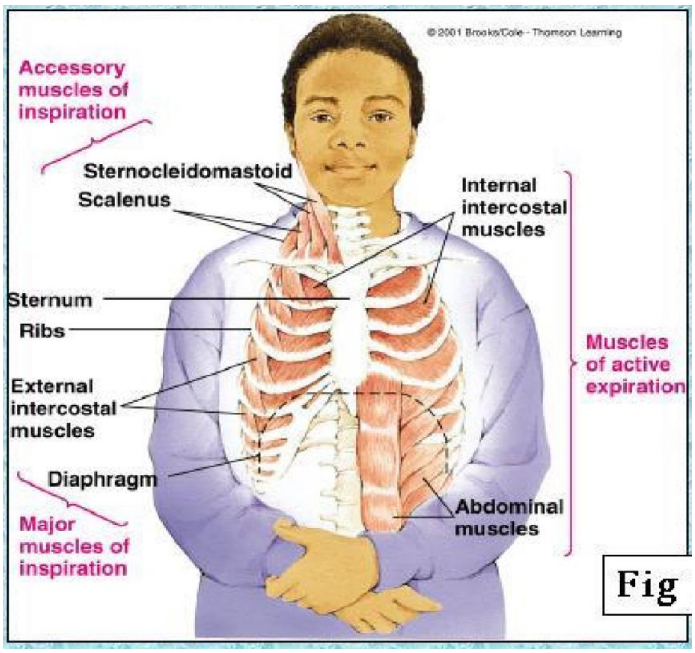


**C. Pectoralis minor ***
– elevate 3rd–5th ribs



***Pectoralis major** (mentioned in anatomy), increases the AP diameter when the arm is fixed.
***Resting expiration** is the passive relaxation of diaphragm, no muscle contraction necessary.

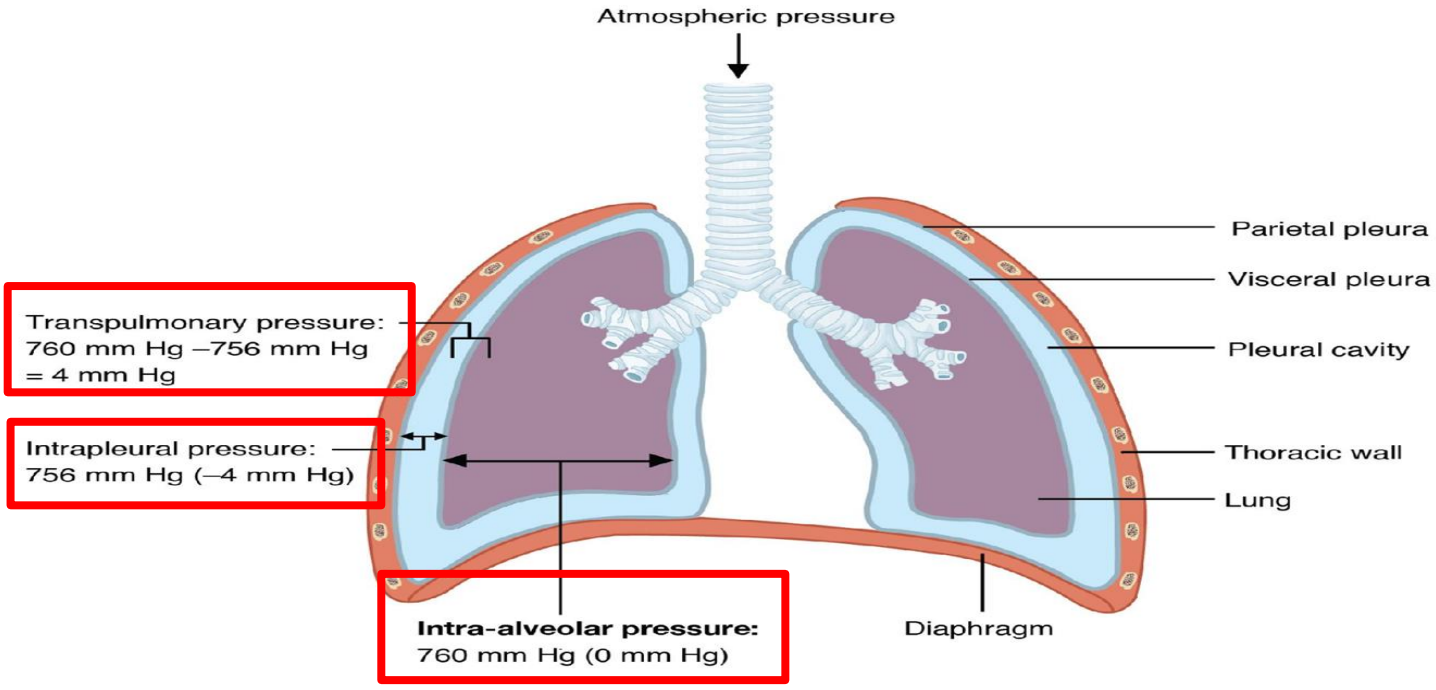



Fig

# Pressures in the Lungs

- Air will flow from a region of **high** pressure to one of **low** pressure-
- The bigger the difference, the faster the flow.
- During inspiration, the pressure in the respiratory passages is **less** than the atmospheric pressure.
- Contraction of inspiratory muscles will **increase** the **volume** of the chest which will **decrease** the **pressure** in the respiratory passages.
- There are 12 cycles per minute (respiratory rate) consisting of inhalation then expiration then pause.
- During pause the pressure inside is **equal** to the pressure outside, meaning there is **no** movement of air.
- Inward movement of air will fill the space, which will return the pressure back to the atmospheric pressure and stop inspiration.
- When inspiration stops, recoil will happen which will **decrease** the **volume**, so the **pressure** will **increase** and the air will move outward. We will have expiration.
- The atmospheric pressure 760 mm HG. (To make it easier we will refer to it as 0).
- If we have a pressure less than the atmospheric pressure we will refer to it as (–).
- If we have a pressure higher than the atmospheric pressure we will refer to it as (+).

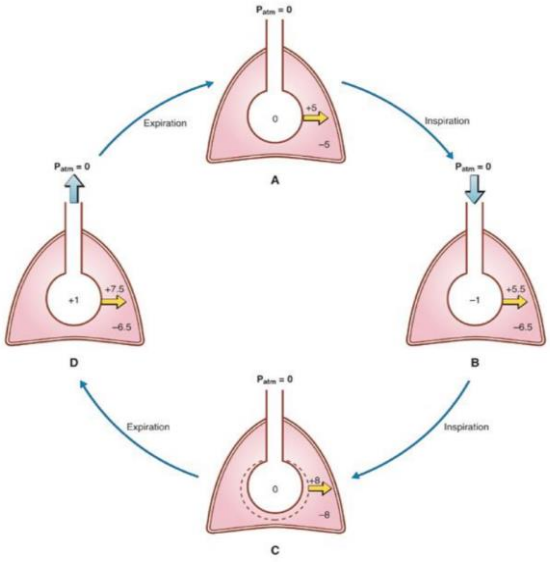


# Pressures changes in the lungs during breathing

| Pressures changes in the lungs during breathing. | | |
|---|---|---|
| **Type** | **Explanation** | **Value** |
| **INTRA-ALVEOLAR "INTRAPULMONARY" PRESSURE (Palv)** | Alveolar pressure is the pressure of the air inside the lung alveoli. | Between breathes (Resting breath)= Zero<br>During inspiration = (-1 mmHg)<br>At the end of inspiration = Zero<br>Pressure During expiration = (+1 mmHg) |
| **INTRAPLEURAL PRESSURE (IPp)** | Pleural pressure is the pressure of the fluid in the thin space between the lung pleura and the chest wall pleura.<br>WHY NEGATIVE:<br>A. The lung's elastic tissue causes it to recoil, while that of the chest wall causes it to expand. Because of these 2 opposing forces the pressure in the pleural cavity becomes negative.<br>B. The pleural space is a potential space, empty due to continuous suction of fluids by lymphatic vessels. | Intrapleural pressure is (-5) cm H2O during resting position between breaths<br>it ecomes more negative (-7.5) cm H2O during resting inspiration.<br>Forced Inspiration : -20 to -40 cmH2O<br>Forced Expiration : +30 cmH2O |
| **TRANSPULMONARYPRESSURE (TPp)**<br>**(the recoil pressure) & (The extending pressure)** | The difference between the alveolar pressure (Palv) and the pleural pressure (Ppl).<br>It prevents lung collapse.<br>The bigger the volume of the lung the higher will be its tendency to recoil.<br>**TPp = Palv-Ppl** | Example of transpulmonary pressure calculation during inspiration: -1 + 7.5 = 6.5 |

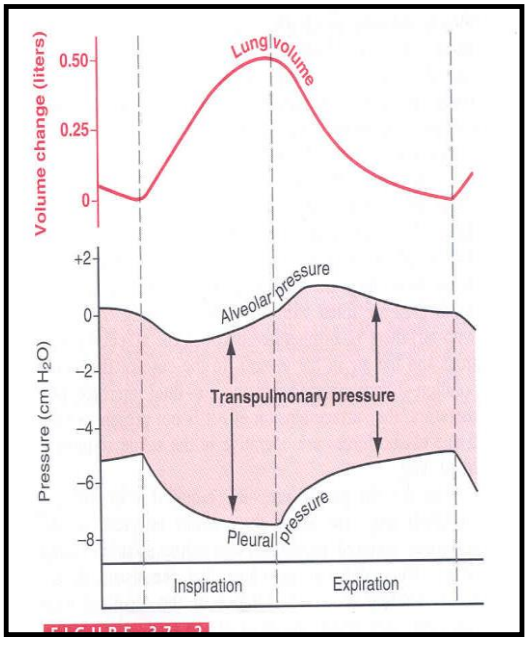
# Pressure and Volume Relationships in a Single Respiratory Cycle

- **A: during rest-**: Intrapleural pressure is -5cmH2O while the Intra-alveolar pressure is 0mmHg.
- **B:during inspiration-** the intra-alveolar pressure becomes -1mmHg, then the air enters the alveoli ,while the Intrapleural pressure becomes -6.5cm H2O(more negative).
- **C:at the end of inspiration-**: Air flow stops since the Intra-alveolar pressure= the Atmospheric pressure(0). While the intrapleural pressure becomes -8cm H2O (more negative).
- **D:during expiration-**: the intra-alveolar pressure becomes +1mmHg, then the air exits alveoli into the atmosphere while the Intrapleural pressure becomes -6.5cmH2O (it isincreased).




# (Compliance of the Lung)

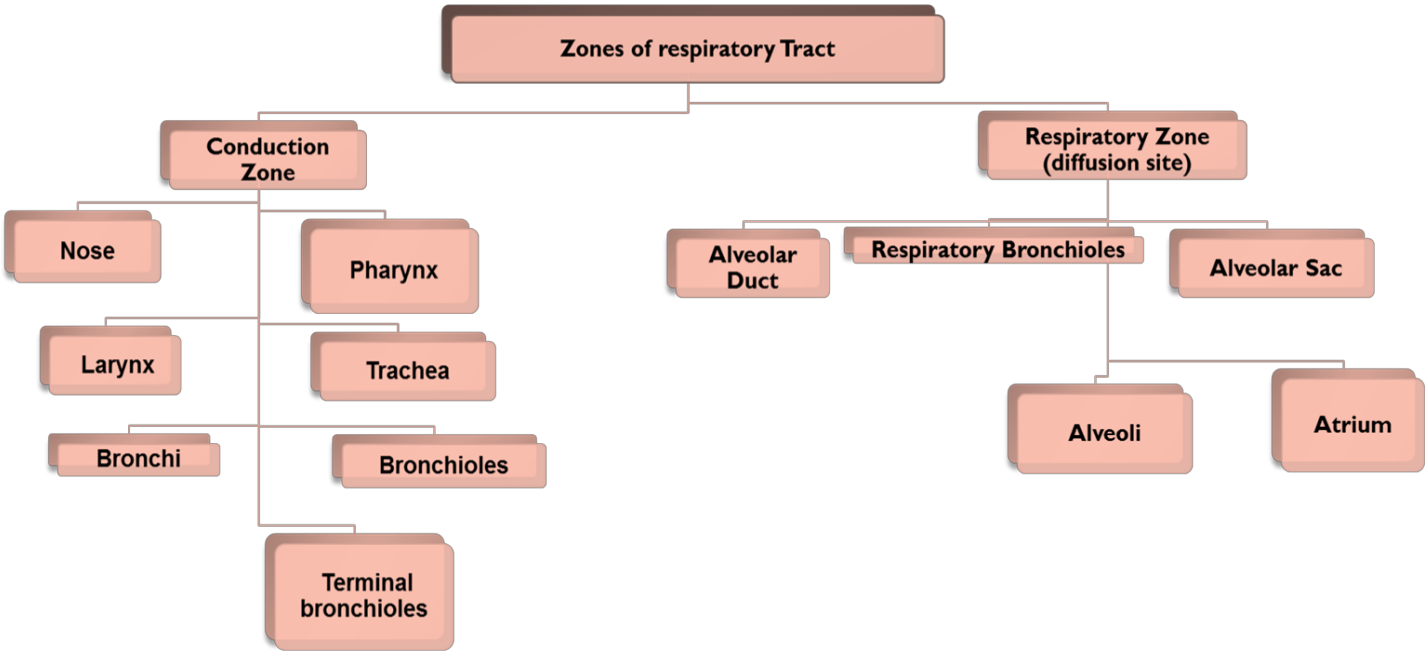
- Compliance (resilience) of the Lungs.
- Increased TPP = increased lung volume.
- Decreased TTP = decreased lung volume.
- It is defined as the ratio of the change in the lung volume produced per unit change in the distending pressure (Transpulmonary pressure).
- The extent to which the lungs expand for each unit increase in the transpulmonary pressure.
- CL =Volume change (Δ V)/ Transpulmonary pressure change (Δ P)
- CL=(Δ V)/(Δ P)
- For both lungs in adult = 200 ml of air /cm H20 if the lung is free not surrounded by the chest.
- For lungs and thorax together = 110 ml/cm H20.
- Is reduced in pulmonary fibrosis, pulmonary edema, diseases of the chest wall ( kyphosis, scoliosis), paralysis of respiratory muscles.
- Emphysema (COPD chronic obstructive pulmonary disease) increases the compliance of the lungs because it destroys the alveolar septal tissue rich with elastic fibers that normally opposes lung expansion.



# Lecture#3 and Lecture #4

## Respiratory Ventilation

## Lung Function in Health and Disease



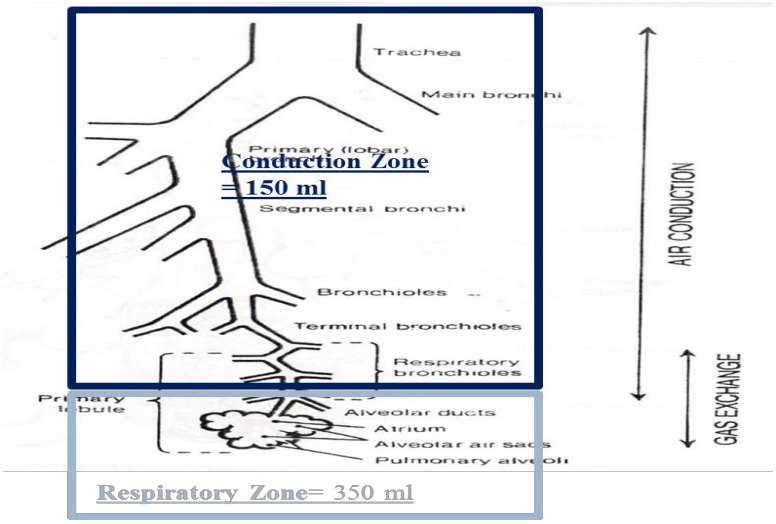

### The conducting zone:

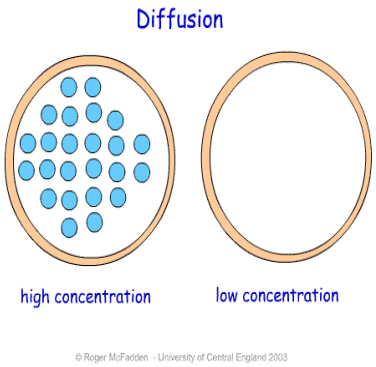
Structures form a continuous passageway for air to move in and out of the lungs.

### Respiratory zone:

Is found deep inside these thin-walled structures; allows inhaled oxygen (O2) to diffuse into the lung capillaries in exchange for carbon dioxide (CO2).

- It occupies the space distal to the terminal bronchioles start from the respiratory bronchioles down to the alveolar sacs.
- Where gas exchange takes place.
- Two thirds of the tidal volume is lost here.
- Volume ≅ 350ml




Gas diffuses in (respiratory zone) from higher concentration (higher pressure) to lower concentration (lower pressure).

# Spirometers

- Spirometry is a method to record volume of the movement of air into and out of the lungs.
  1. Widely used.
  2. Effort dependent basic lung function test.

- We use the spirometer to measure the lung volume and lung capacity, Except:
  1. Residual volume.
  2. Functional residual capacity (FRC)
  3. Total lung volume (capacity).
  - The floating drum contains either oxygen or normal air.
  - Counterbalancing weight contains a pen that will move to draw when the patient breath in or out.

## Very Important Note:

Spirometers are **NOT** used for measuring the: 1. Residual Volume, 2. Functional Residual Capacity, and 3. Total Lung Volume (**Capacity** not volume according to Guyton).

**The Residual Volume is the only volume that cannot be measured by the spirometer**

| | |
|---|---|
| **Spirometry is a simple most commonly used test to:** | 1- Assess the lung performance. |
| | 2- Measure the physiological parameters: Lung volumes, Capacities, Flow rate. |
| | 3- Differentiate between the obstructive and restrictive lung conditions. |
| | 4- Play a critical role in the diagnosis, differentiation and management of respiratory diseases. |

# Indications of Spirometry

Based on clinical features / abnormal lab tests

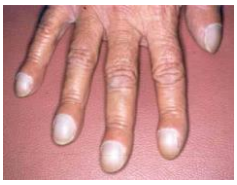
- Indications of spirometry
  - Clinical features (symptoms and signs) & lab tests
  - Occupation (pre-employment)
  - Certain diseases
  - Pre-operative
  - Asses therapeutic intervention

**1. Symptoms**

- Dyspnea ضيق التنفس
- Cough
- Sputum -لعاب) (بلغم production
- Or phlegm production
- Chest pain

**2. Signs**

- Cyanosis, ازرقاق للثة وأطراف الأصابع
- Clubbing *see in picture



- Chest deformity (like: kyphosis)
- Diminished chest expansion → Hyperinflation
- Diminished breath sounds → Prolongation of expiratory phase & crackles

**3. Arterial Blood Gas Analysis**

- Hypoxemia (low $O_2$ in blood)
- Hypercapnia (high $CO_2$ in blood)
- ^= Low Ph (acid)

**4. Abnormal Chest X Ray.**

- Diminished chest expansion (When you ask the patient to take deep breathe, it be limited movement)
- Hyperinflation
- Diminished breath
- Prolongation of expiratory phase & crackles (Who have fluid in the chest)

# Cont….

Occupational Settings:

Pre employment

Periodic lung function examination for workers exposed to toxic substances including dust and fumes in industrial sectors such as:

Cement / Asbestoses

Welding / Wood / Steel

Flour / Coal mine / Oil

- To detect respiratory disease in patients presenting with symptoms of breathlessness, and to distinguish respiratory from cardiac disease.
- To diagnose or manage asthma
- To diagnose and differentiate between obstructive and restrictive lung diseases.

➢ **Describe the course of diseases affecting PFT:**

Neuromuscular diseases: Gillian Barre Syndrome, Myasthenia gravis

Pulmonary diseases: Obstructive airway diseases, Interstitial lung diseases

Adverse reactions: Drugs with known pulmonary toxicity [Pulmonary fibrosis]

➢ **Monitoring indications (to assess the therapeutic interventions):**

(To measure the response to treatment of conditions which spirometry is used in the detection of e.g. COPD, etc..)

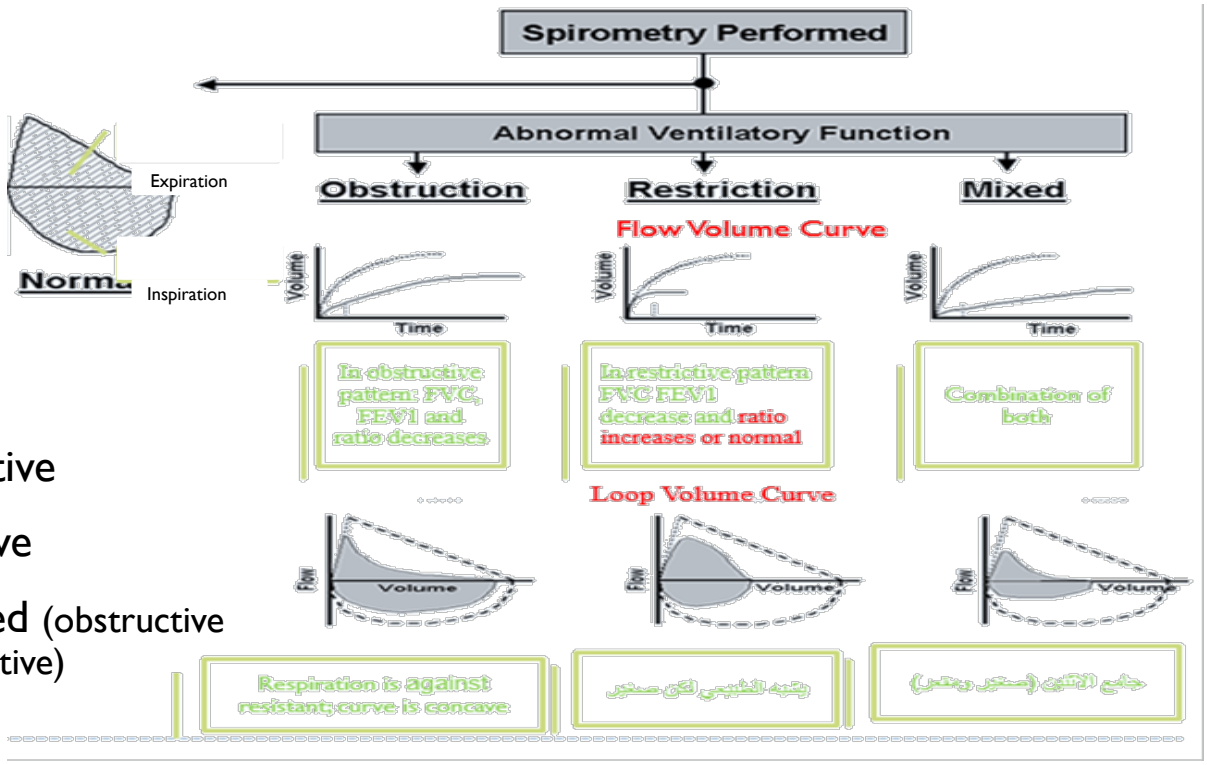
- Bronchodilator therapy
- Steroid treatment for asthma

Used on Chronic obstructive lung disease and Interstitial (restrictive) lung disease

➢ **Pre operative indications** (to conduct pre-operative risk assessment before anesthesia**):**
To determine the suitability of patients for anesthesia **during and after anesthesia** To assess the risk for surgical procedures known to affect lung function.

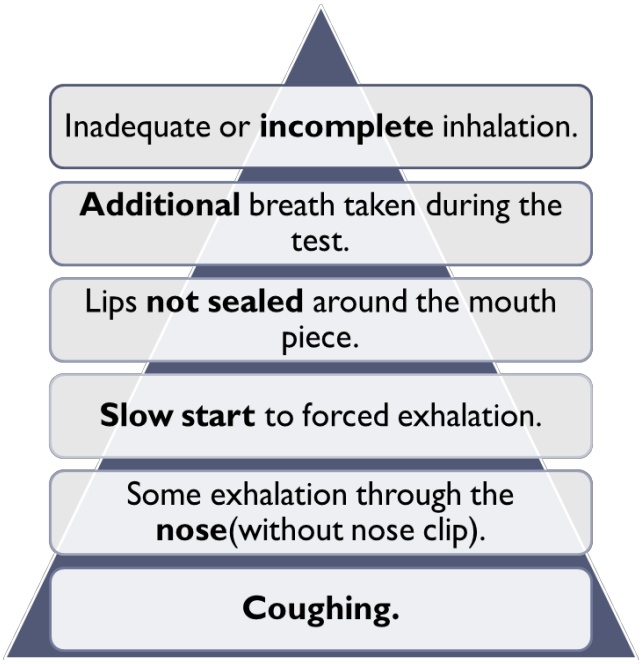
# Assessment of Spirometry

1. Normal
2. Obstructive
3. Restrictive
4. Combined (obstructive and restrictive)




# Maintaining Accuracy

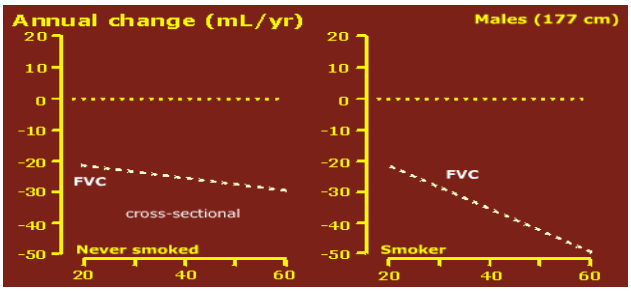
- The most common reasons for inaccurate results:



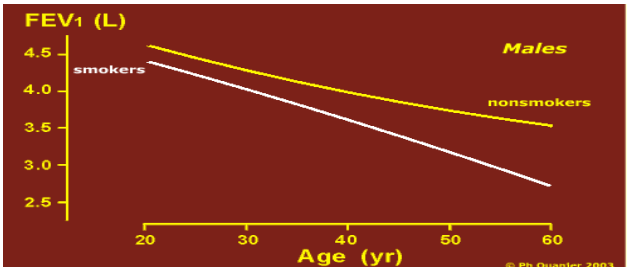
# Smoking and Spirometry

Effects of smoking on lung function:

- Non-Smoker: In normal healthy non-smoker subject after the age of 30-35 the expected decline in lung function parameter [FEV1] is 25–30 ml/ year




- Smokers: The average rate of decline of lung function in smokers as measured by Forced Expiratory Volume in 1 sec [FEV1] is 60-70 ml / year




# Diagnosis of COPD

All of these indicate the use of Spirometry to diagnose COPD:

**Symptoms**

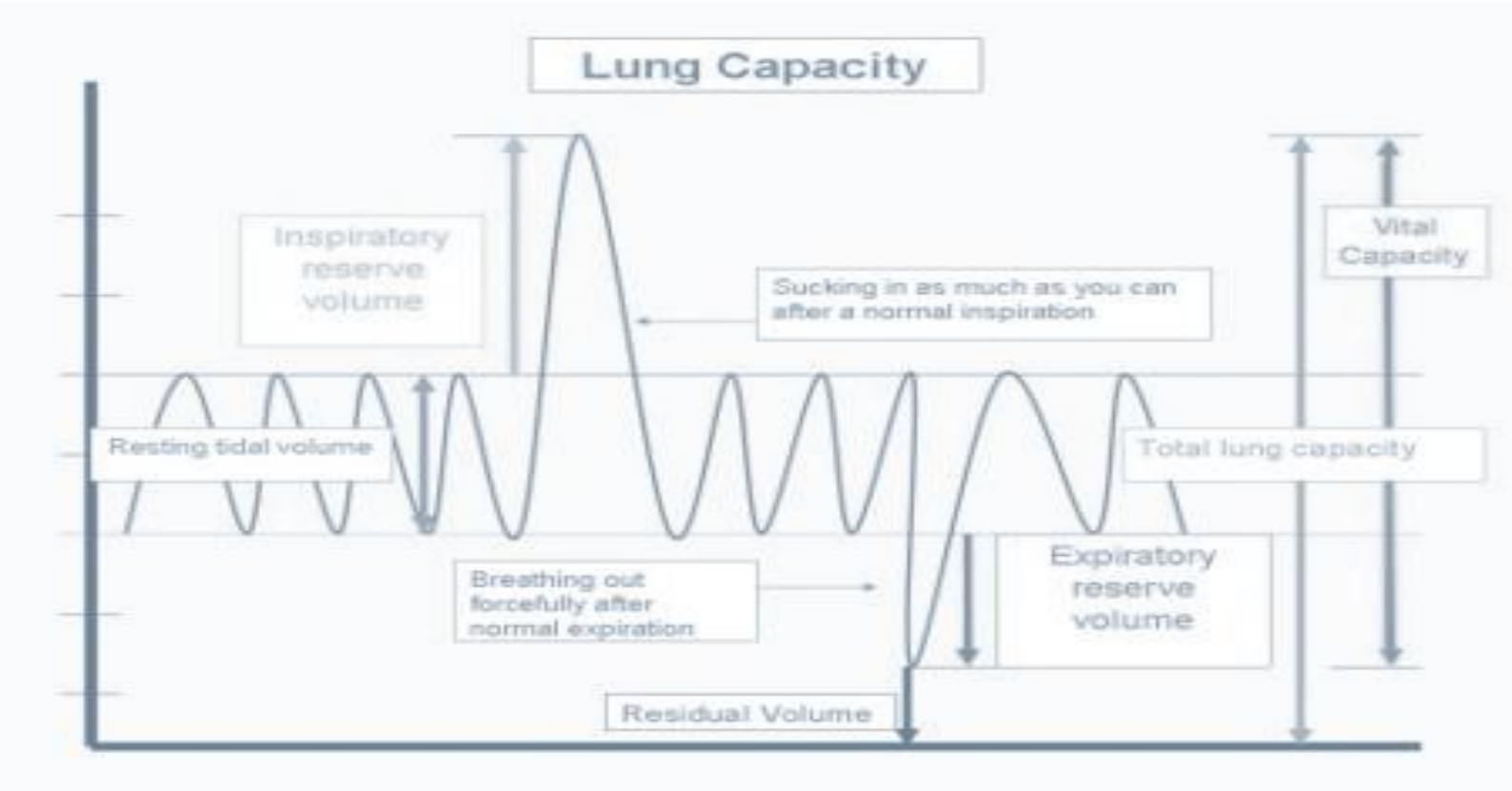
- Septum
- Cough
- Dyspnea

**Risk Factors**

- Occupation
- Indoor/outdoor pollution
- Tobacco

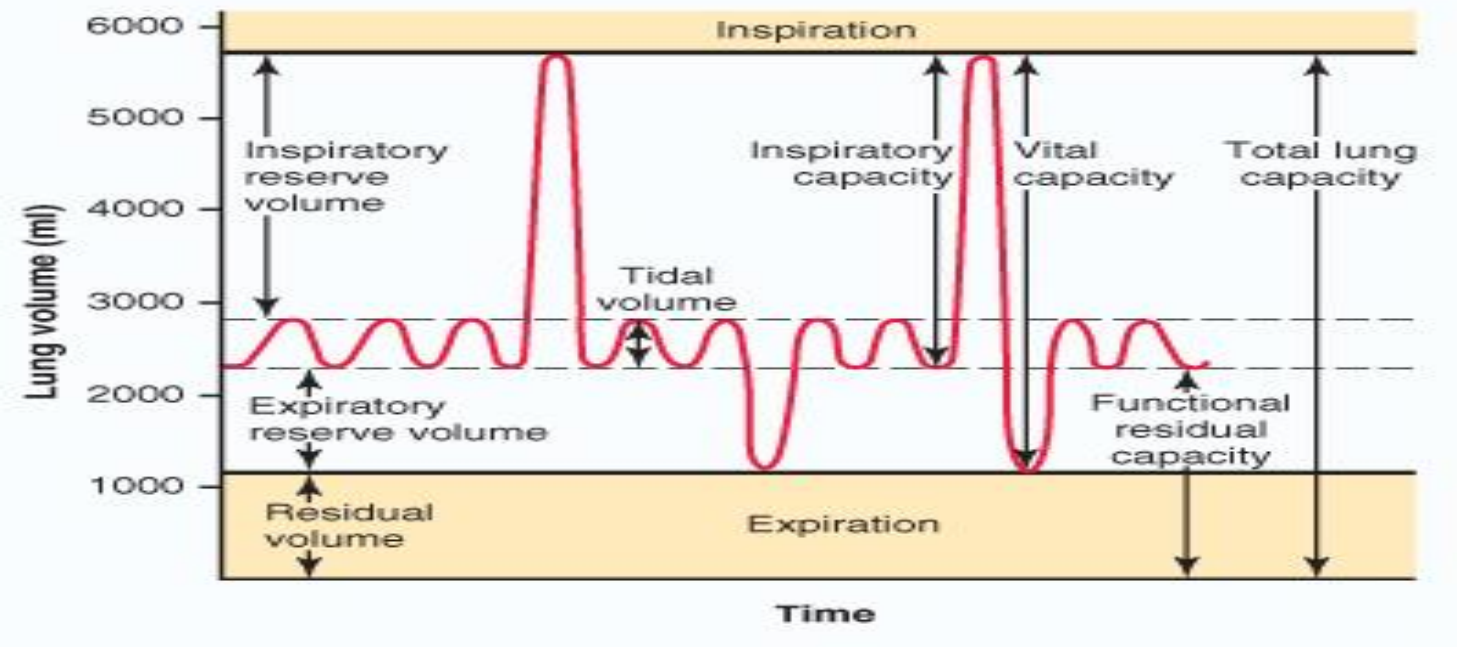
# Lung volumes

| Lung volumes | | | |
|---|---|---|---|
| **Type** | **Explanation** | **Value in male** | **Value in female** |
| Tidal Volume (TV) | The amount of air inspired or expired with each normal breath. | 500 ml | 500 ml |
| Inspiratory Reserve Volume (IRV) | Is the extra volume of air that can be inspired over and above the normal tidal volume when the person inspires with full force. | 3000 ml | 2000ml |
| Expiratory Reserve Volume (ERV) | Is the maximum extra volume of air that can be expired by forceful expiration after the end of a normal tidal expiration. | 1100 ml | 700ml |
| Residual Volume (RV) | The volume of air remaining in the lung after a maximal expiration. | 1200ml | 1100ml |



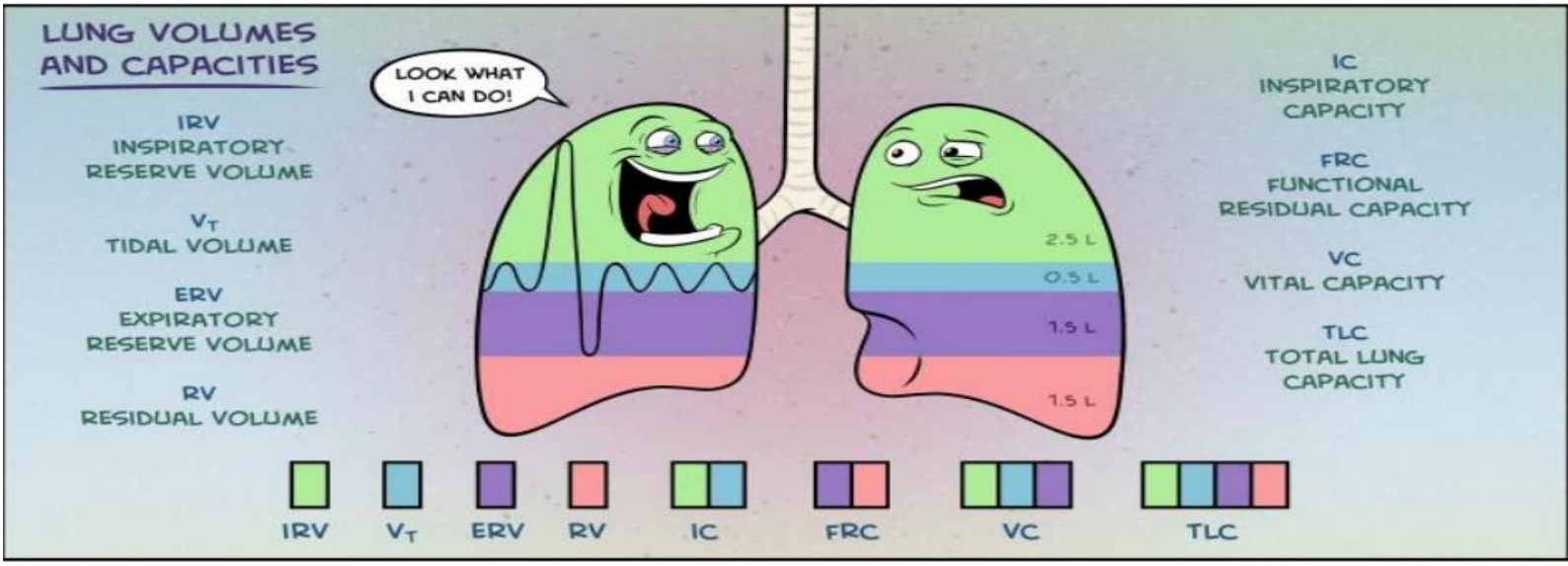
# Lung capacity

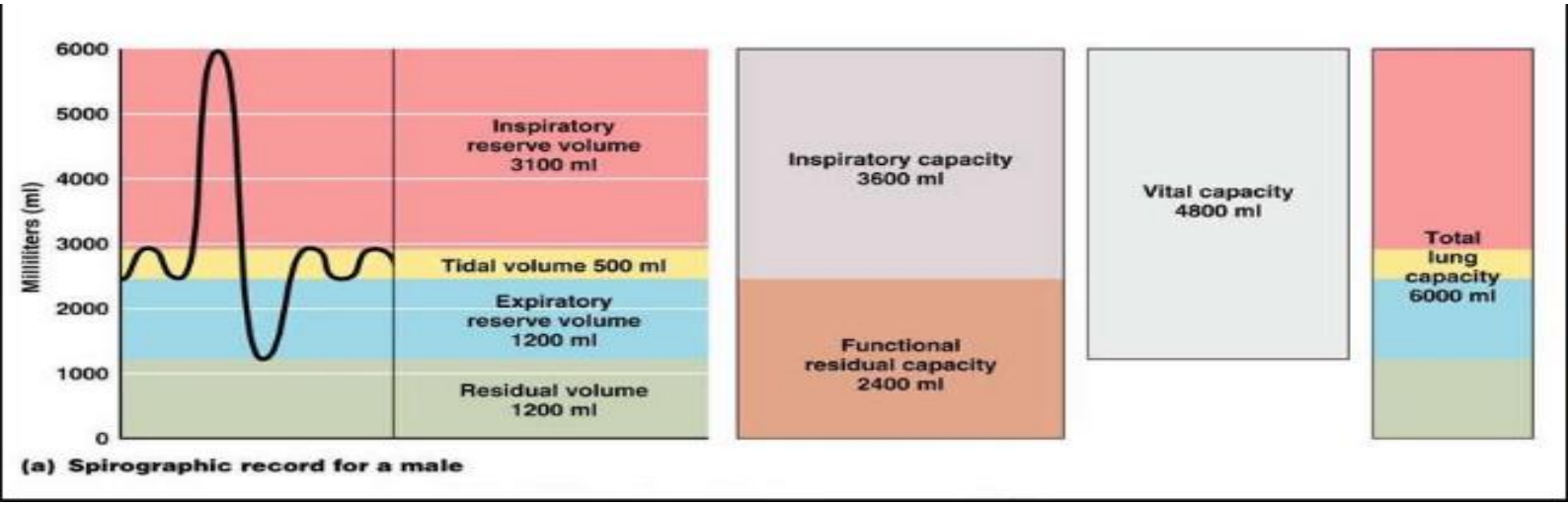
| Lung capacity | | | |
|---|---|---|---|
| **Type** | **Explanation** | **Value in male** | **Value in female** |
| Inspiratory Capacity (IC) | Is the amount of air a person can breath in, beginning at the normal expiratory level and distending the lungs to the maximum amount . | IC= TV+IRV 500+3000 =3500 ml | IC= TV+IRV 500+2000 =2500ml |
| Functional Residual Capacity (FRC) | The volume of air remaining in the lung at the end of a tidal expiration. Acts as a buffer against extreme changes in alveolar gas levels with each breath. | FRC= RV+ERV 1100+1200= 2300 ml | FRC= RV+ERV 700+1100= 1800ml |
| Vital Capacity (VC) | The maximum volume of air that can be forcefully expelled from the lungs following a maximal inspiration. | VC= IRV+TV+ERV =500+3000+ 1100 = 4600 ml | VC= IRV+TV+ER V=500+2000 +700 = 3200ml |
| Total Lung Capacity (TLC) | The volume of air contained in the lungs at the end of a maximal inspiration. Or with the greatest possible effort. | TLC=ALL OF THE VOLUMES = 5800 ml | TLC=ALL OF THE VOLUMES = 4300ml |




All lung volumes and capacities are 20-25% less in women than men, they are greater in large athletic people than in small athletic people.

# Summary Lung Volumes And Capacities




(a) Spirographic record for a male

| | Measurement | Adult male average value | Adult female average value | Description |
|---|---|---|---|---|
| Respiratory volumes | Tidal volume (TV) | 500 ml | 500 ml | Amount of air inhaled or exhaled with each breath under resting conditions |
| | Inspiratory reserve volume (IRV) | 3100 ml | 1900 ml | Amount of air that can be forcefully inhaled after a normal tidal volume inhalation |
| | Expiratory reserve volume (ERV) | 1200 ml | 700 ml | Amount of air that can be forcefully exhaled after a normal tidal volume exhalation |
| | Residual volume (RV) | 1200 ml | 1100 ml | Amount of air remaining in the lungs after a forced exhalation |
| Respiratory capacities | Total lung capacity (TLC) | 6000 ml | 4200 ml | Maximum amount of air contained in lungs after a maximum inspiratory effort: TLC = TV + IRV + ERV + RV |
| | Vital capacity (VC) | 4800 ml | 3100 ml | Maximum amount of air that can be expired after a maximum inspiratory effort: VC = TV + IRV + ERV (should be 80% TLC) |
| | Inspiratory capacity (IC) | 3600 ml | 2400 ml | Maximum amount of air that can be inspired after a normal expiration: IC = TV + IRV |
| | Functional residual capacity (FRC) | 2400 ml | 1800 ml | Volume of air remaining in the lungs after a normal tidal volume expiration: FRC = ERV + RV |

(b) Summary of respiratory volumes and capacities for males and females

# Physiological Conditions and Pulmonary Volumes and Capacities (Important)

Age: Pulmonary capacities/Vol keep on increasing until the age of 35 then begins to decrease after the age 35.

Gender: All lung volumes and capacities are about 20 to 25% less in women than in men.

Diurnal variation, seasonal, climate.

Height: Height increase, increases pulmonary Vol/Cap.

Health: If the patient is normal or has lung diseases.

Geographical location.

Ethnic group

Weight: Weight increase (obesity), decreases pulmonary Vol/Cap.

Exercise: Increases pulmonary Vol/Cap . All lung volumes and capacities are greater in athletic persons than in small and asthenic persons.

Posture: Pulmonary Vol/Cap while standing is higher than while sitting.

Pregnancy: Decreases pulmonary Vol/Cap.

Customary activity.

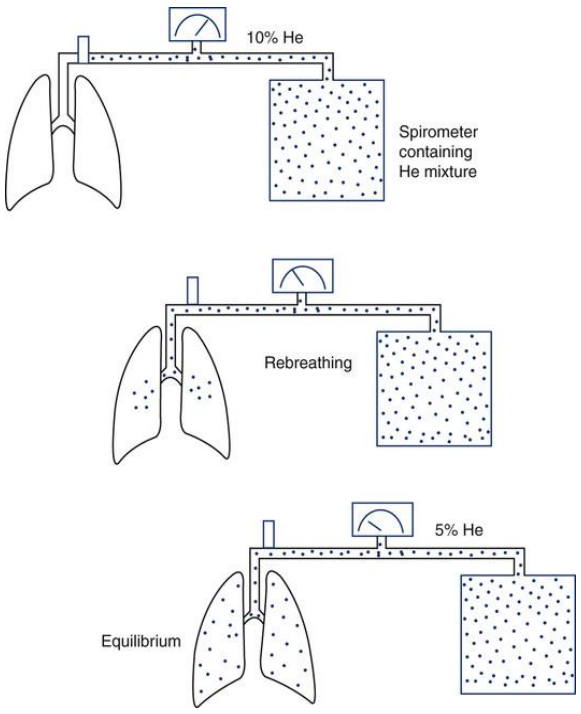
# Dead space

| Anatomical dead space | Functional dead space |
|---|---|
| The portion where there is no possibility of gas exchange (conductive zone)<br>The air present in conductive zone = 150 ml. | The portion where there is possibility of gas exchange but its not happening<br>Due to absent or poor blood flow |

**Physiological dead space**

Anatomical and functional dead spaces together defines the physiological dead space.

# Closed Circuit Helium Dilution Method




- We use Closed circuit Helium Dilution to determine FRC, RV, and TLC.
- **C1xV1 = C2xV2**

  C1: concentration of Hi in spirometry

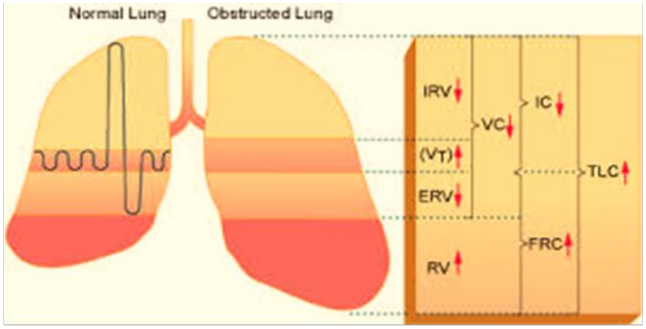
  V1: volume of air in the spirometry.

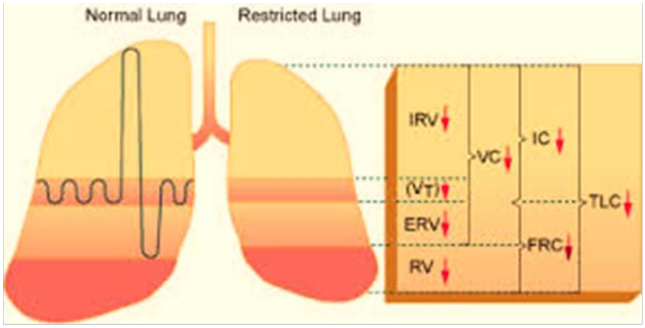
  C2: Final concentration of helium

  V2 :Volume of spirometry+ FRC

$$FRC = \left( \frac{Ci\ He\ (C1)}{Cf\ He\ (C2)} - 1 \right) \times Vi\ Spi\ (V1)$$

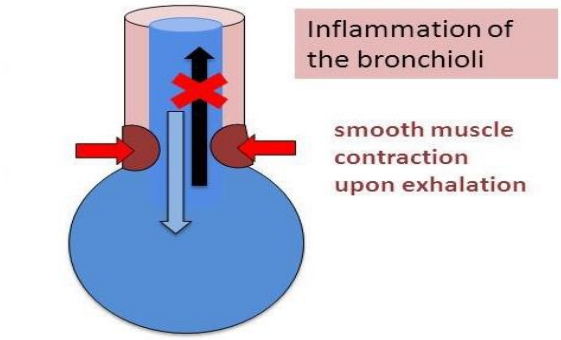
# Obstructive and Restrictive Diseases

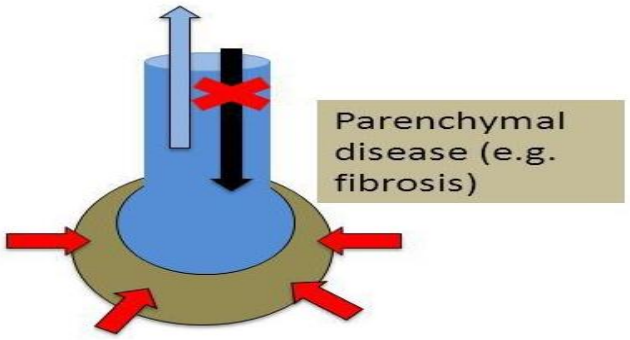



Obstructive disease causes **airway obstruction** leading to decrease in airflow into and out of the lungs and trapping of air inside the lung (that is why **RV, FRC** TLC are increased)

Restrictive disease restrict expansion of lungs causing decrease in lung volume (notice the decrease in amount of air according to all parameters)




Obstructive disease prohibits air from flowing OUT of alveoli, trapping air in the lungs and overinflating them

Restrictive disease prohibits air from flowing INTO the alveoli by not allowing expansion

## Minute Ventilation Rate and Volume

- **Respiratory rate:** Number of breaths taken per minute.
- **Minute ventilation:** Total amount of air moved into and out of respiratory system per minute.
- **Minute respiratory volume: MRV:** The total amount (volume) of new air that moves into the respiratory passages in each minute is called the minute respiratory volume.

Minute respiratory volume (MRV)= tidal volume x respiratory rate.

- **Rate of Alveolar Ventilation:**

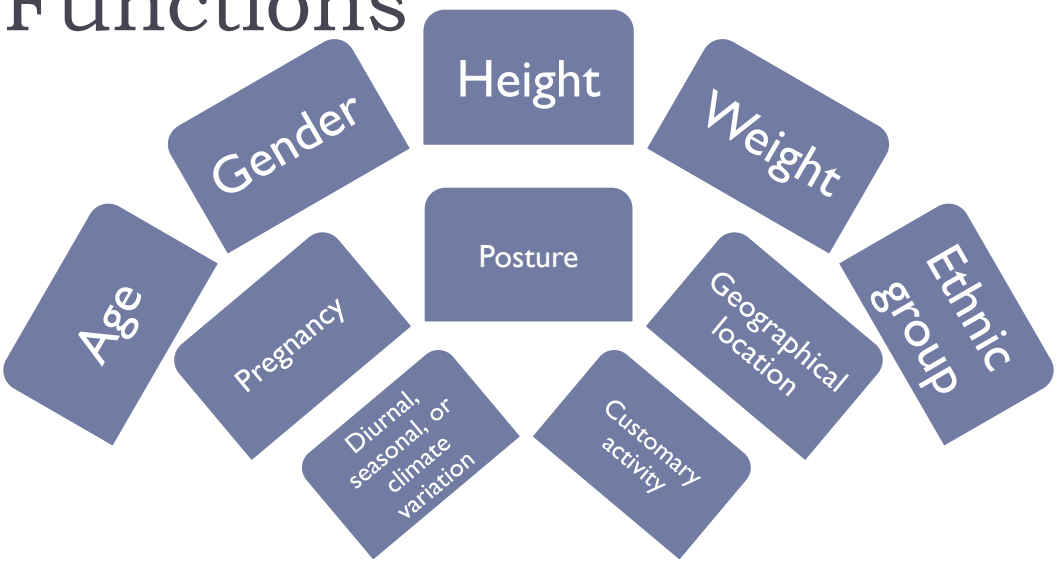
**Alveolar ventilation = respiratory rate X (Tidal volume – air in dead space)**

Alveolar ventilation per minute is the total volume of new air entering the alveoli and other adjacent gas exchange areas (respiratory zone) each minute. **(Because 1/3 is lost in conductive zone.)**

# Types of Lung Function Tests

| | |
|---|---|
| Spirometry | • It is the measurement of the speed and the amount of air that can be exhaled and inhaled.<br>•There are two types: 1. Static (for lung volumes) and<br>•2. dynamic (for FEV1 and flow rate) |
| Body Plethysmography test: | • The patient is required to sit in an airtight chamber that resembles a small telephone booth. Inside the chamber is an affixed spirometer, which is used to determine the flow properties of the patient. |
| Cardiopulmonary "Exercise" Stress Testing | • Used for evaluation of dyspnea that is out of proportion to findings on static pulmonary function tests (to differentiate between cardiac and chest problems) |
| Diffusing Capacity of Lung for Carbon Monoxide | • To evaluate the presence of possible parenchymal lung disease |
| Pulse Oximetry | • The principle is measurement of O2 saturation by spectrophotometry |

# Physiological Conditions Affecting Lung Functions



# Lecture#5

## Effects of Exercise on Respiration

### The respiratory system and exercise

- During exercise muscles need more oxygen and more CO2 must be removed from working muscles
- As a result we increase

1- Breathing rate

2- Depth of breathing increases up to our vital capacity

3- Blood flow through the lungs

4- Oxygen intake and consumption

Oxygen used during exercise can increase up to 20 times a person's normal oxygen uptake= 250ml

## Energy system

**Anaerobic systems**

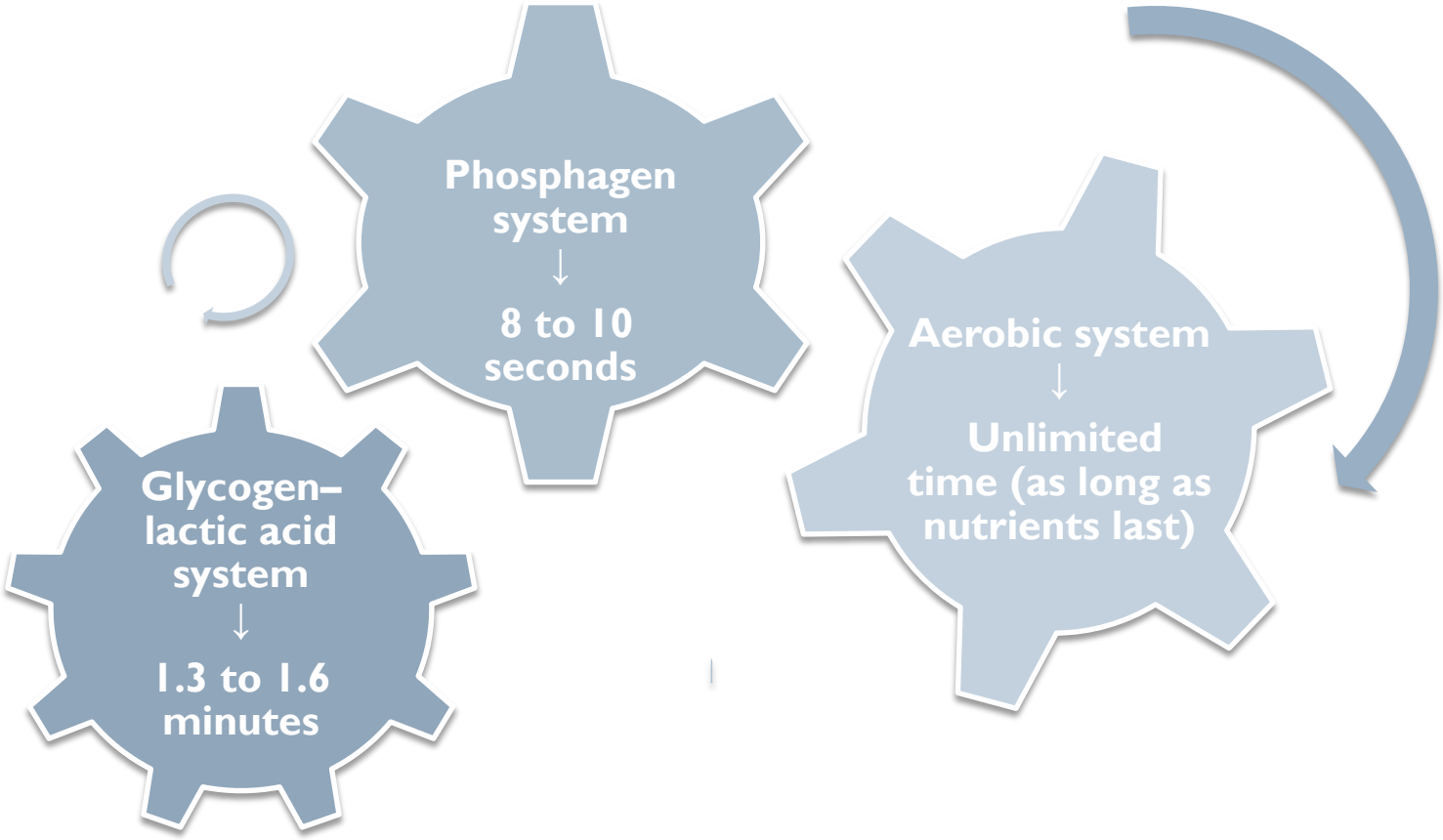
- **I. Phosphocreatine** → Creatine + PO3
  **II. Glycogen** →Lactic acid

**Aerobic systems**

- Uses fatty acids, glucose or amino acids
- X + O2 →CO2 + H2O+Urea
- X = glucose, fatty acids, or amino acids

# The phosphagen energy system

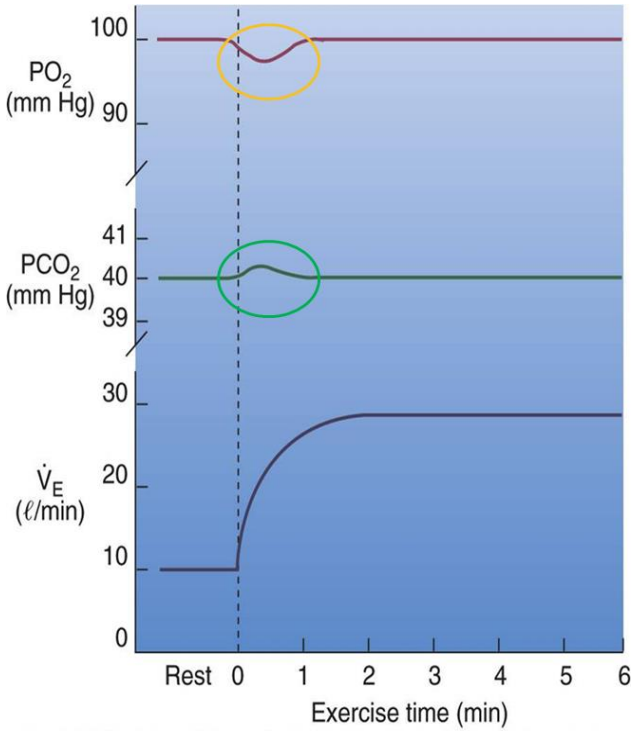
Relation between exercise duration & energy source




- The combined amounts of cell ATP and cell phosphocreatine
- These (ATP & phosphocreatine) together can provide maximal muscle power for 8 to 10 seconds, almost enough for the 100-meter run.
- Thus, the energy from the phosphagen system is used for maximal short bursts of muscle power.

# Effect of Exercise on the respiratory system

- During exercise the respiration is stimulated by neurogenic mechanisms and in this situation the blood gases do not have to become abnormal .
- Arterial tensions of PCO2 and PO2 are relatively unchanged during submaximal exercise .
- but arterial PO2 decreases & PCO2 increases slight in transition from rest to steady state exercise .




## Regulation of respiration during exercise

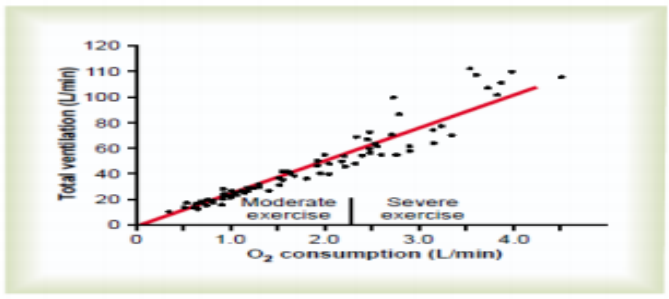



**Figure 41–8**

Effect of exercise on oxygen consumption and ventilatory rate. (From Gray JS: Pulmonary Ventilation and Its Physiological Regulation. Springfield, Ill: Charles C Thomas, 1950.)

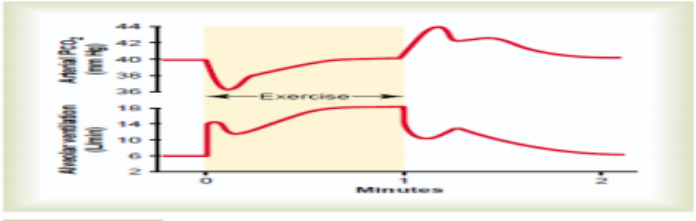



**Figure 41–9**

Changes in alveolar ventilation *(bottom curve)* and arterial $PCO_2$ *(top curve)* during a 1-minute period of exercise and also after termination of exercise. (Extrapolated to the human being from data in dogs in Bainton CR: Effect of speed vs grade and shivering on ventilation in dogs during active exercise. J Appl Physiol 33:778, 1972.)

The blood gases do not always have to become abnormal for respiration to be stimulated in exercise.

Instead, respiration is stimulated mainly by neurogenic mechanisms during exercise.

In exhausting "strenuous" exercise O2 consumption and CO2 formation increase 20 times than normal but alveolar ventilation increases almost exactly in step with the increased levels of metabolism.

Therefore the arterial PO2, PCO2, PH all remain almost exactly normal.

# Intense ventilation during exercise?

- The brain, on transmitting **motor impulses** to the exercising muscles, transmits at the same time collateral impulses into the brain stem to excite the respiratory center.
- A large share of the total increase in ventilation begins immediately on initiation of the exercise, before any blood chemicals have had time to change.
- It is likely **that most** of the increase in respiration results from neurogenic signals transmitted directly into the brain stem respiratory center at the same time that signals go to the body muscles to cause muscle contraction.

## Causes:

1. Neural signals from the motor areas of the brain to the respiratory center.
2. The joint proprioceptors .
3. Body temperature (hypothalamus).
4. Possibility that the neurogenic factor for control of ventilation during exercise is a learned response.

**During maximal effort:**

- Pulmonary ventilation at maximal exercise 100-110 L/min
- Maximal breathing capacity 150-170L/min
- Maximal breathing capacity is about 50 % greater than the actual pulmonary ventilation during maximal exercise to giving athletes extra ventilation E.g.:
  1. Exercise at high altitudes
  2. Exercise under very hot conditions.
  3. Abnormalities in the respiratory system

# Interrelation Between Chemical Factors and Nervous Factors in the Control of Respiration During Exercise.

- Direct nervous signal stimulate the respiratory center almost the proper amount to supply the extra oxygen required for exercise and to blow off extra carbon dioxide.
- Occasionally, the nervous respiratory control signals are either **too strong or too weak**.
- Then chemical factors play a significant role in bringing about the final adjustment of respiration required to keep the O2, Co2, and H+ ion concentrations of the body fluids as nearly normal as possible.

# The Neurogenic Factor for Control of Ventilation During Exercise is a Learned response.

- The ventilatory response during exercise, is at least partly a learned response.
- That is, with repeated periods of exercise, the brain becomes progressively more able to provide the proper signals required to keep the blood PCO2 at its normal level.
- The cerebral cortex is involved in this learning, because experiments that block only the cortex also block the learned response.

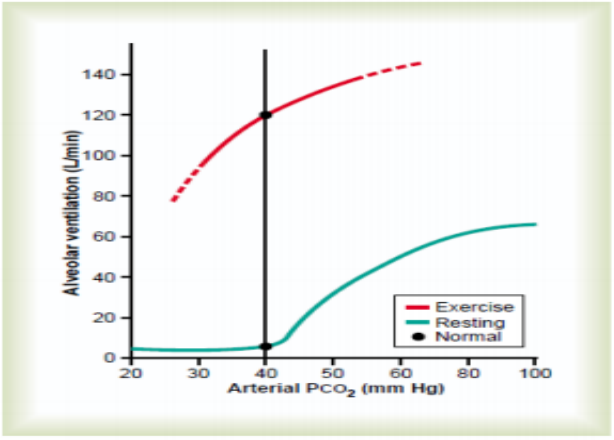
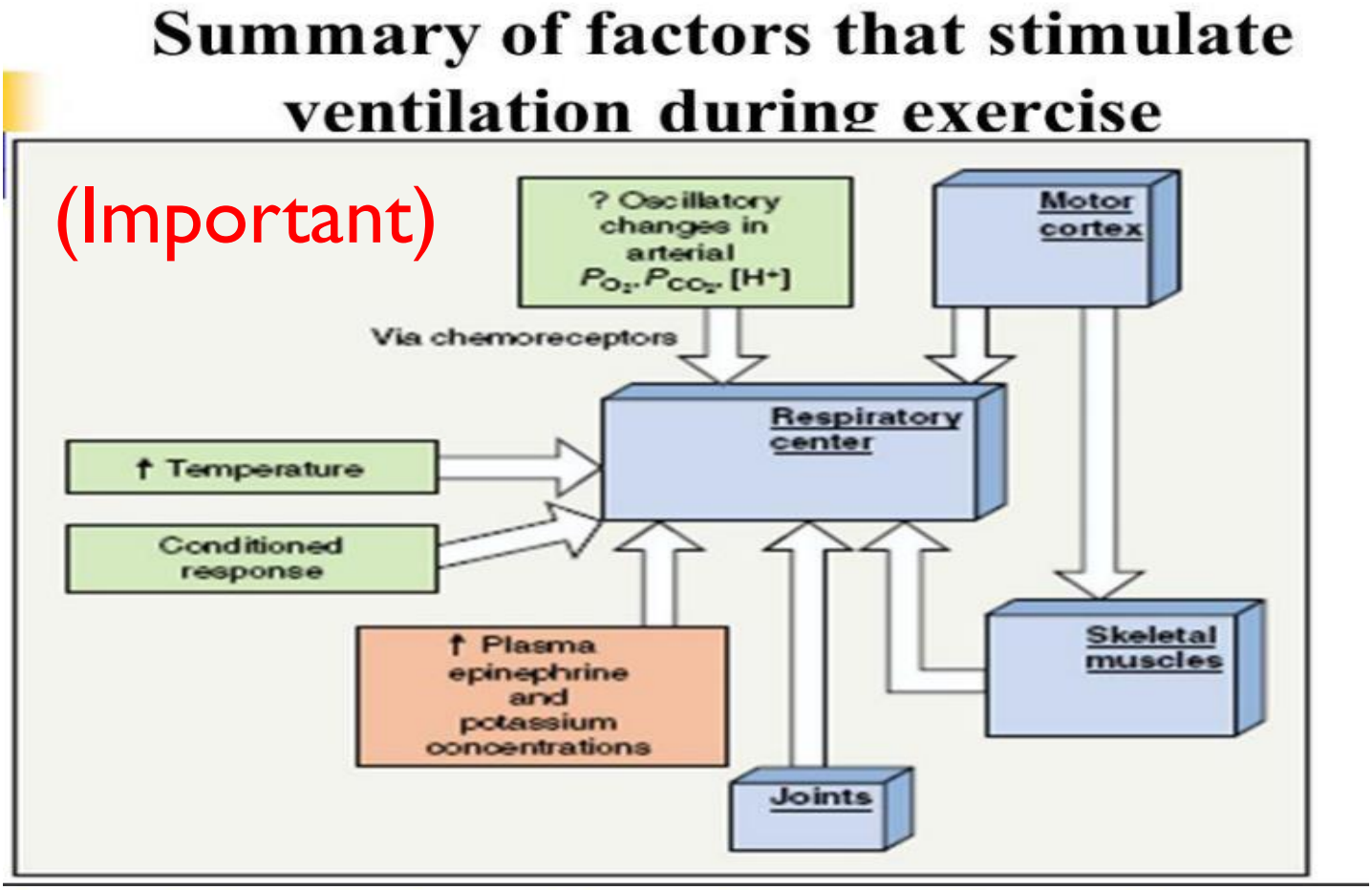
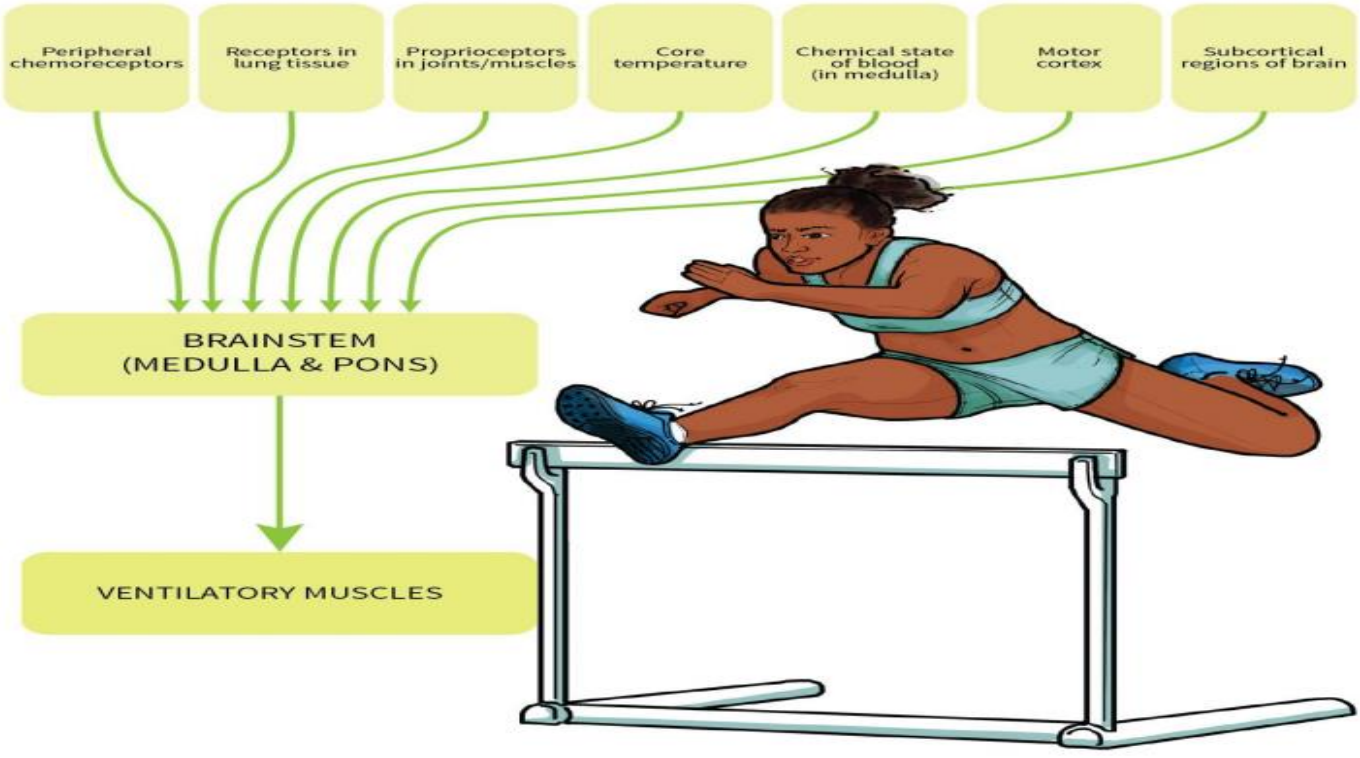



**Figure 41–10**

Approximate effect of maximum exercise in an athlete to shift the alveolar $P_{CO_2}$–ventilation response curve to a level much higher than normal. The shift, believed to be caused by neurogenic factors, is almost exactly the right amount to maintain arterial $P_{CO_2}$ at the normal level of 40 mm Hg both in the resting state and during heavy exercise.

# Summary


Summary of factors that stimulate ventilation during exercise
(Important)
? Oscillatory changes in arterial $P_{O_2}$, $P_{CO_2}$, $[H^+]$
Motor cortex
Via chemoreceptors
Respiratory center
↑ Temperature
Conditioned response
↑ Plasma epinephrine and potassium concentrations
Skeletal muscles
Joints

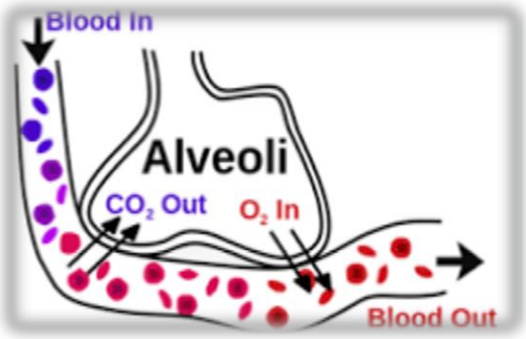

Peripheral chemoreceptors
Receptors in lung tissue
Proprioceptors in joints/muscles
Core temperature
Chemical state of blood (in medulla)
Motor cortex
Subcortical regions of brain
BRAINSTEM (MEDULLA & PONS)
VENTILATORY MUSCLES

# Diffusion capacity of the respiratory membrane

- Is the volume of gas that diffuses through the membrane each minute for a pressure difference of 1mmHg.
- Diffusing capacity for oxygen at rest =21ml/min/mmHg
- Even if the oxygen pressure difference across the respiratory membrane is 11mmHg
- 11x21= 230ml oxygen diffusing through the membrane each minute.
- During rest tissues consume 250 ml O2 per min.
- Changes occur on oxygen diffusing capacity during exercise it will become = **65ml/min/mmHg**
- This is due to **increased number of open pulmonary capillaries** which were dormant (inactive), thereby increasing the surface area for gas exchange.
- In addition to **increased alveolar ventilation**.

# Diffusing capacity for carbon dioxide

- During rest tissues consume 250 ml O2 per min, CO2 diffuses 20 times greater than oxygen due to greater diffusion coefficient which is 20 times that for oxygen.
- Diffusion capacity for carbon dioxide **=400ml/min/mmHg.**
- During exercise =**1200 to 1300ml/min/mmHg.**



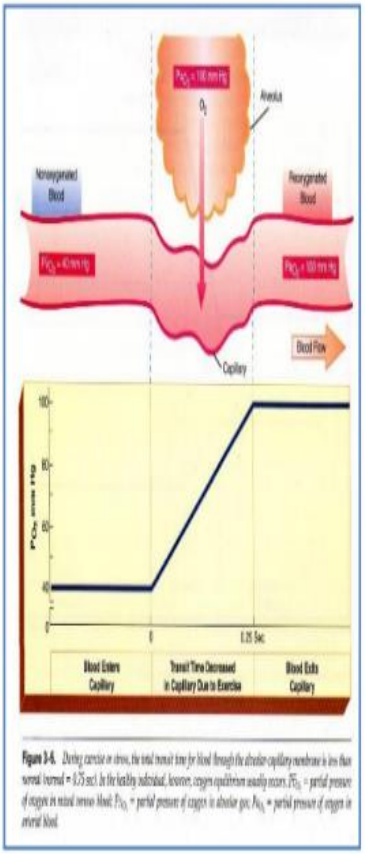
# Cont..

- During exercise the oxygen requirement increases 20 times, and cardiac output increases and so the time blood remains in the pulmonary capillaries becomes less than half normal despite the fact that additional capillaries open up.
- But the blood is almost completely saturated with oxygen when it leaves the pulmonary capillaries.

## Reasons for this are as follow:

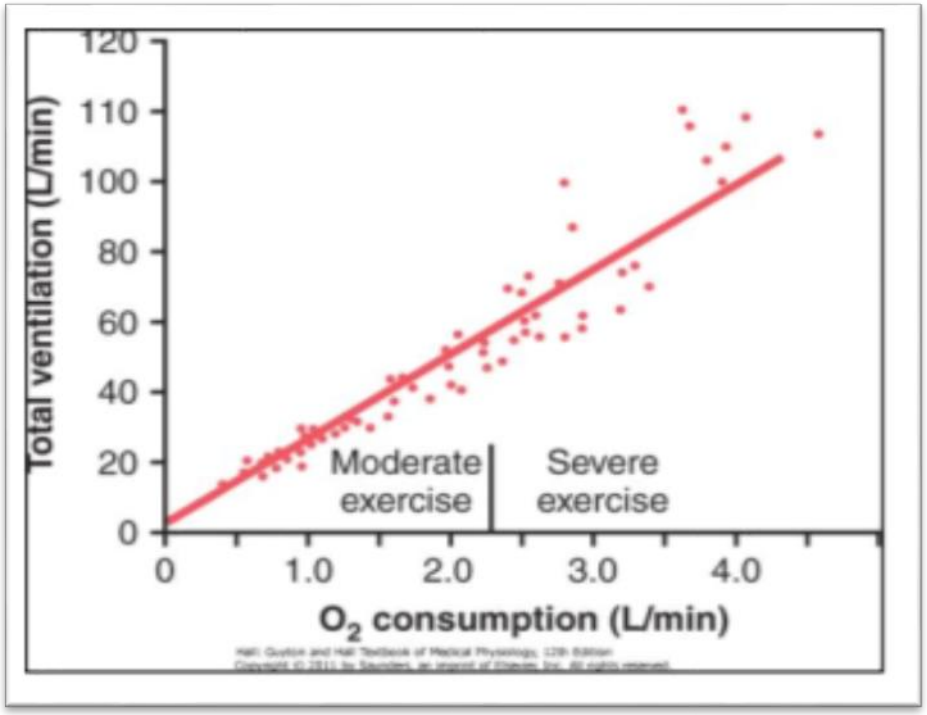
1. The diffusing capacity for oxygen increases almost three folds during exercise, this results mainly from increasing numbers of capillaries participating in the diffusion and a more V/Q ratio all over the lung.
(It is the ratio of alveolar ventilation to pulmonary blood flow (cardiac output) per minute).

2. At rest the blood normally stays in the lung capillaries about three times as long as necessary to cause full oxygenation. Therefore, **even with shortened time of exposure in exercise, the blood is still fully oxygenated or nearly so.**



# Relation between oxygen consumption and total pulmonary ventilation at different levels of exercise.

There is a linear relationship between both oxygen consumption (Vo2 Max) and total pulmonary ventilation increase about 20-folds between the resting state and maximal intensity of exercise in the well-trained athlete.



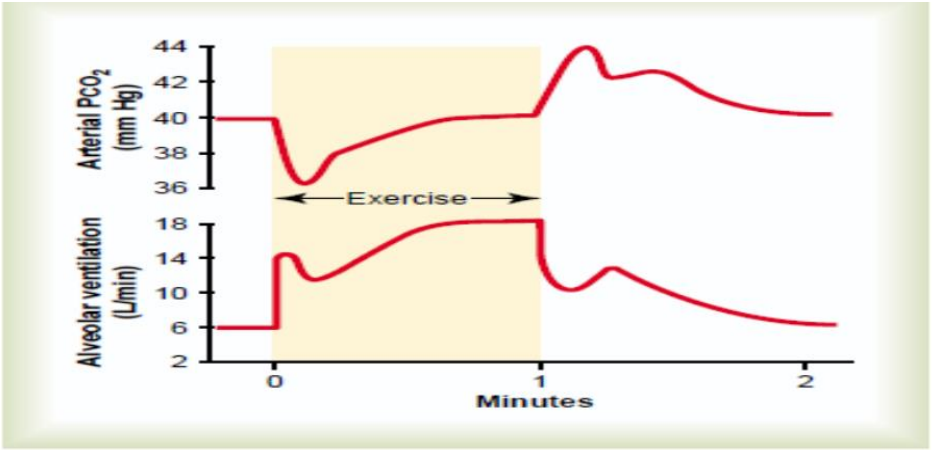
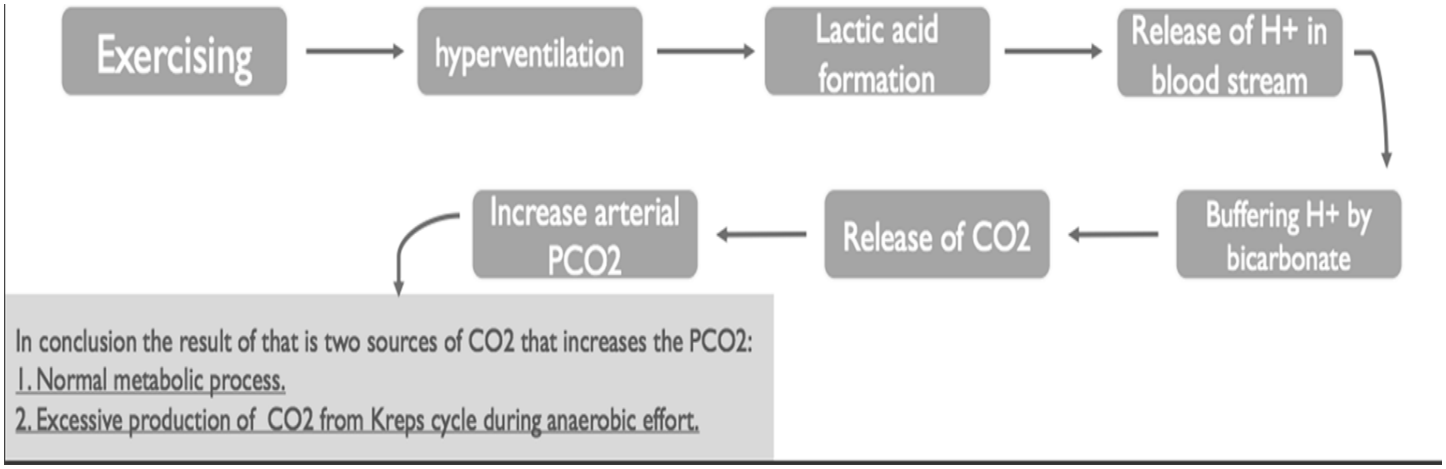




**Figure 41–9**

Changes in alveolar ventilation *(bottom curve)* and arterial PC( *(top curve)* during a 1-minute period of exercise and also after termination of exercise. (Extrapolated to the human being from data in dogs in Bainton CR: Effect of speed vs grade and shivering c ventilation in dogs during active exercise. J Appl Physiol 33:77( 1972.)

# Effects of exercise on arterial PO2, PCO2 and H+ ions.

Many studies have since reported that the lactate threshold (LT) is strongly correlated with ventilatory anaerobic threshold (VAT) The term VAT actually refers to the onset of exercise induced hyperventilation during effort. This increase in  is a homeostatic response to deal with the consequences of the excess lactate production which can dissociate to release H+ ions from lactic acid into the blood stream. The H+ ions are buffered by bicarbonate and release CO2 .This buffering of lactic acid results in extra CO2 production over that produced by aerobic metabolism and increases the arterial CO2 partial pressure (PaCO2). The increase in PaCO2 stimulates excess ventilation that follows on from the lactate threshold.




# Pulmonary ventilation and MBC

During maximal effort:

- Pulmonary ventilation (minute ventilation) at maximal exercise 100-110L/min
- Maximal breathing capacity 150-170L/min
  - Maximal breathing capacity is about 50 % greater (70% in some references) than the actual pulmonary ventilation during maximal effort (i.e. exercise).

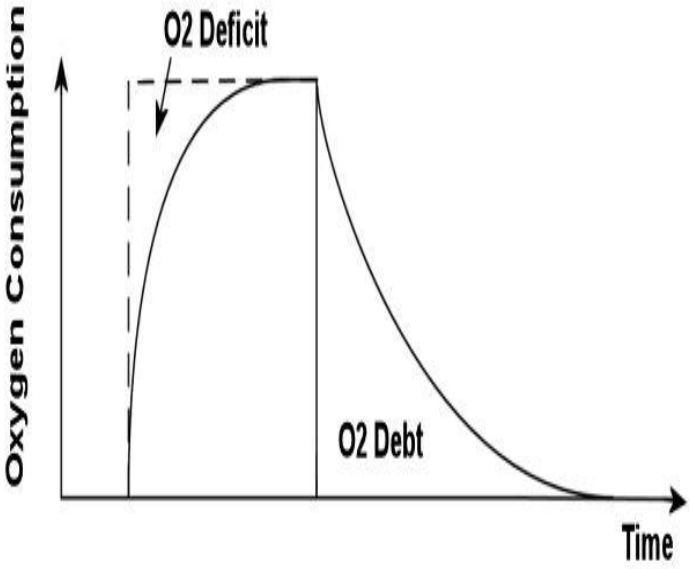
# Oxygen Consumption and Pulmonary Ventilation in Exercise.

- Normal oxygen consumption for a young man at rest is about 250 ml/min.
- However, under maximal conditions, this can be increased to approximately the following average levels:
  - Untrained average male= 3600 ml/min
  - Athletically trained average male =4000 ml/min
  - Male marathon running = 5100 ml/min

# Oxygen debt

- Oxygen debt is the extra consumption of oxygen after completion of strenuous exercise (about 11.5 liters).

- You will develop oxygen debt after about 5 minutes or more of constant exercise. This is the point when the exercise becomes anaerobic (without the use of oxygen) and which has to be paid back. If the exercise is just aerobic (with oxygen) there will be no oxygen debt.




Example: when you have a short intense burst of exercise such as sprinting you generate energy for this anaerobically or without oxygen. When you stop exercising you are still breathing heavily. This is your body taking in extra oxygen to 'repay' the oxygen debt.

# Summary

- Oxygen consumption (Vo2) at rest → 250 ml/min
- Untrained average male → 3600 ml/min
- Athletically trained average male → 4000 ml/min
- Male marathon runner → 5100 ml/min

- Diffusing capacity for oxygen at rest → 21ml/min/mmHg
- Diffusing capacity of oxygen during exercise →65ml/min/mmHg
- Diffusion capacity for carbon dioxide at rest → 400ml/min/mmHg.
- Diffusion capacity for carbon dioxide during exercise →1200 to 1300ml/min/mmHg.

- O2 consumption and CO2 formation and pulmonary ventilation (minute ventilation) increase 20 folds during strenuous (exhausting) exercise
- Pulmonary ventilation (minute ventilation) at rest → 12 L/min
- Pulmonary ventilation (minute ventilation) at maximal exercise →100-110L/min
- Maximal breathing capacity → 150-170L/min

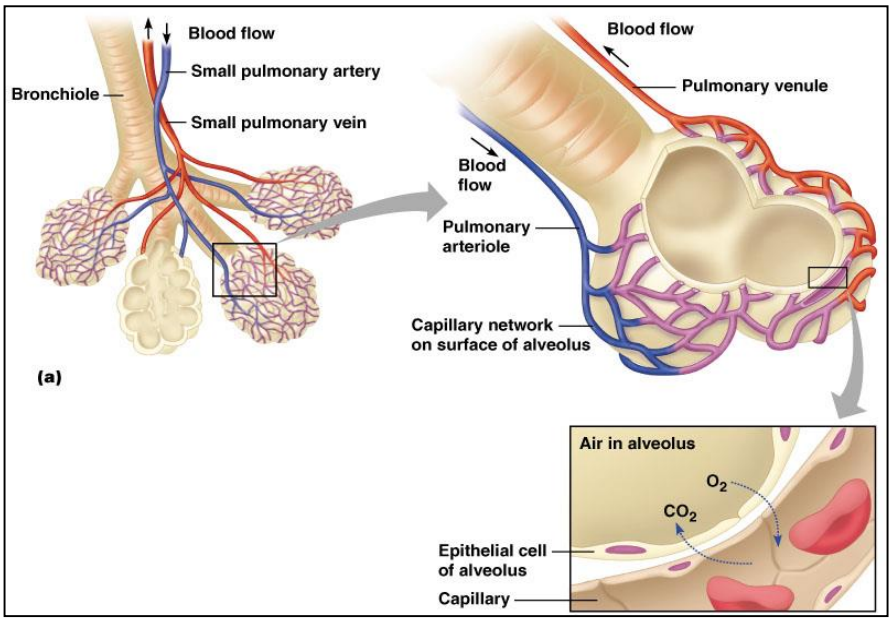
- Phosphagen system → 8 to 10 seconds
- Glycogen–lactic acid system →1.3 to 1.6 minutes
- Aerobic → unlimited
- Oxygen debt → (about 11.5 liters).

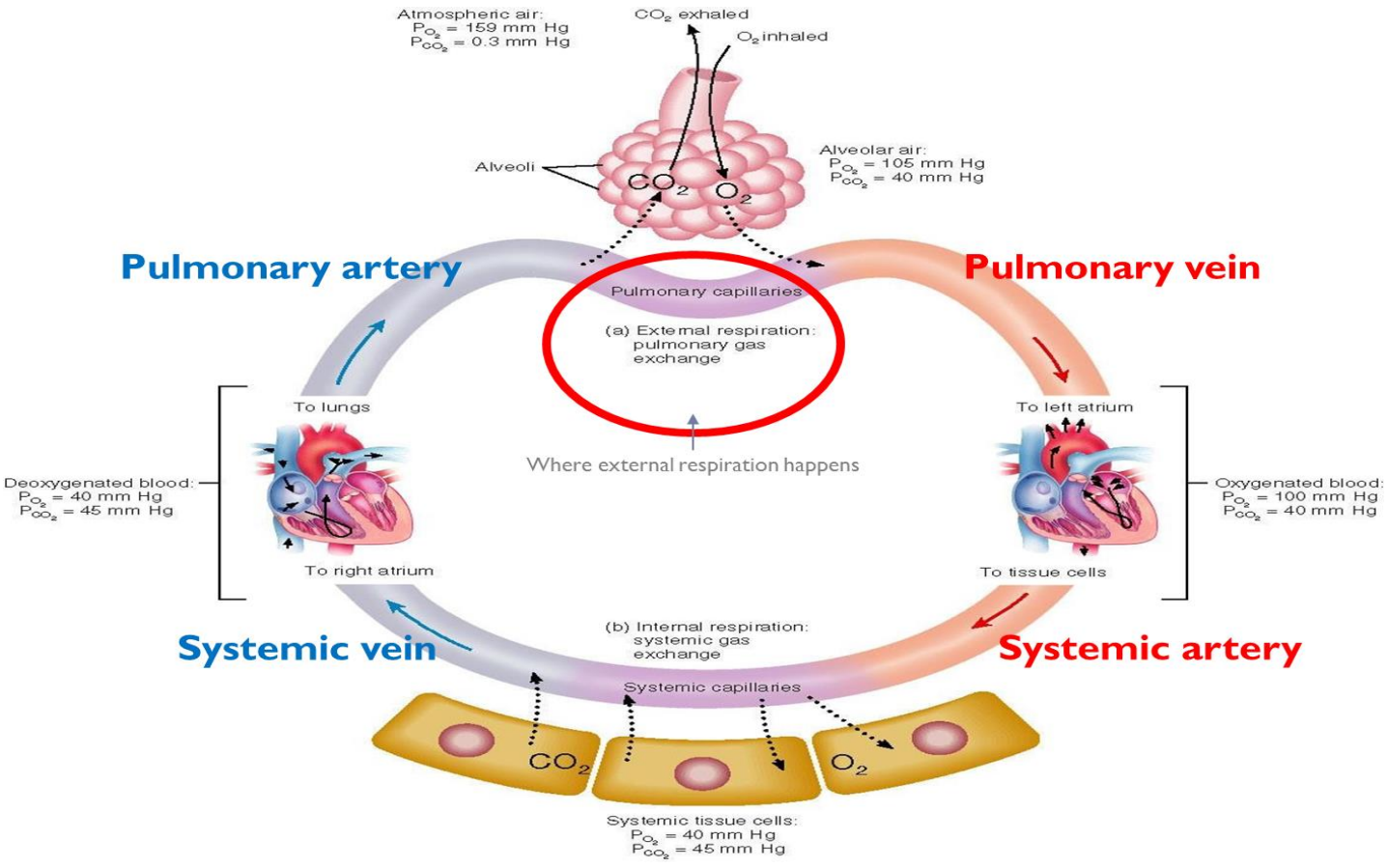
# Lecture#6

## Gas Transfer (Diffusion of O2 and CO2)

### Gas Exchange Through the Respiratory Membrane

- Gas exchange happens between alveolus and capillaries that surround it.
- Both the alveoli and capillaries have to be patent (open) and functioning for gas exchange to occur.
- If one of them was not patent (collapsed), diffusion will not happen.

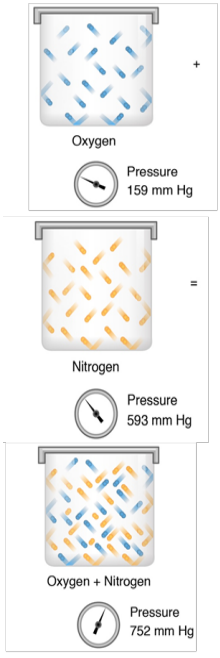


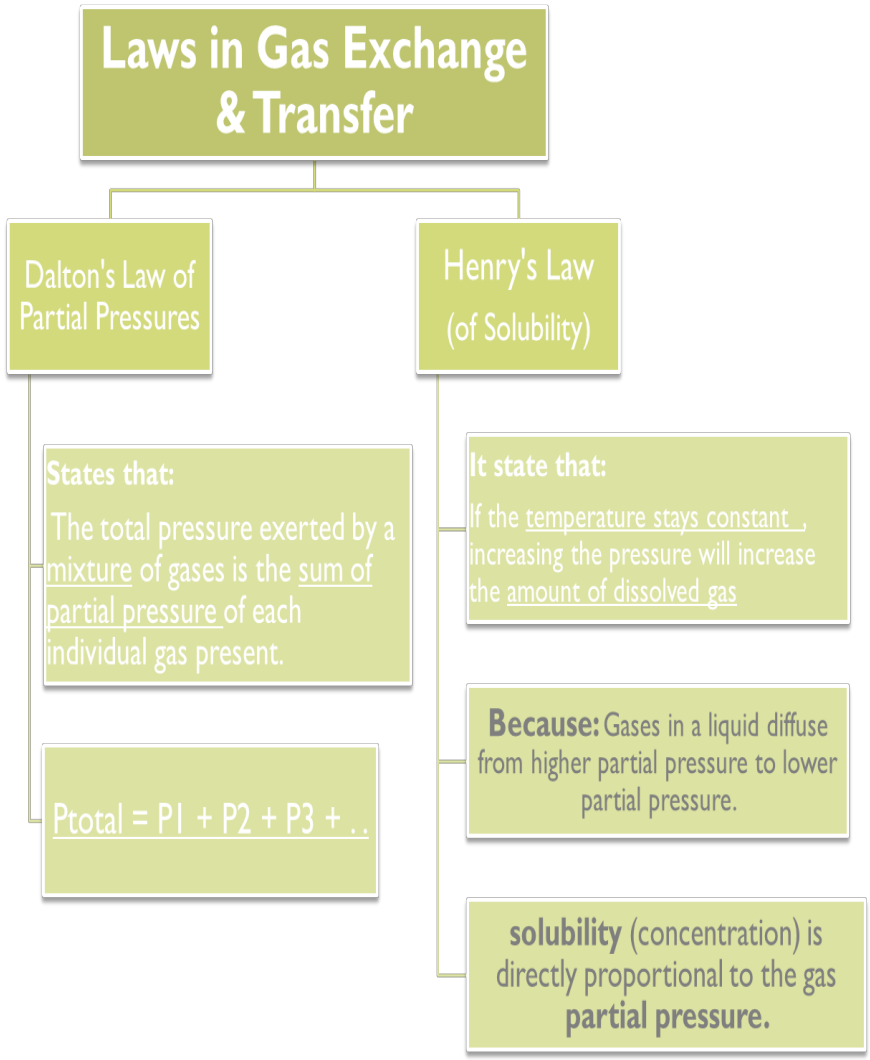



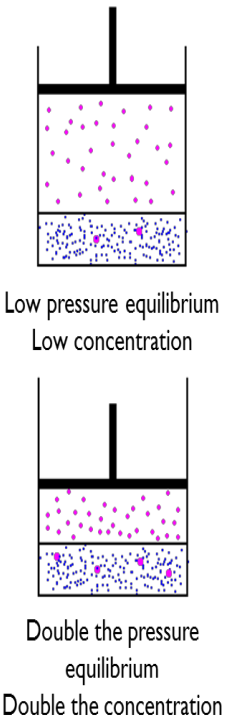

23.18

# Partial Pressure of Gases (in a Mixture)

- It is caused by: the constant kinetic movement of gas molecules against the surface.
- The 3 main gases (mixture) in respiratory physiology: $O_2$ , $N_2$, and $CO_2$.
- The rate of **diffusion** of each of these gases: is directly proportional with the partial pressure of the gas.
- Pressure of gases dissolved in water and tissue:
  1. The **pressure** of gases dissolved in fluid is **similar** to their **pressure** in the gaseous phase and
  2. They exert their own individual partial pressure.



# Factors effecting gas diffusion..Important

$$D \propto \frac{\Delta P \times A \times S}{d \times \sqrt{MW}}$$

- **D =** diffusion rate.
- $\Delta P$ **=** Partial pressure difference.

**(increase partial pressure = increase diffusion)**

- $A$**=** surface area for gas exchange

**(increase surface area = increase diffusion)**

The surface area can decrease:

In alveoli: with trypsin enzyme or by obstruction of some bronchioles by mucus or tumor.

In pulmonary capillaries: by thrombus or blood clot.

- $S$**= solubility of gas.**

**( increase solubility of gas=increase the diffusion)**

**Co2 is more soluble than O2, so it more diffusible.**

- **d= distance=thickness of respiratory membrane**.

**(Increase Distance= Decrease diffusion)**

**How do we increase the thickness of respiratory membrane?**

**By accumulation of fibrous tissue. For example:** people who have interstitial pulmonary fibrosis.

**By increasing extra cellular fluid between alveoli or even inside the alveoli. For example:** people who have Edema.

**MW= molecular weight.**

**( increase MW = decrease in diffusion)**

# Cont….

## P: Partial Pressure Differences:

- The difference in gas pressure between the two sides of the membrane (between the alveoli and the capillary blood).
- This difference dictates the direction of diffusion:
  - **For Oxygen:** the pressure of the gas in the alveoli is greater than the pressure of the gas in the blood, so the gas will diffuse from the alveoli into the blood. (from high to low)
    - $PO_2$ in alveoli = 104, In blood = 40
  - **For $CO_2$:** the pressure of the gas in the blood is greater than the pressure in the alveoli, net diffusion from the blood into the alveoli occurs.
    - $PCO_2$ in blood = 45, In alveoli = 40.

## A: Surface Area of the Membrane:

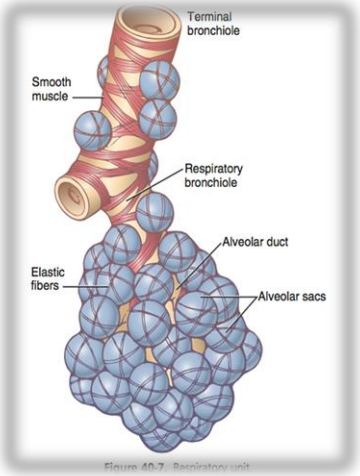
- Average surface area is 70 $m^2$. in normal adult.
- Removal of an entire lung decreases the surface area to half normal.
- In emphysema (usually due to heavy smoking) with dissolution (loss) of the alveolar walls → decrease surface area to 5-folds.

## D: Diffusion Distance (the Thickness of the Respiratory Membrane):

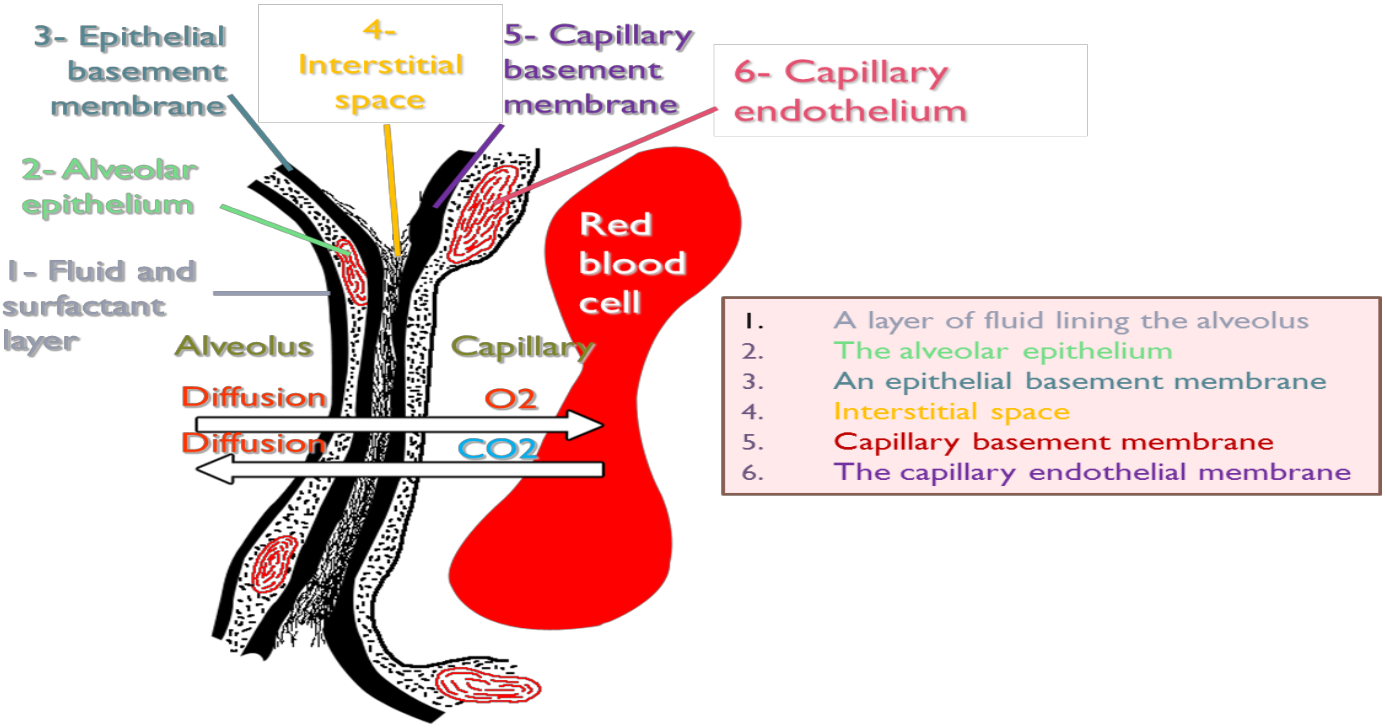
- Membrane thickness is almost 0.5 µm (very thin) and presents little obstacle to diffusion.
- If the membrane thickens, diffusion will take more time + gas exchange is inhibited (inversely proportional) like during exercise.
- Higher thickness like in edema, infection, or fluid effusion → lower rate of diffusion.
- **The thickness of the respiratory membrane is inversely proportional to the rate of diffusion through the membrane.**

# Respiratory Unit

- Also called: respiratory lobule.
- Composed of: a respiratory bronchiole, alveolar ducts, atria, and alveoli.
- Number of alveoli in 2 lungs: about 300 million alveoli in the two lungs.
- Alveoli diameter: an average of 0.2 millimeters.
- Respiratory membrane thickness: in some areas it is as little as 0.2 micrometer, on average it is 0.6 micrometers.
- Total quantity of blood in the capillaries of the lungs: is (60 to 140 milliliters).




## Layers of Respiratory Membrane (Alveolus) From Inside Out



# Partial Pressures of Respiratory Gases as They Enter and Leave the Lungs (at Sea Level)

| | $N_2$ | $O_2$ | $CO_2$ | $H_2O$ |
|---|---|---|---|---|
| Atmospheric Air (mmHg) | 597.0 (78.62%) | 159.0 (20.84%) | 0.3 (0.04%) | 3.7 (0.50%) |
| Humidified Air (mmHg) = conductive zone or anatomical dead space. | 563.4 (74.09%) | 149.3 (19.67%) | 0.3 (0.04%) | 47.0 (6.20%) Because it is humidified |
| Alveolar Air (mmHg) = respiratory zone. | 569.0 (74.9%) | 104.0 (13.6%) | 40.0 (5.3%) | 47.0 (6.2%) |
| Expired Air (mmHg) | 566.0 (74.5%) | 120.0 (15.7%) | 27.0 (3.6%) | 47.0 (6.2%) |

- **O2** concentration in the atmosphere is 21%
- PO2 in atmosphere = 760mmHg x 21% = 160 mmHg.
- This mixes with "old" air already present in alveolus to arrive at PO2 of 104 mmHg in alveoli. (drop)

- **CO2** concentration in the atmosphere is 0.04%
- PCO2 in atmosphere = 760mmHg x 0.04% = 0.3 mmHg
- This mixes with high CO2 levels from residual volume in the alveoli to arrive at PCO2 of 40 mmHg in the alveoli. (Increase)

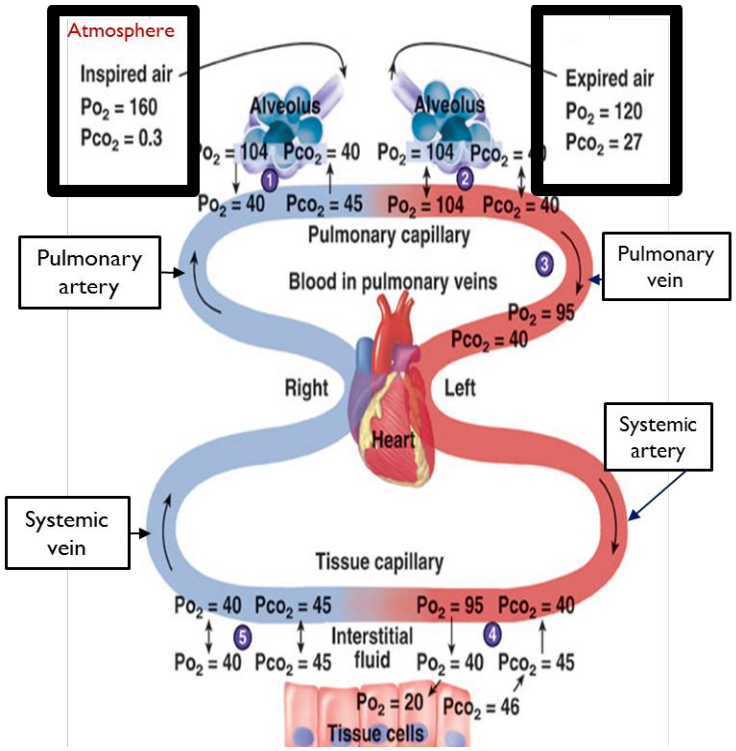
# PO2 and PCO2 in Various Potions of Normal Expired Air

## PO2:

1. Diffusion from atmosphere into alveolus.
2. Diffusion from alveolus into pulmonary blood:
   - In Alveolus = 104
   - In venous (pulmonary) blood = 40
   - Pressure difference of O2 between alveoli and blood is : 104-40 = 64mmHg
3. Diffusion from Capillaries into interstitial fluid:
   - In arterial (end)blood = 95
   - In interstitial = 40
4. Diffusion from interstitial fluid into cells:
   - In interstitial = 40
   - Inside cell = 20 (females' slides) 23 (males' slides).

## PCO2:

1. Diffusion from cell into interstitial fluid:
   - Inside cell = 46
   - In interstitial = 45
2. Diffusion from interstitial fluid into capillaries:
   - In interstitial = 45
   - In venous blood = 40
3. Diffusion pulmonary into alveoli:
   - In pulmonary blood = 45
   - In Alveolus = 40
4. Diffusion from alveolus into Atmosphere



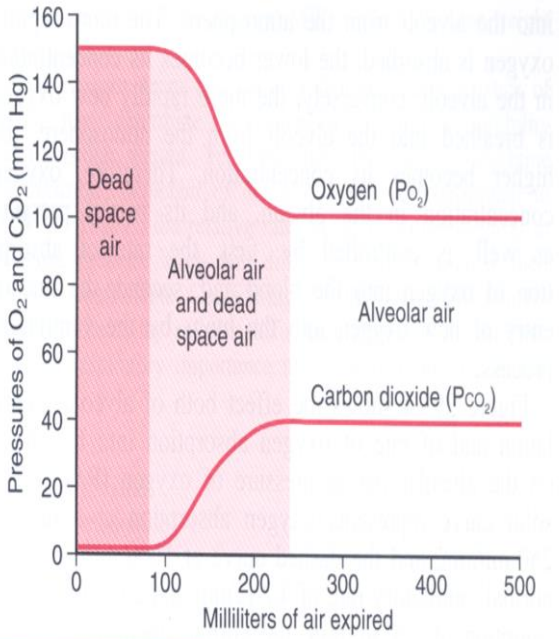
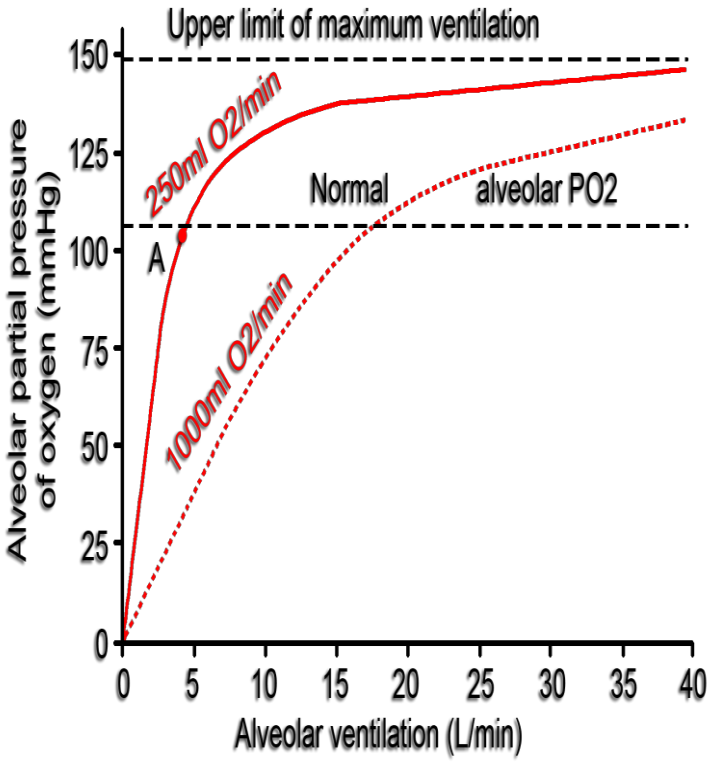




FIGURE 39-6

Oxygen and carbon dioxide partial pressures in the various portions of normal expired air.

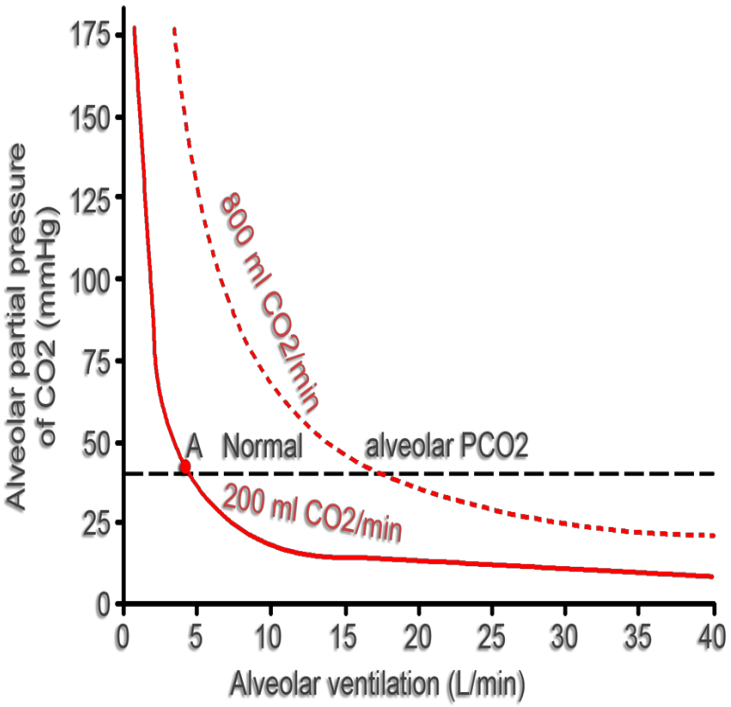
# O2 Concentrations in the Alveoli.

- At resting condition: 250 ml of **oxygen** enters the pulmonary capillaries per minute (normal rate), as shown in the solid curve.
- At normal ventilation rate: 4.2 L/min.
- **Alveolar PO2** is at point A at 104 mmHg
- During exercise: 1000 ml of **oxygen** is absorbed by the pulmonary capillaries/min, as represented by the dotted curve.
- The alveolar (pulmonary) ventilation rate must increase 4 times (folds) to maintain the **alveolar PO2** (partial pressure) at the normal value of 104 mmHg.




# CO2 Concentrations in the Alveoli

- At resting condition: 200ml of **CO2** is excreted per minute (normal rate), as shown in the solid curve.
- At normal ventilation rate: 4.2 L/min.
- The operating point for **Alveolar $PCO_2$** is at point A at 40mmHg.
- During exercise: 800 ml of **CO2** is excreted per minute, as represented by the dotted curve (Also 4 folds).
- **Alveolar $PCO_2$** increases directly in proportion to the rate of **$CO_2$ excretion.**
- **Alveolar $PCO_2$** decreases in inverse proportion to **alveolar ventilation.**



# Lecture#7

## TRANSPORT OF OXYGEN AND CARBON DIOXIDE

**Function of blood (main) :** Transport of respiratory gases between the lungs and body.

Hb. + Oxygen = Oxyhaemoglobin

**Binding :**

- O2 binds loosely and reversibly to Haem portion on hemoglobin.

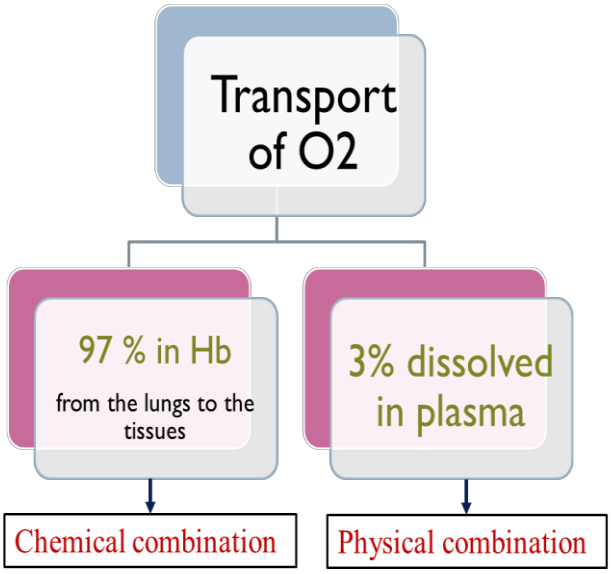
**Haem part :**

- The haem part of the Hb contains 4 atoms of iron → able to combine with a molecule of oxygen.

**Result :**

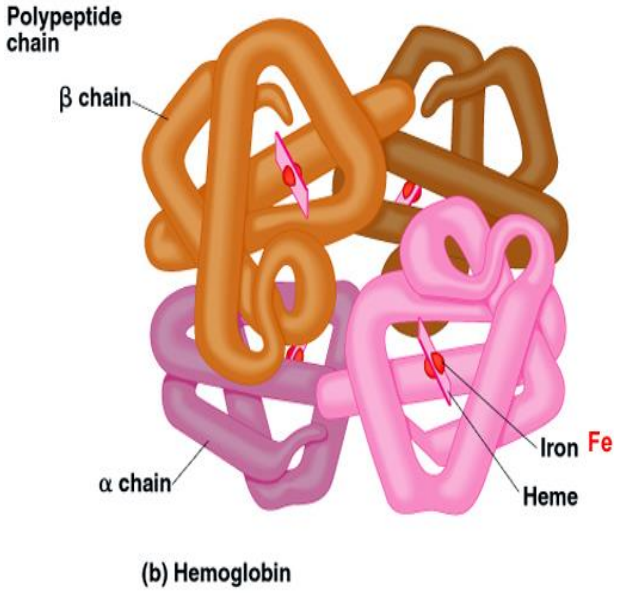
- Hemoglobin can carry 4 molecules of O2 or 8 atoms of O2

$$Hb + 4O_2 \rightarrow Hb(O_2)_4$$




# Binding to Hemoglobin

- The amount of $O_2$ carried in the blood in oxyhaemoglobin depends on : the amount of Hb present in the blood.
- The normal amount of Hb in young adults :14-16 gm/dl of the blood.
- Each gram of Hb can bind with : 1.34 ml of $O_2$.
- Thus, if a person's Hb is 16 gm/dl of blood his blood can carry :
- 16 x 1.34 = 21.44 ml of $O_2$/dl.



(b) Hemoglobin

# TRANSPORT OF O2

**Oxygen combining with Hb is determined by : $PO_2$.**

- If the PO2 is high : oxygen binds with Hb .
- result in : Greater Hb saturation
- Example: pulmonary capillaries
- At normal arterial PO2 of 95 mmHg , about 0.29 ml of oxygen is dissolved in each 100ml of blood.

- If the PO2 is low ,: oxygen is released from Hb
- result in : Lesser Hb saturation
- Example : tissue capillaries

When the PO2 of the blood falls to 40 mmHg in tissue capillaries, only 0.12 of oxygen remains dissolved.
Therefore 0.17 ml of oxygen is normally transported in the dissolved state to the tissues per each 100 ml of blood

## O2 capacity, content and saturation. Important

- **O2 content :** amount of O2 in blood (ml O2/100 ml blood)
- **O2-binding capacity :** maximum amount of O2 bound to hemoglobin (ml O2/100 ml blood) measured at 100% saturation.
- **Percent saturation :** percentage ( % ) of heme groups bound to O2
- **Percentage of saturation of Hb** $= \frac{\text{oxygen content}}{\text{oxygen capacity}} \times 100$
- **Dissolved O2 :** Unbound O2 in blood (ml O2/100 ml blood).

# Transport of oxygen in arterial blood

- Blood is 100% saturated with O2 : each gram of Hb carry 1.34 ml O2.
  - So →O2 content = 15g Hb x 1.34 O2 = 20 ml.
- Blood is only 97% saturated with O2 : contain 19.4 ml O2\ 100 ml blood ).
- Amount of oxygen released from the hemoglobin to the tissues : is 5ml O2 / 100ml blood.
  - So →O2 content in venous blood =19.4 - 5= 14.4 ml.
- During strenuous (hard) exercise: the oxygen uptake by the tissue increases 3 folds so 5x3=15 ml O2 /100 ml blood
  - So→ O2 content in venous blood =19.4 -15 = 4.4 ml O2 /100ml blood.
- At rest : tissues (our bodies) consume 250 ml O2 /min and produce 200 ml CO2

# Transport of carbon dioxide in the blood

- $CO_2$ produced in :large amount produced in the body .
- CO2 + globin part = carbaminohemoglobin
- Remember : O2 + Haem part = Oxyhaemoglobin
- $CO_2$ in normal resting conditions :

  4ml of $CO_2$ \ 100 ml of deoxygenated blood
- $CO_2$ which is carried in the blood in three forms:
  1. 70% of $CO_2$ is transported in bicarbonate form.
  2. 23% combines with the globin part of Hb to form carbamino-haemoglobin.
  3. 7% is dissolved in plasma

# Cont….

## 1- CO2 transport (bicorbonate) and chloride shift.

**Tissue Capillaries**

1 Co2 + water (in RBC) —Carbonic Anhydrase→ H2CO3 (carbonic acid)

2 H2CO3 —dissociates→ HCO3 (bicorbonate)+ H+

3 H ion + hemoglobin → hydrogenhemoglobin

Bicorbonate gets out of RBC, and Cl enters to maintain negative charge of cell ( bicorbonate shift phenomena).
If chloride doesn't enter the cell will shrink.

**Pulmonary Cappilaries**

1 HCO3 (bicorbonate)+ H+ → H2CO3

2 H2CO3 —Carbonic Anhydrase→ Co2 + water

Bicorbonate enters RBC, Cl gets out to maintain negative charge of cell.

## 2- Transport of CO2 Combined with Hb

**CO2 (carbon monoxide) binding :**

- Co2 combines with Hb at the same point on the Hb molecule as does oxygen.
- It binds with Hb about 250 times as much as O2 (affinity of Hb to CO is (250 times) that to O2).
- It causes Left shift of the O2-Hb curve (discussed later )

## 3- Transport of CO2 dissolved in plasma (7%)

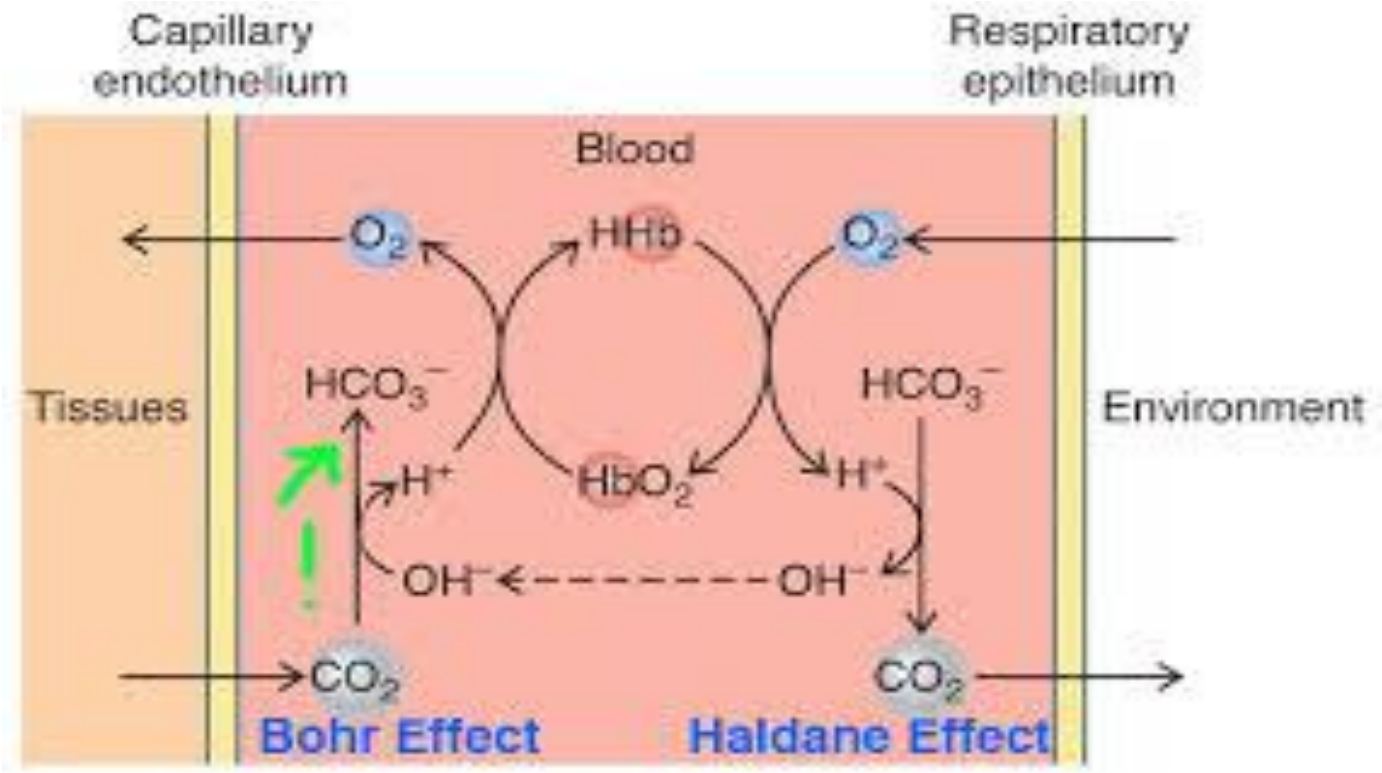
- $PCO_2$ of venous blood : is 45 mm Hg .
- $PCO_2$ of arterial blood : is 40 mmHg.
- The amount of $CO_2$ dissolved in the blood at 45 mmHg is 2.7 ml/dl (2.7%).
- The amount of $CO_2$ dissolved at 40 mmHg is about 2.4 ml.
- The difference between 2.7 and 2.4 is only 0.3 ml.
- About 0.3 ml $CO_2$ / 100 ml of blood in the form of dissolved $CO_2$

# The Haldane effect

- When oxygen binds with hemoglobin , carbon dioxide is released- to increase CO2 transport.
- Binding of Hb with O2 at the lung causes the Hb to become a stronger acid and , this in turn displaces CO2 from the blood and into the alveoli.
- Change in blood acidity during CO2 transport:

  Arterial blood has a pH of 7.41, and the pH of venous blood (which has higher PCO2) falls to 7.37 (i.e change of 0.04 unit takes place).

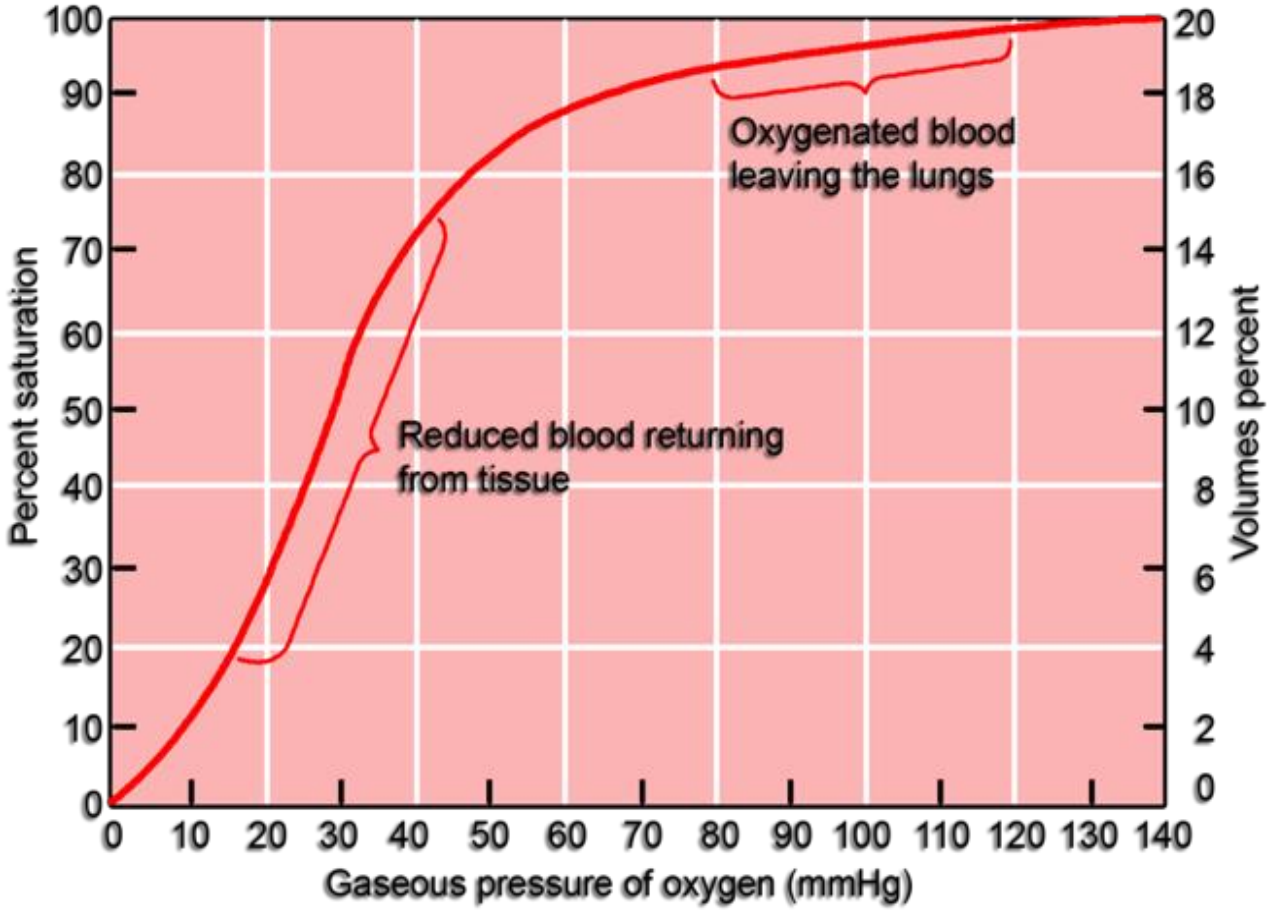
- $R = \frac{\text{Rate of CO2 output}}{\text{Rate of O2 uptake}}$
- Normally (normal diet) it is 4/5 (200\250) = 82% =0.825
- When Carbohydrate diet is used , R = 1
- When fats only is used , R=0.7



# Oxygen-hemoglobin dissociation curve.. Important

- **Curve Shape:** is S - shaped or sigmoid (not linear)
- **Represent:** relationship between the percent $O_2$ saturation of Hb and the partial pressure of $O_2$ (PO2)
- This curve is called the oxyhemoglobin saturation curve.
- **Relation:** shows progressive increase in the percentage saturation of the Hb with the increase in the PO2 in the blood.
- Higher PO2 results in greater Hb saturation.

| | Arterial blood | Venous blood (returning from the tissues) |
|---|---|---|
| PO2 | 95 mmHg | 40 mmHg |
| saturation of Hb with O2 | 97%. | 75%. |



# Shift of Oxygen-Hemoglobin dissociation curve

## Shifted to the left

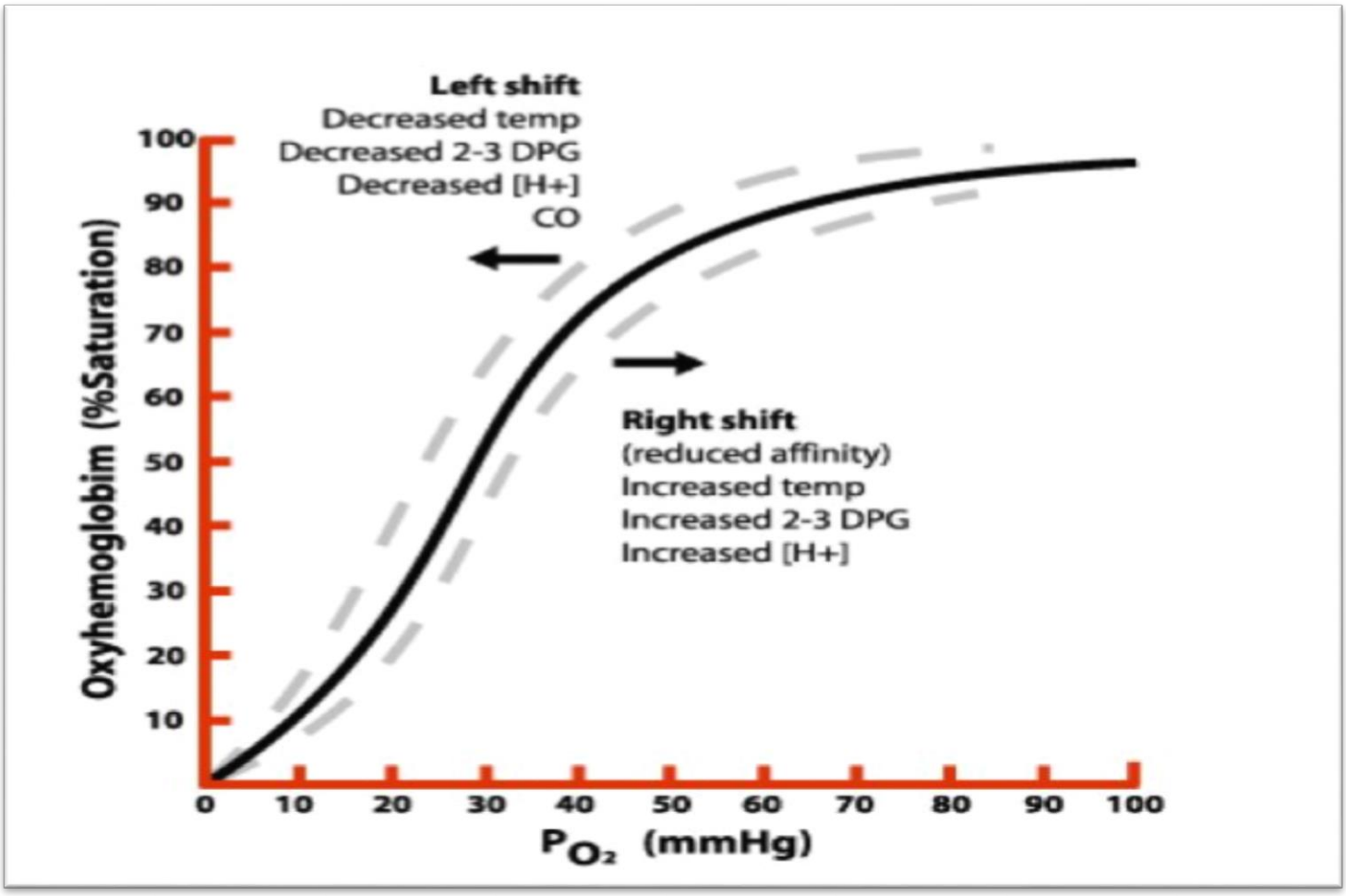
Means :

- Hb affinity for oxygen is increased
- loading or attachment of oxygen to Hb.

## Shifted to the right

Means :

- O2 is dissociated or released (unloaded) to the tissues from Hb



# Cont…

## Factors shifting to the Left

- Increasing affinity

1. Decreased CO2 (Bohr effect)
2. Decreased hydrogen ions ( PH )
3. Decreased temperature
4. Decreased 2,3 DPG
5. Fetal haemoglobin (coming next)

## Factors shifting to the Right

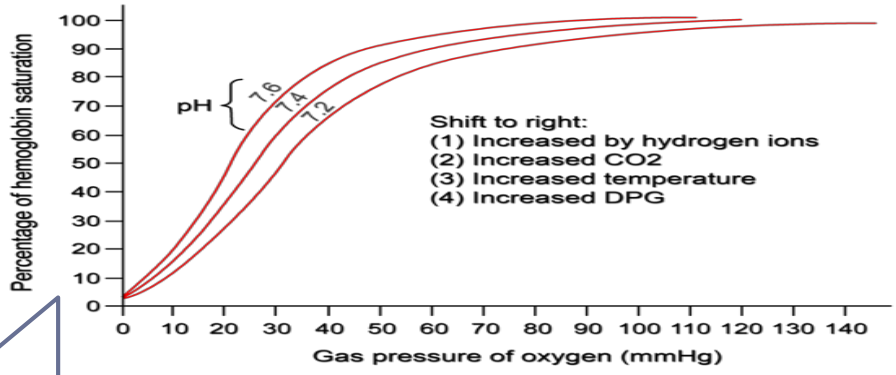
- Decreasing affinity

1. Increased PCO2 (Bohr effect)
2. Increased hydrogen ions ( PH )
3. Increased temperature
4. Increased 2, 3 DPG :
   - synthesized in RBCs from the glycolytic pathway
   - facilitate the oxygen release
   - binds tightly to reduced Hb.

**Anemia and hypoxemia :**
2,3 DPG increases in the RBCs in case of these diseases → serves as an important adaptive response in maintaining tissue oxygenation.

# (Bohr effect)

- Effect of CO2 and H ions on the curve (Bohr effect)




**At lung:**

Movement of CO2 from blood to alveoli will decrease blood CO2 &H+ → shift the curve to left.

Increased O2 affinity of Hb allowing more O2 transportation into the tissue.

**At tissues:**

- Increase CO2 &H+ in blood leads to →shift the curve to Right.
- Decreased O2 affinity of Hb allowing more O2 transportation to the tissues.

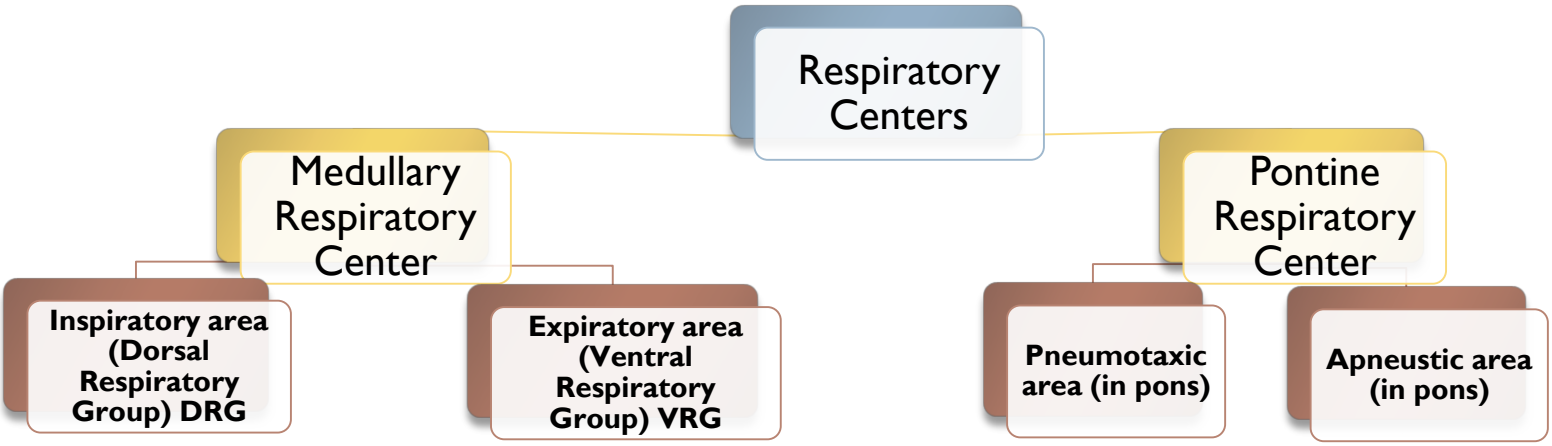
# Shift of dissociation curve during exercise

- Fetal Hb: has a P50 of 20 mmHg in comparison to 27 mmHg of adult Hb.
- **Utilization Coefficient** :The percentage of the blood that gives up its oxygen as it passes through the tissues capillaries
- Utilization Coefficient: $= \frac{\text{O2 delivered to the tissues}}{\text{O2 content of arterial blood}}$
- Normally at rest = 5 ml/20 m, which = 25% .
- During exercise = 15 ml/20 ml, which = 75 % - 85%

# Lecture #8
# Control of Breathing

## Controls of Rate and Depth of Respiration

- Arterial PO2
  - When PO2 is VERY low (Hypoxia), ventilation increases.
- Arterial PCO2
  - The most important regulator of ventilation is PCO2, small increases in PCO2, greatly increases ventilation.
  - Changes in PCO2 stimulate the respiratory center immediately. Unlike Po2 because the accumulation of CO2 is very dangerous for the body.
- Arterial pH: As hydrogen ions increase (acidosis), alveolar ventilation increases.




- Determines basic rhythm of breathing.
- Causes contraction of diaphragm and external intercostals.
- It will stimulate the phrenic nerve which supplies the diaphragm.

- Inactive during normal quiet breathing.
- Activated by inspiratory area during forceful breathing.
- Causes contraction of internal intercostals and abdominal muscles.

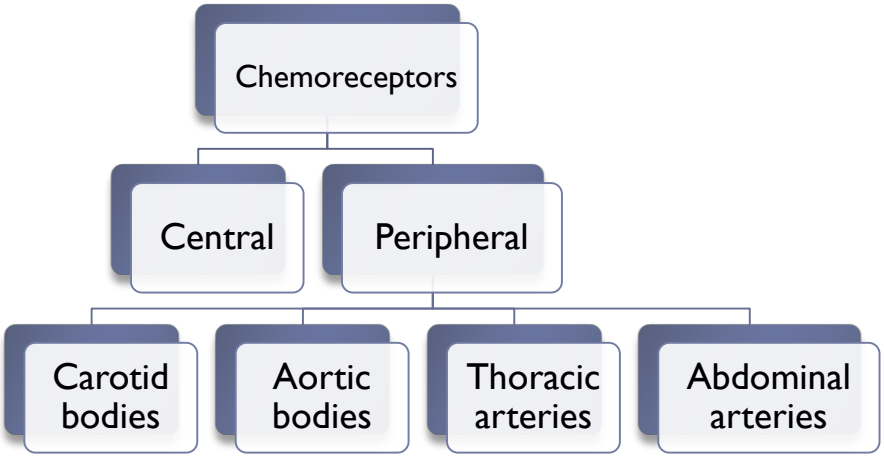
**Inhibits** inspiratory area of medulla to stop inhalation. Breathing is more rapid when pneumotaxic area is active.

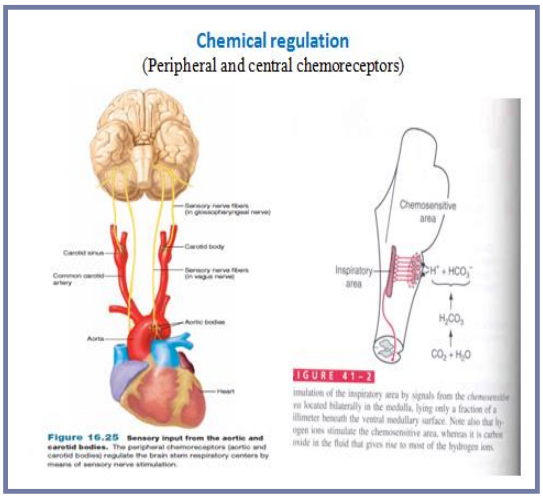
**Stimulates** inspiratory area of medulla to prolong inhalation.

The medullary respiratory center stimulates basic inspiration for about 2 seconds and then basic expiration for about 3 seconds (5sec/ breath = 12breaths/min).

Pontine Respiratory center is the transition between inhalation and exhalation it is controlled by pneumotaxic and apneustic areas.

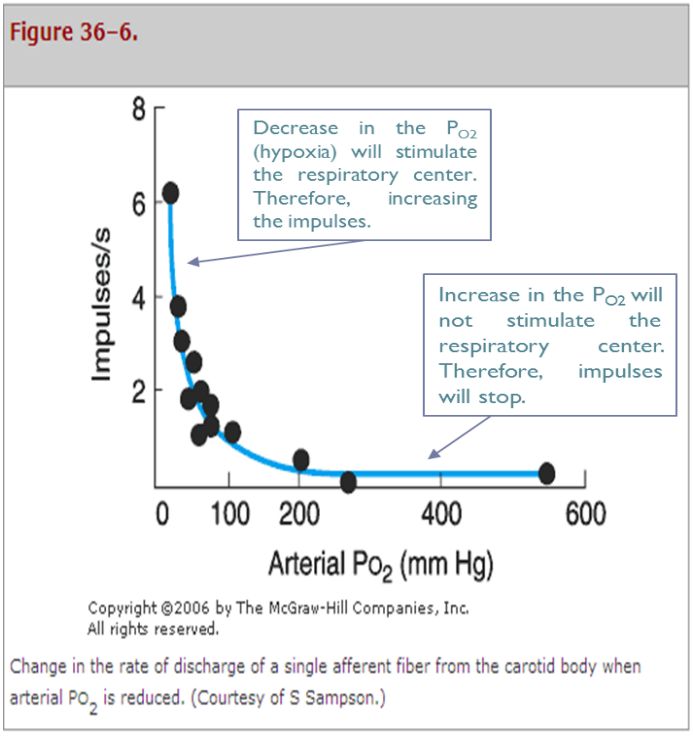
# Chemoreceptors

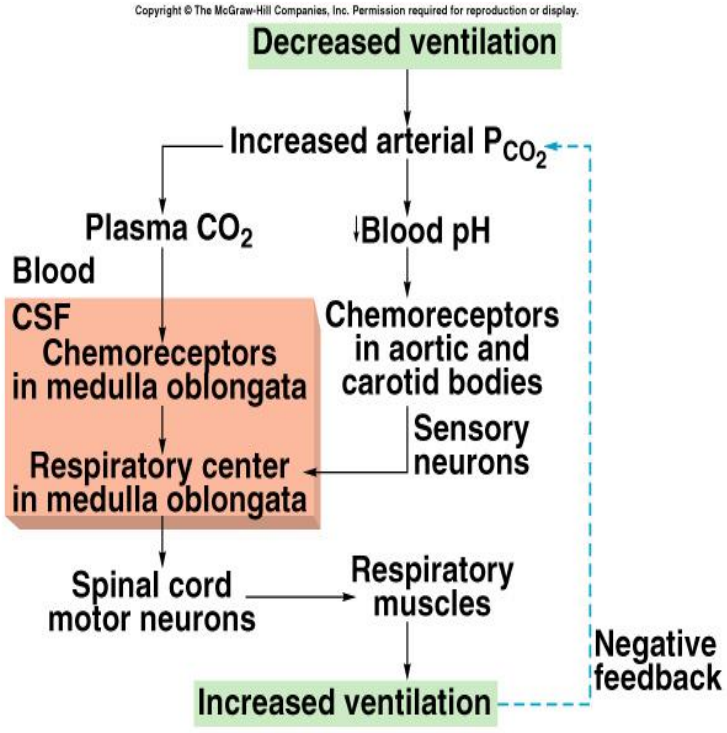



Most of the chemoreceptors are in the *carotid bodies*. However, a few are also in the *aortic bodies*, and very few are located elsewhere in association with other arteries of the thoracic and abdominal regions.

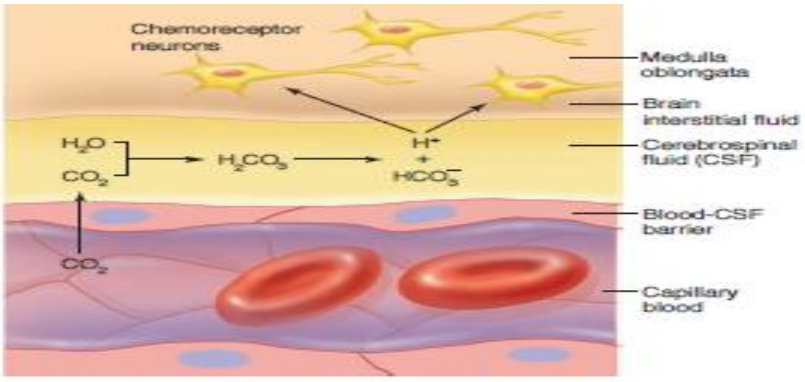
1- Excess $CO_2$, $H^+$ in the blood mainly acts directly →Central
2- Others in arteries of the thoracic and abdominal regions →Peripheral



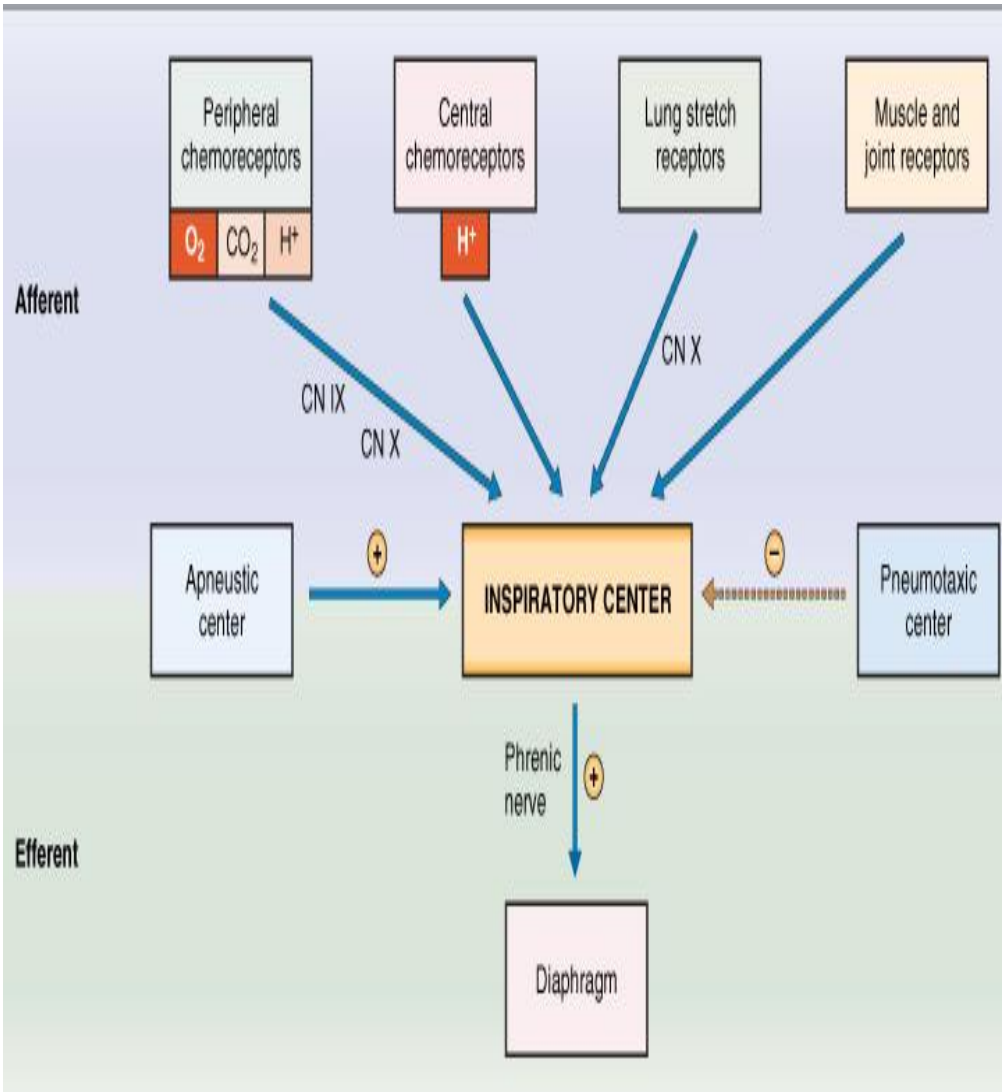



# Effect of Blood CO2 Level on Central Chemoreceptors

An increase in blood $CO_2$ stimulates breathing indirectly by lowering the pH of blood and cerebrospinal fluid (CSF). This figure illustrates how a rise in blood $CO_2$ increases $H^+$ concentration of CSF and thereby stimulates chemoreceptor neurons in the medulla oblongata.




# Factors Affecting Respiration




## Effect of irritant receptors in the airways:

The epithelium of trachea, bronchi, and bronchioles is supplied by irritant receptors that are stimulated by irritants (i.e. dust) that enter the respiratory airways causing coughing, sneezing, and bronchoconstriction in bronchial asthma and emphysema.

## Function of lung J receptors:

Few receptors in the wall of the alveoli in juxta position to the pulmonary capillaries. They are stimulated especially when pulmonary capillaries become engorged by blood or when pulmonary edema occurs i.e. in congestive heart failure (CHF), their excitation cause the person a feeling of dyspnea.

# Hering-Breuer Inflation Reflex

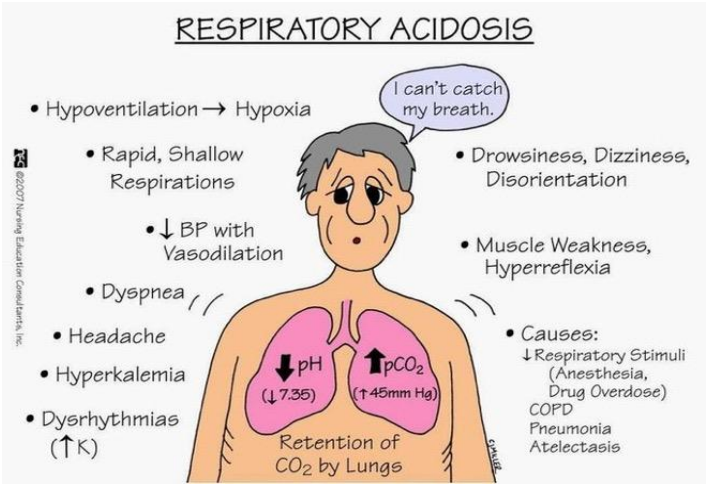
- When the lung becomes overstretched (tidal volume is 1L or more), stretch receptors located in the wall of bronchi and bronchioles transmit signals through vagus nerve to DRG producing an effect similar to **pneumotaxic center** stimulation:

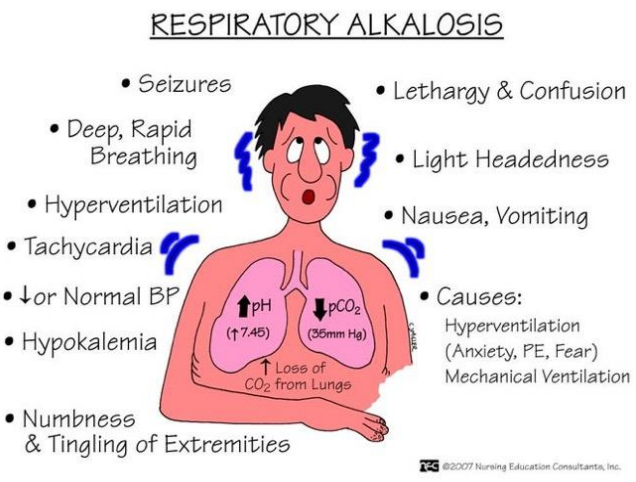
1. Switches off inspiratory signals and thus stops further inspiration.
2. This reflex also increases the rate of respiration as does the pneumotaxic center.

| Respiratory Acidosis* | Respiratory Alkalosis* | Metabolic Acidosis* | Metabolic Alkalosis* |
|---|---|---|---|
| ▸ Hypoventilation.<br>▸ Accumulation of $CO_2$ in the tissues.<br>▸ $P_{CO2}$ increases.<br>▸ pH decreases.<br>▸ Acidity increases. | ▸ Hyperventilation.<br>▸ Excessive loss of $CO_2$.<br>▸ $P_{CO2}$ decreases (35 mmHg).<br>▸ pH increases.<br>▸ Acidity decreases. | ✓ Ingestion, infusion, or production of fixed acid.<br>✓ Decreased renal excretion of hydrogen ions.<br>✓ Loss of bicarbonate or other bases from the extracellular compartment. | ✓ Excessive loss of fixed acids from the body.<br>✓ Ingestion, infusion, or excessive renal reabsorption of bases such as bicarbonate.<br>✓ pH increases. |

Acidosis* means an increase in Acidity or a Decrease in pH of the blood.
Alkalosis* means a decrease in Acidity or an Increase in pH of the blood.

The respiratory system can compensate for metabolic acidosis or alkalosis by altering alveolar ventilation.
If the patient has ACIDOSIS the respiratory system will respond by HYPERVENTILATION, if he has ALKALOSIS the response will be HYPOVENTILATION.



# Lecture #9
# Hypoxia and cyanosis

**Hypoxia** is defined as an inadequate supply (deficiency) of oxygen to the body tissues.

## Histotoxic hypoxia

This is caused by inhibition of the tissues' use of oxygen even though plenty of oxygen is available due to inhibition of the oxidative enzyme activity in respiration electron transport chain.

The best example is cyanide poisoning, where tissue cytochrome oxidases are knocked out (blocked) and tissue is unable to utilize oxygen.

## Stagnant hypoxia (Hypokinetic or Ischemic hypoxia)

Reduced blood flow through the body or part of the body (tissue). Due to slow circulation less oxygen is carried by the blood at the lung, leading to hypoxia.

It may be caused by congestive heart failure, circulatory shock and arteriosclerosis.

- General slowing of circulation (heart failure and shock)
- Local slowing: vasoconstriction, cold, arterial wall spasm.

## Anemic hypoxia

In this condition the PO2 and %Hb-O2 are normal.

This condition is characterized by decreased oxygen carrying capacity of the blood due to decreased hemoglobin level or abnormal type of Hb which is unable to carry oxygen.

### Causes:

1. Anemia
2. Abnormal Hb
3. Anemic hypoxia is seen in hemorrhagic anemia: Failure of hemoglobin to carry its normal concentration of oxygen, as in carbon monoxide (CO) poisoning.
4. Altered hemoglobin formation

## Hypoxic hypoxia (arterial hypoxia)

It is defined as a lack of oxygenation of blood in the lungs which leads to a low PO2 in arterial blood (reduced arterial PO2). Since less amounts of Hb are converted into oxy-Hb The tissues are supplied with blood deficient in oxygen. Hypoxic hypoxia can occur in

### the following conditions:

- High altitude
- Emphysema
- Fluid in the lungs (pulmonary edema)
- Obstruction in the respiratory passages
- Alveolar hypoventilation
- Diffusion abnormalities
- Right to left shunt
- V/Q mismatch

# General signs and symptoms of hypoxia

- *Cyanosis*, *Tachy-cardia* and *Tachypnea* (abnormally rapid breathing).

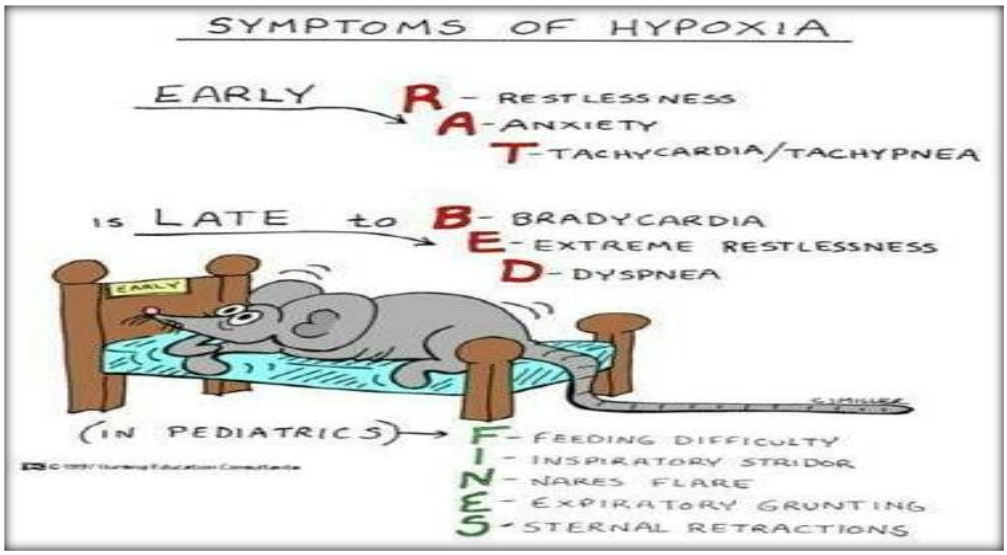
**Signs and symptoms of hypoxia:**

Hypoxia's clinical features depend on (the degree of hypoxia):

How fast & how severely the **partial pressure of $O_2$ is decreased**.

## Classification of hypoxia

| Fulminant hypoxia | Acute hypoxia | Chronic hypoxia |
|---|---|---|
| This occurs very rapidly &suddenly. Its symptoms : Unconsciousness occurs in 15-20 seconds and brain tissue death occurs in 4-5 minutes. | In acute hypoxia body reflexes are slowed, symptoms : there may be slurred (unclear) speech, unconsciousness, coma and death may occur. | The symptoms : fatigue, difficulty in breathing, and shortness of breath can occur. |

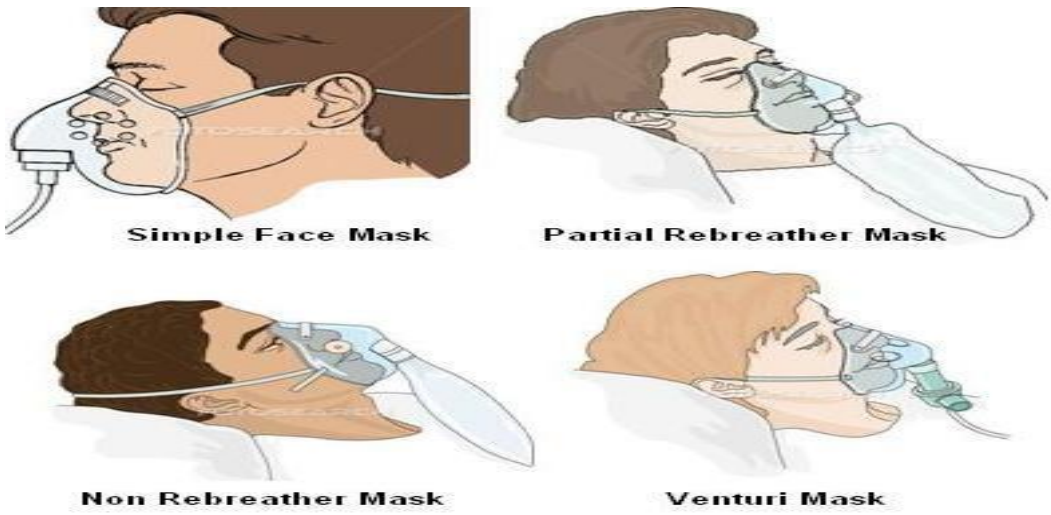


# Effects of Hypoxia on the Body

1. Impairment of judgment.
2. Inability to perform complex calculations.
3. Headache, nausea, irritability and dyspnea.
4. Increased heart rate.
5. Reduction in muscles working capacity.
6. Coma and death if severe.

# Treatment of Hypoxia

- Hypoxia is treated by giving oxygen therapy in a tent or high oxygen tension mask.
- This is useful in **hypoxic hypoxia**, but of less value in other types of hypoxia.
- **Histiotoxic hypoxia will not** benefit from O2 therapy.



# Hypercapnea (in Guyton the spelling is Hypercapnia)

- Excess of CO2 in body fluids.
- It usually occurs with hypoxia.
- PCO2 increases above 52 mmHg and therefore it decreases the pH.
- Features of hypercapnea:
  1. Peripheral vasodilatation.
  2. Sweating.
  3. Warm extremities and bounding pulse.
  4. Muscle twitching.
  5. Headache, drowsiness and coma
  6. Papilledema (swelling of optic disc).

# Cyanosis

Definition: The term cyanosis means blueness of the skin and mucus membrane due to excessive amount for more than 5 g/dl of reduced (deoxygenated) hemoglobin in blood (skin blood vessels especially in the capillaries).

This deoxygenated hemoglobin has an intense dark blue-purple color that is transmitted through the skin. (This is the cause of the blue skin color for people with cyanosis).

Cyanosis appears whenever the arterial blood contains more than 5 grams of deoxygenated hemoglobin in each 100 milliliters of blood.

- A person with anemia never becomes cyanotic because there is not enough hemoglobin for 5 grams to be deoxygenated in 100 milliliters of arterial blood.

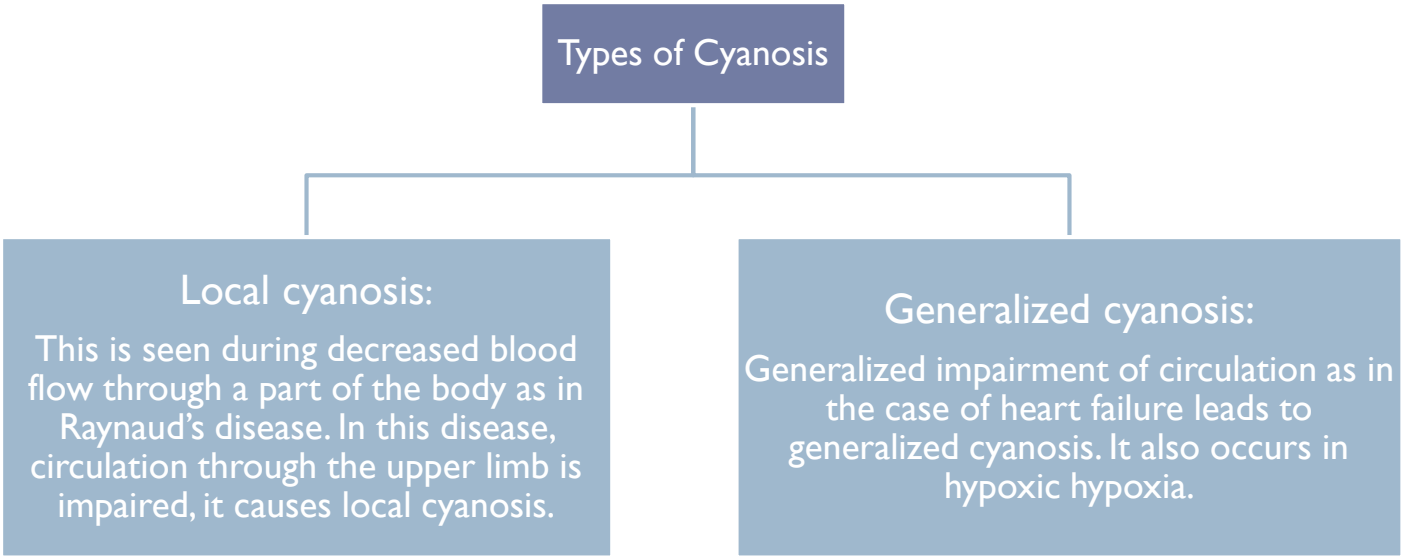
(Anemia → **Less** blood cells → **Less** hemoglobin to be deoxygenated → Cyanosis cannot be achieved due to the **lack of hemoglobin** and red blood cells).

- While In a person with excess red blood cells in the case of polycythemia, has a great excess of available hemoglobin that can become deoxygenated leads frequently to cyanosis.

(Polycythemia → **More** blood cells → **More** hemoglobin to be deoxygenated → Cyanosis can be achieved due to the **excess hemoglobin** and red blood cells.)

# Causes of Cyanosis

- Inadequate oxygenation of blood in the lungs:
  - High altitude.
  - Obstruction of respiratory passages.
  - Pneumoconiosis.
  - Emphysema.
  - CO-poisoning.
- Presence of an aerated shunt between vessels:
  - Coaractation of aorta.
  - Fallot's tetralogy.
- Other Causes:
  - Moderate cold.
  - Diminished blood flow to tissues.



# Ventilation-Perfusion Ratio (V/Q)

- Ventilation-Perfusion Ratio (VA/Q): This is the ratio of alveolar ventilation to the pulmonary blood flow per minute.
- Ventilation-perfusion ratio is expressed as VA/Q
- When VA (alveolar ventilation) is normal for a given alveolus and Q (blood flow) is also normal for the alveolus, the ventilation-perfusion ratio (VA/Q) is also said to be normal.
- Ventilation - Perfusion Ratio (V/Q): The alveolar ventilation at rest is about (4.2 L/minute)
- Calculated as: Alveolar ventilation = respiration rate x (tidal volume – dead space air)
- The pulmonary blood flow is equal to right ventricular output per minute (5 L/min). Hence ventilation perfusion ratio is (V/Q) = 4.2/ 5= 0.84

| Ventilation-perfusion ratio (V/Q) | | |
|---|---|---|
| Ventilation (V)<br>Alveolar<br>Minute ventilation = 4 to 6 L | Perfusion (Q)<br>Normal cardiac output = 5 L | Normal ventilation / perfusion ratio<br>(average)(V/Q ratio) = 0.8 to 1.2<br>At the apex V/Q ratio = 3<br>(moderate degree of physiologic dead space)<br>At the base V/Q ratio = 0.6<br>(represent a physiologic shunt).<br>*So the apex is more ventilated (gravity pulls it down) than perfused and the base is more perfused than ventilated.<br>*During exercise the V/Q ratio becomes more homogenous among different parts of the lung |

# Decrease in Ventilation-Perfusion Ratio (V/Q)

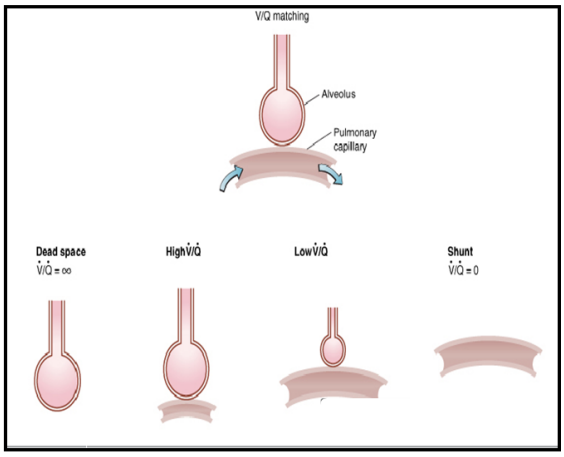
A decrease in V/Q ratio is produced by either decreasing ventilation or increasing blood flow.

**DECREASED VENTILATION**

- Bringing in less oxygen to meet metabolic need
- Blowing out less CO2 from the lungs

**INCREASED PERFUSION**

- More blood cells are coming to remove O2 from alveolus
- They deliver more CO2 than exhaled




# Increase in Ventilation-Perfusion Ratio (V/Q)

Either caused by increased ventilation or decreased perfusion.

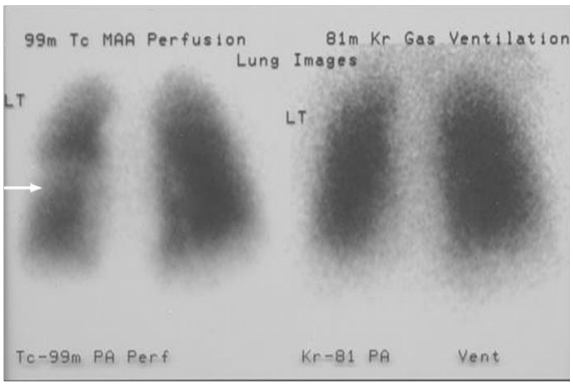
**INCREASED VENTILATION**

- Bringing in more oxygen to the alveoli
- Blowing out more CO2 from the lungs

**DECREASED PERFUSION**

- Blood takes away less oxygen
- Delivers less CO2

Ventilation- Perfusion Lung Scan



# Ventilation-Perfusion Ratio (V/Q)

- The normal value means on average lungs are over-perfused but under-ventilated at rest.
- The main function of this ratio is to determine the state of oxygenation in the body.
- Any mismatch in the ratio can result in hypoxia.
- During exercise: Less difference in pulmonary blood flow between basal and apical portions of the lungs occurs. This makes ventilation more closely matching perfusion of the lungs.

- The ratio is variable from apex to base of the lung
- As lung is centered vertically around the heart, part of the lung is superior to the heart, and part is inferior

High (V/Q) ratio of the apex leads to an increase in alveolar and arterial oxygen levels while decreasing the carbon dioxide. The blood leaving the apex of each lung in a standing person is estimated to have a $PaO_2$ of 130 mm Hg and a $PaCO_2$ of 28 mm Hg.

# Abnormalities of the V/Q Ratio

- When the V/Q ratio is less than normal this is called physiologic shunt (a certain fraction of the venous blood is passing through the pulmonary capillaries without being oxygenated i.e. shunted blood).
- When V/Q is more than normal this is called Physiologic dead space (when the ventilation of some of the alveoli is great but the alveolar blood flow is low, ventilation of these alveoli is wasted).
- In Chronic Obstructive Lung disease COPD because of bronchial obstruction in some areas and destruction of the alveolar septa in other areas with patent alveoli those people have some areas of the lung exhibit serious physiologic shunt and in other areas serious physiologic dead space.
- COPD is the most prevalent cause of pulmonary disability today, lung effectiveness as a gas exchange organ may decrease to 10%.

# Regional Blood Flow Distribution Important

## Zone 1

**alveolar air pressure > arterial and venous pressures**

- Vessels are collapsed and flow is close to zero

## Zone 2

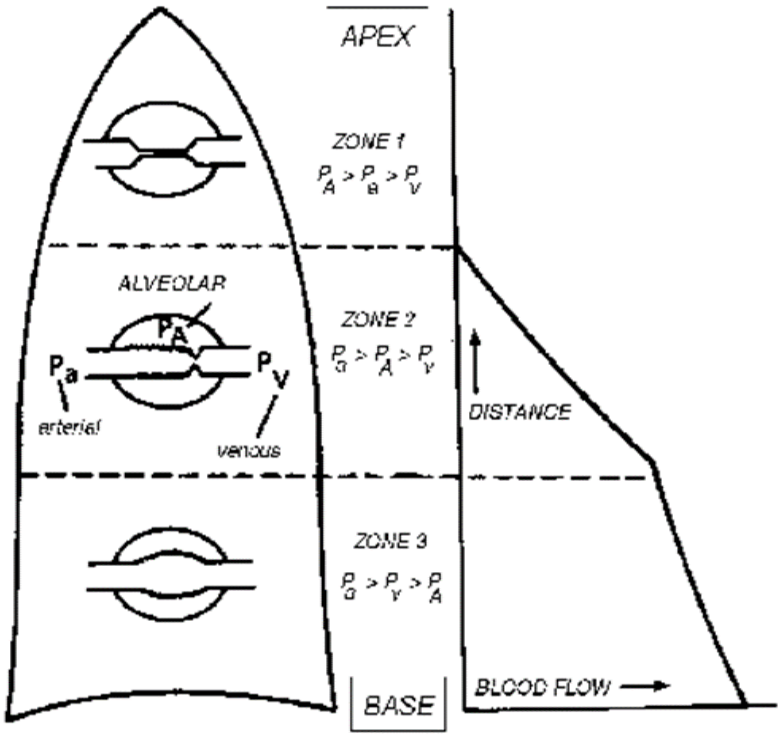
**venous pressure < alveolar air pressure < arterial pressure**

- vessels partially collapsed and flow is low

## Zone 3

**alveolar air pressure < arterial and venous pressure**

- vessels open for full length and flow highest



# Lecture #10

## Effects of Low and High Gas Pressure on the Body

## Effect of Increased Barometric Pressure (Deep Sea Diving)

When humans **descend below** the sea, the pressure around them **INCREASES**.

→ To prevent the lungs from collapsing, air must be supplied at high pressure

→ This exposes the blood in the lungs to extremely high alveolar gas pressure **(Hyperbolism).**

Under certain limits these high pressures cause tremendous alterations in the physiology of the body ( nitrogen narcosis).

The surrounding pressure increases by
1 atmosphere every 10 meters (33 feet) of depth in sea water
Therefore at a depth of 31 meters (100 feet) in the ocean the diver is exposed to a pressure of 4 atmospheres.
Therefore, a person 33 feet beneath the ocean surface is exposed to 2 atmospheres pressure, 1 atmosphere of pressure caused by the weight of the air above the water and the second atmosphere by the weight of the water itself.
At 66 feet, the pressure is 3 atmospheres, and so forth..
These problems confront SCUBA (Self Contained Under Water Breathing Apparatus)

### Effect of depth on the volume of the gases – Boyle's Law:

- Compression of gases to smaller and smaller volumes.
- 1L (sea level)→1/2 L at 33 feet and so on.
- Volume to which a given quantity of gas is compressed is inversely proportional to the pressure. .

### Effect of depth on the density of gases:

- Increase in density of gas and hence increased work of breathing.

### Nitrogen effect at high nitrogen pressure:

- Has 2 principle effects:
- Nitrogen narcosis (anesthetic effect).
- Decompression sickness.

# Nitrogen Narcosis

Nitrogen like most other anesthetic gases, dissolves freely in the fats of the body, Including the membranes and other lipid structures of the neurons.

This leads to alteration of the electrical conductance of the membranes, and therefore reduces their excitability and subsequent narcosis develops.

**At 120 feet:** The diver exhibits cheerful behavior and loses his cares.

**At 150-200 feet:** There is a feeling of euphoria and drowsiness and impaired performance.

**At higher pressure:** Loss of coordination and finally coma might develop.

When neurons become less excitable = anesthesia effect.
Nitrogen is a very soluble gas so when it enters through the capillaries it becomes a liquid form in the body with toxic effect
Here nitrogen is shown to have similar effects to alcohol consumption.
About 4/5 of the air is nitrogen. At sea-level pressure, the nitrogen has **no significant effect on bodily functions** but at high pressures it can cause varying degrees of narcosis.
Nitrogen narcosis has characteristics similar to those of **alcohol intoxication.**

# Caisson's disease

It is a syndrome caused by a decrease in the ambient pressure which occur in animals and men when the tissues of the body contain an excess of physically gas.

Upon Ascent

Inert gas comes out of physical solution forming a gaseous phase (bubbles), leading to symptoms and signs.

**During slow ascent**
(decompression is fast) $N_2$ is slowly removed from the tissues since the partial pressure there is higher than that in the arterial blood and alveolar gas. (To avoid getting Cassion's disease)

**During fast ascent**
(decompression is rapid) bubbles of gaseous nitrogen are released in tissues and blood, causing the symptoms of decompression sickness (The Bends Or Caisson disease)

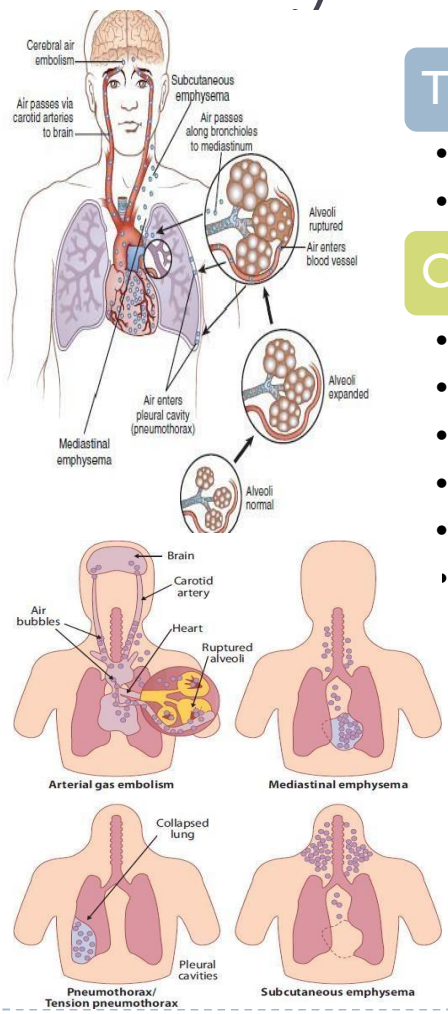
The high partial pressure of nitrogen (encountered when breathing compressed air at depth) forces this poorly soluble gas into solution in body tissue particularly in fat (it has a high $N_2$ solubility).

Upon Descent

# Decompression Sickness (Caisson's Disease) (The Bends)

- Breathes air under high pressure for a long time -> **nitrogen dissolved in the body fluids increases.**
- And because nitrogen is **not metabolized by the body**, it remains dissolved in all the body tissues until the nitrogen pressure in the lungs is **decreased** back to some lower level, however, **this removal often takes hours to occur** and **is the source of multiple problems** collectively called **decompression sickness.**
- If the diver suddenly comes back to the surface of the sea, **nitrogen bubbles can develop** in the body fluids either intracellularly or extracellularly -> **decompression sickness**.
- Due to decompression, gases can escape from the dissolved state and form actual bubbles; in both tissues and blood where they plug many small blood vessels. **The bubbles may not appear for many minutes to hours because sometimes the gases can remain dissolved in the "supersaturated" state for hours before bubbling.**

# Symptoms & Signs

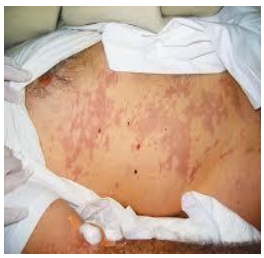



**The mildest form of DS is:**

- **Fatigue or drowsiness.**
- **Local skin itch.**

**Other severe symptoms may occur e.g.:**

- **Bubbles in the tissues**
- **Neurological symptoms.**
- **Thoracic pains.**
- **Bubbles in coronary arteries.**
- **Decompressing sickness shock**
- **Edema.**



# Treatment of Decompression Symptoms

**1- Rapid recompression** in a pressure chamber followed by slower **decompression.**

- This reduces the volume of the bubbles and forces them back into solution.
- In very deep dives, the risk of decompression sickness can be reduced if a helium-$O_2$ mixture is breathed during the dive.

**2- Helium is more desirable than nitrogen in deep dives because it has:**

- 1/4-1/5 the narcotic effect of nitrogen on CNS.
- 1/7 the molecular weight of nitrogen.
- Low density leading to decreased airway resistance of diver.
- High diffusion through tissues (so it easily diffuses from capillary to alveoli and leaves the body).
- Helium is about 1/2 as soluble as nitrogen in body fluids. This reduces the quantity of bubbles that can form in tissues when the diver is decompressed after diving.

# High Altitude (Low Pressure)

Table 43-1. Effects of Acute Exposure to Low Atmospheric Pressures on Alveolar Gas Concentrations and Arterial Oxygen Saturation*

| | | Breathing Air | | | | Breathing Pure Oxygen | | |
|---|---|---|---|---|---|---|---|---|
| Altitude (ft/meters) | Barometric Pressure (mm Hg) | $P_{O_2}$ in Air (mm Hg) | $P_{CO_2}$ in Alveoli (mm Hg) | $P_{O_2}$ in Alveoli (mm Hg) | Arterial Oxygen Saturation (%) | $P_{CO_2}$ in Alveoli (mm Hg) | $P_{O_2}$ in Alveoli (mm Hg) | Arterial Oxygen Saturation (%) |
| 0 | 760 | 159 | 40 (40) | 104 (104) | 97 (97) | 40 | 673 | 100 |
| 10,000/3048 | 523 | 110 | 36 (23) | 67 (77) | 90 (92) | 40 | 436 | 100 |
| 20,000/6096 | 349 | 73 | 24 (10) | 40 (53) | 73 (85) | 40 | 262 | 100 |
| 30,000/9144 | 226 | 47 | 24 (7) | 18 (30) | 24 (38) | 40 | 139 | 99 |
| 40,000/12,192 | 141 | 29 | | | | 36 | 58 | 84 |
| 50,000/15,240 | 87 | 18 | | | | 24 | 16 | 15 |

*Numbers in parentheses are acclimatized values.

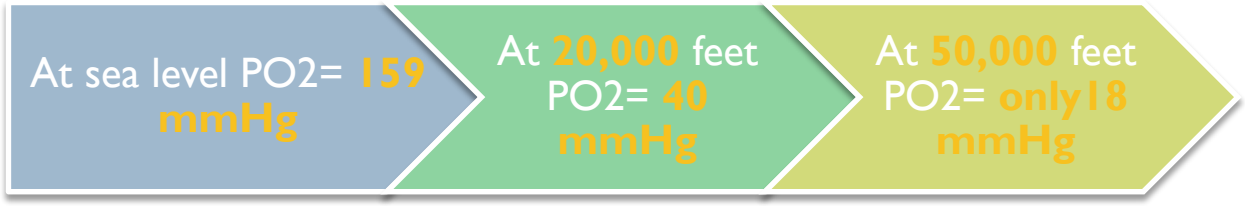
# Effects of Low Oxygen Pressure on the Body (Aviation-Ascend to High Altitude)

- At the sea level the **barometric pressure** is 760 mmHg
- At 10,000 feet is 523 mmHg
- At 50,000 feet 87 mmHg.

This decrease in barometric pressure is the basic cause of all the problems of hypoxia in high altitude physiology.

## Alveolar PO2 at Different Altitudes

- As the **barometric pressure** decreases, the **oxygen partial pressure** decreases **proportionally**, leaving less than 21% of the total barometric pressure.
- At high altitudes, carbon dioxide is continuously excreted from the pulmonary blood into the alveoli. Also, water vaporizes into the inspired air from the respiratory surfaces.
- These two gases dilute the oxygen in the alveoli, thus reducing the oxygen concentration, therefore, **hypoxia develops.**




## Effects of Acute Hypoxia

### At 12000 feet

- Drowsiness
- Lassitude
- Mental and muscle fatigue
- Headache sometimes
- Occasionally nausea
- Euphoria (happiness) sometimes

### Above 18000 feet

- All the effects at 12000 and
1. Twitching
2. Convulsions

### Above 23000 feet

- Coma
- Shortly followed by death

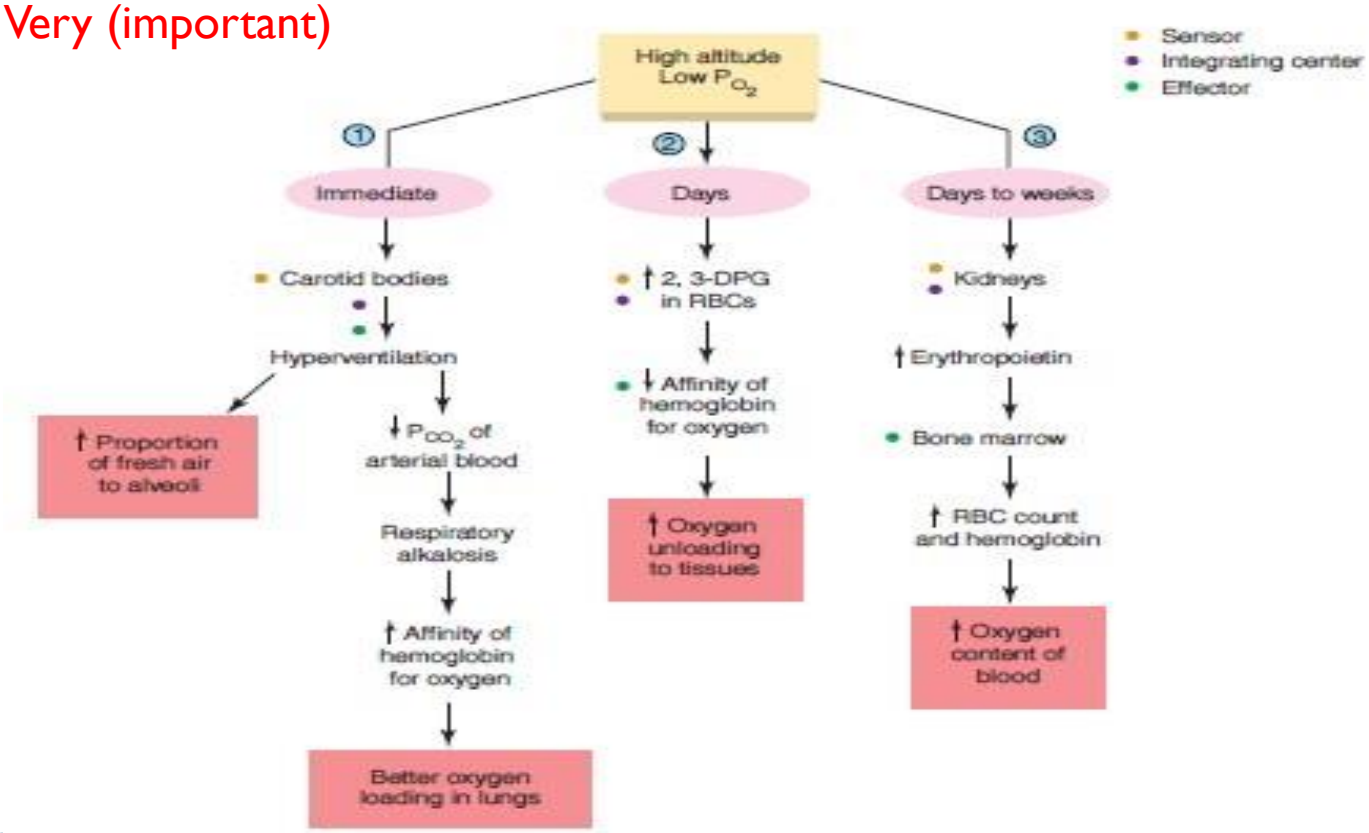
# Acclimatization to Low PO2 (important)

- A person remaining at high altitudes for days, weeks or years becomes more and more acclimatized to low PO2. Acclimatization is important to prevent or have fewer deleterious effects on the body so it becomes possible for the person to work harder without hypoxic effects or to ascend to a higher altitude.

- **Principle means of acclimatization:**

1. • Increase in pulmonary ventilation.
2. • Increased red blood cells.
3. • Increased vascularity of the tissues.
4. • Increased ability of the cells to utilize oxygen despite the low PO2.
5. • Increased diffusing capacity of the lungs.

Very (important)



# YOU ARE DONE!

اللهم إنّي استودعتك ماحفظت وماقرأت وماتعلمت فردّه لي وقت حاجتي إليه، أنك على كل شيء قدير.

تفائل بالله خيراً، فـ «كل متوقَّعٍ آت»

# Good luck our DOCTORS!

Physiology Team436

PHYSIOLOGY
TEAM 436