GAS EXCHANGE

Physical Principles of Gas Exchange

- **Diffusion:**
- Continuous random motion of gas molecules.
- **Partial Pressure:**
- The individual pressure of each gas in a gas mixture.
- **Henry's Law:**
- The amount of dissolved gas is directly proportional to the partial pressure of the gas.
- **Graham's Law:**
- When gases are dissolved in liquids, the relative rate of diffusion of a given gas is inversely proportional to the square root of its molecular mass (Density).
- CO_2 Diffuses 19 times faster than O_2.

$$R = \frac{1}{\sqrt{M}}$$

$$\frac{R1}{R2} = \sqrt{\frac{M2}{M1}}$$

- **Fick's Law:**

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

P Pressure	
S Solubility	
d Membrane Thickness	T Temperature
MW Molecular Weight	A Surface Area

Dr Abdalla Elsamahy

- In the lungs, the pressure gradients favor the diffusion of oxygen toward the blood and the diffusion of carbon dioxide toward the alveolar air.

FACTORS AFFECTING GAS DIFFUSION

PRESSURE	SURFACE AREA	THICKNESS
V/Q RATIO	DIFFUSION CAPACITY	

1. Pressure Gradient of Gases:

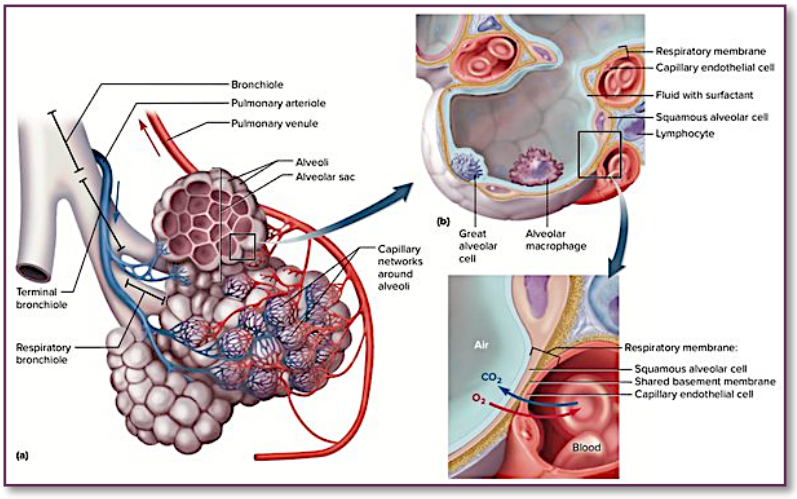
- Normal O2 in alveoli = 100 mmHg and in venous blood = 40 mmHg.
  So diffusion of O2 from alveoli to venous blood by 60 mmHg.
- Normal Co2 in alveoli = 40 mmHg and in venous blood = 46mmHg.
  So diffusion of Co2 from venous blood to alveoli by 6 mmHg.
- In exercise → ↑ alveolar O2 → ↑ diffusion.

2. Surface area of the Pulmonary Membrane:

- Normally = 70 m^2.
- It increases in exercise by increasing the alveolar size, opening and dilation of capillaries.
- It decreases in old age, emphysema, pneumothorax, collapse of lungs or removal of part of lung.

3. Thickness of Pulmonary Membrane:

- The pulmonary membrane is the membrane through which gas exchange between alveoli and venous blood occur.
- Normally = 0.6 micron.



- Formed of:

1. Surfactant.
2. Fluid layer.
3. Alveolar epithelium.
4. Basement membrane of alveolus.
5. Interstitial fluid.
6. Basement membrane of capillary.
7. Capillary endothelium.

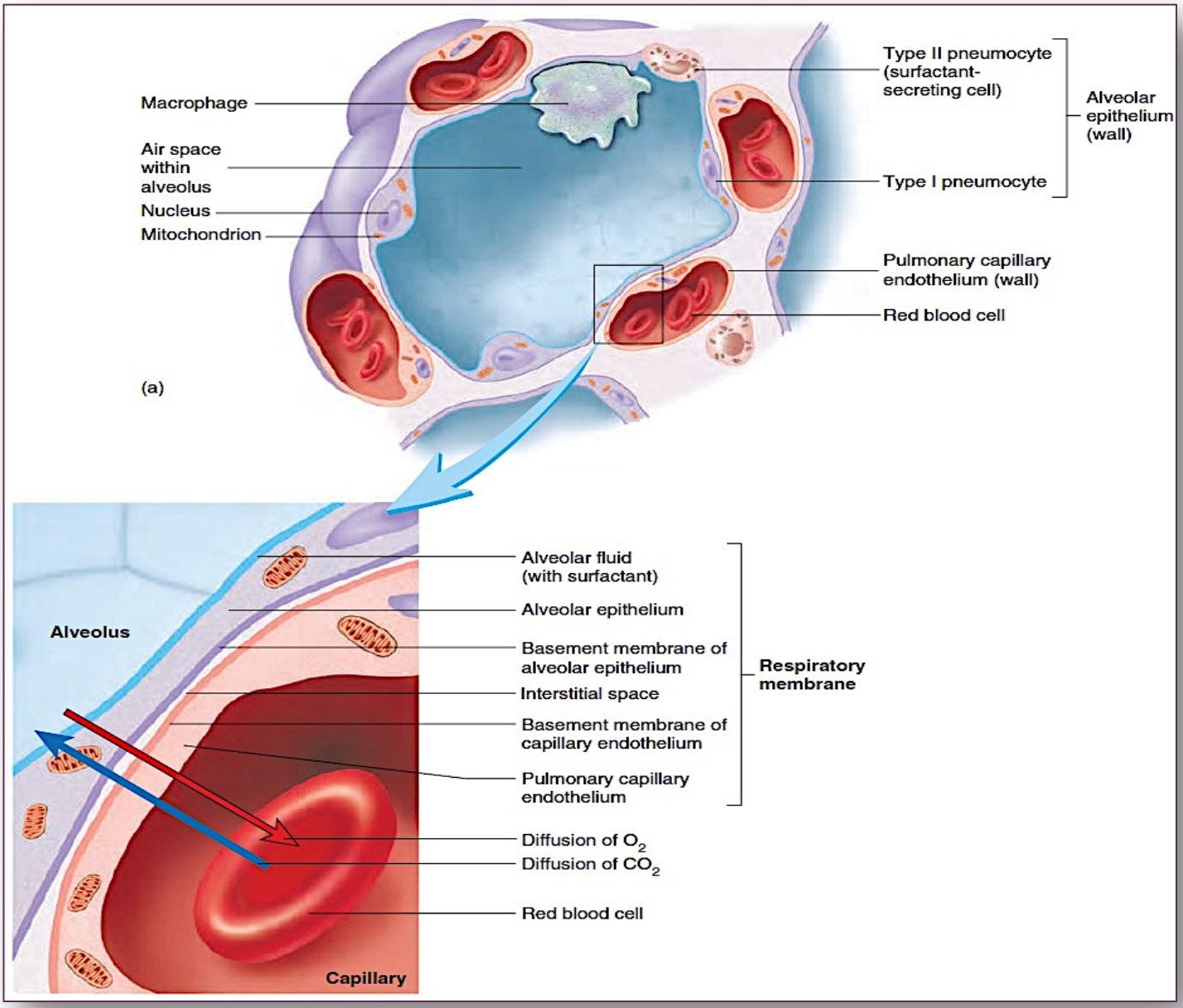
- Increase the thickness causes decrease diffusion as in pulmonary oedema and lung fibrosis.

4. Ventilation - perfusion ratio (V/Q):

- Alveolar ventilation (V) = 4.2 L
- Pulmonary blood flow (Q) = 5 L
- V/Q = 0.84 (optimal ratio of air supply and blood supply).
- It is measured by radioactive xenon injection in breathing air to measure ventilation and in pulmonary artery to measure perfusion.

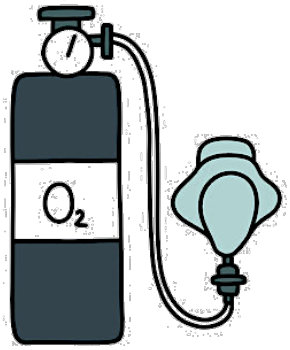
5. Diffusion capacity of the lung:

- The volume of gas transferred from alveoli to capillary blood through the respiratory membrane each minute per unit gas pressure differences.
- O2 lung diffusion capacity is 25 ml/min/mmHg.
- CO2 lung diffusion capacity is 500 ml/min/mmHg.
- Any disease will affect the respiratory membrane will affect the diffusion capacity of the gases.



OXYGEN TRANSPORT

FORMS OF O_2 TRANSPORT



1. O_2 in physical solution:

- **Nature:**
- **O_2 molecules dissolved in blood.**
- **Value:**
- **In arterial blood (with tension of 100 mmHg) = 0.3 ml/100 mL.**

2. O_2 in chemical form:

- **Nature:**
- **O_2 combines with the iron of hemoglobin while still in the ferrous state (It is called oxygenation).**
- **Value:**
- **19 ml O_2/100 ml arterial blood.**
- **14 ml O_2/100 ml venous blood.**
- **Factors affecting chemical form of Oxygen transport:**

	O2 Capacity of HB	O2 Content of HB	O2 Percentage saturation of HB
Definition	**Maximal** volume of O_2 present in chemical form /100 ml blood **If Hb is fully saturated** with O_2.	**Actual volume** of O_2 present in chemical form **/100 blood**.	(O_2 Content / O2 Capacity) X 100
Value	• Each gram Hb can carry 1.33 ml O2 **(with full saturation)**. If Hb content = 15 gm% • So, O2 capacity = 15 x 1.33 = **20 ml O2/100 ml blood.**	• Arterial blood contains: **19 ml O_2/100 ml.** • Venous blood contain: **14 ml O_2/100 ml.**	• Arterial blood: = 19 / 20 = **95 %** • Venous blood: = 14 / 20 = **70 %.**
Depend on	Amount of Hb only .	Amount of Hb & PO_2.	PO_2 only.

• Coefficient of Oxygen utilization (oxygen extraction ratio):

- It is the percentage of O2 in arterial blood that is taken by tissue.

$$= \frac{O_2 \text{ utilized by tissue}}{\text{Arterial } O_2 \text{ content}} \times 100 \qquad = \frac{\text{Arterial} - \text{Venous}}{\text{Arterial } O_2 \text{ content}} \times 100$$

At rest $= \frac{19-14}{19} \times 100 =$ **25%.** **In exercise** $= \frac{19-5}{19} \times 100 =$ **75%.**

O_2 Dissociation Curve of Hemoglobin

• Definition:

- **It shows relationship between O_2 tension and percentage saturation of Hb at different O_2 tensions.**

• Method:

- **3 ml of blood in Tonometer are allowed to form thin layer and then exposed to different O_2 tensions and percentage (%) saturation is calculated and plotted against the O_2 tensions.**

• Physiological significance of the curve:

1. At the lung and arterial blood:

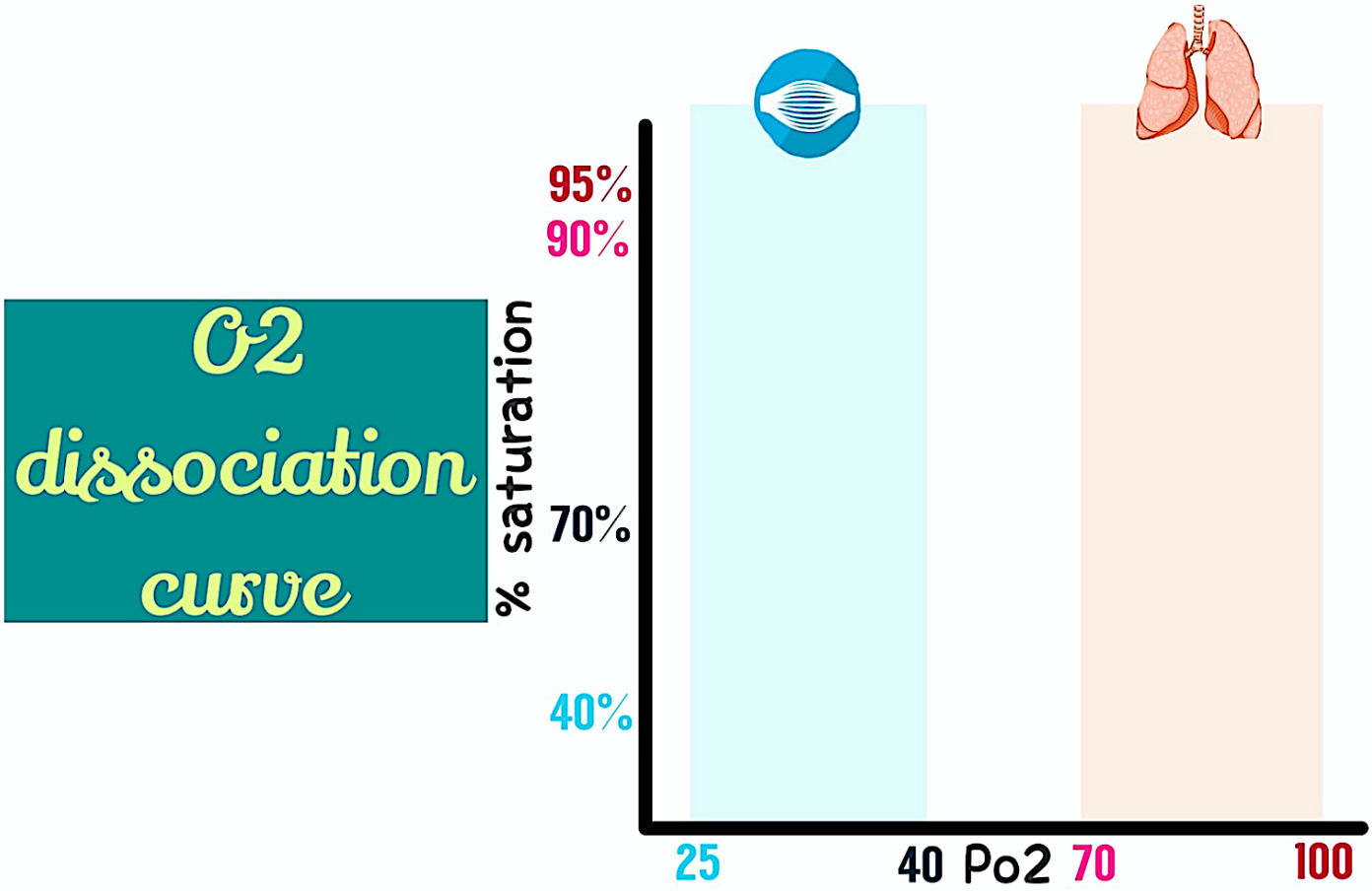
- **O_2 tension of 100 mmHg (normal alveolar O_2 tension) → 95% saturation.**
- **O_2 tension of 70 mmHg (alveolar tension at high altitude or diseased lung) → 90% saturation.**
- **The change of O_2 tension from 100 to 70 mmHg causes mild change of % saturation, the curve is nearly horizontal (high affinity of Hb to O_2 at high O_2 tension).**

2. At the tissue and venous blood:

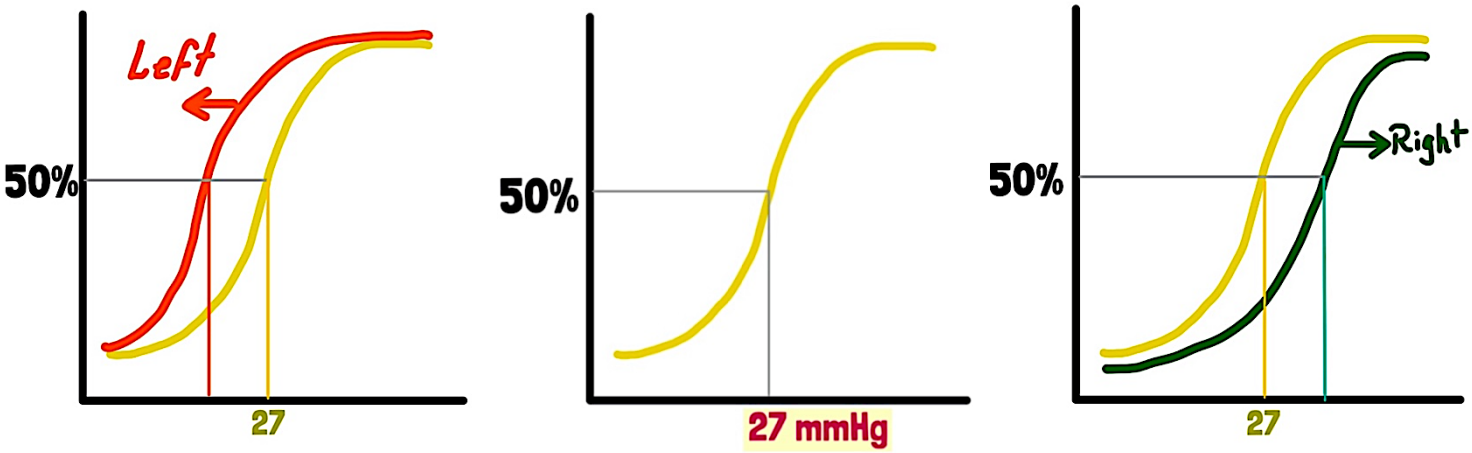
- **O_2 tension of 40 mmHg (resting muscle and venous blood tension) → 70% saturation.**
- **O_2 tension of 25 mmHg (during exercise) → 40% saturation.**
- **Mild change in O_2 tension from 40 to 25 mmHg → large decrease in saturation from 70 to 40% to give oxygen to tissue, so the curve is nearly vertical (steep) at tissue and venous side due to low affinity of Hb to O_2 at lower O_2 tension.**

3. The curve is Sigmoid or (S) shaped:

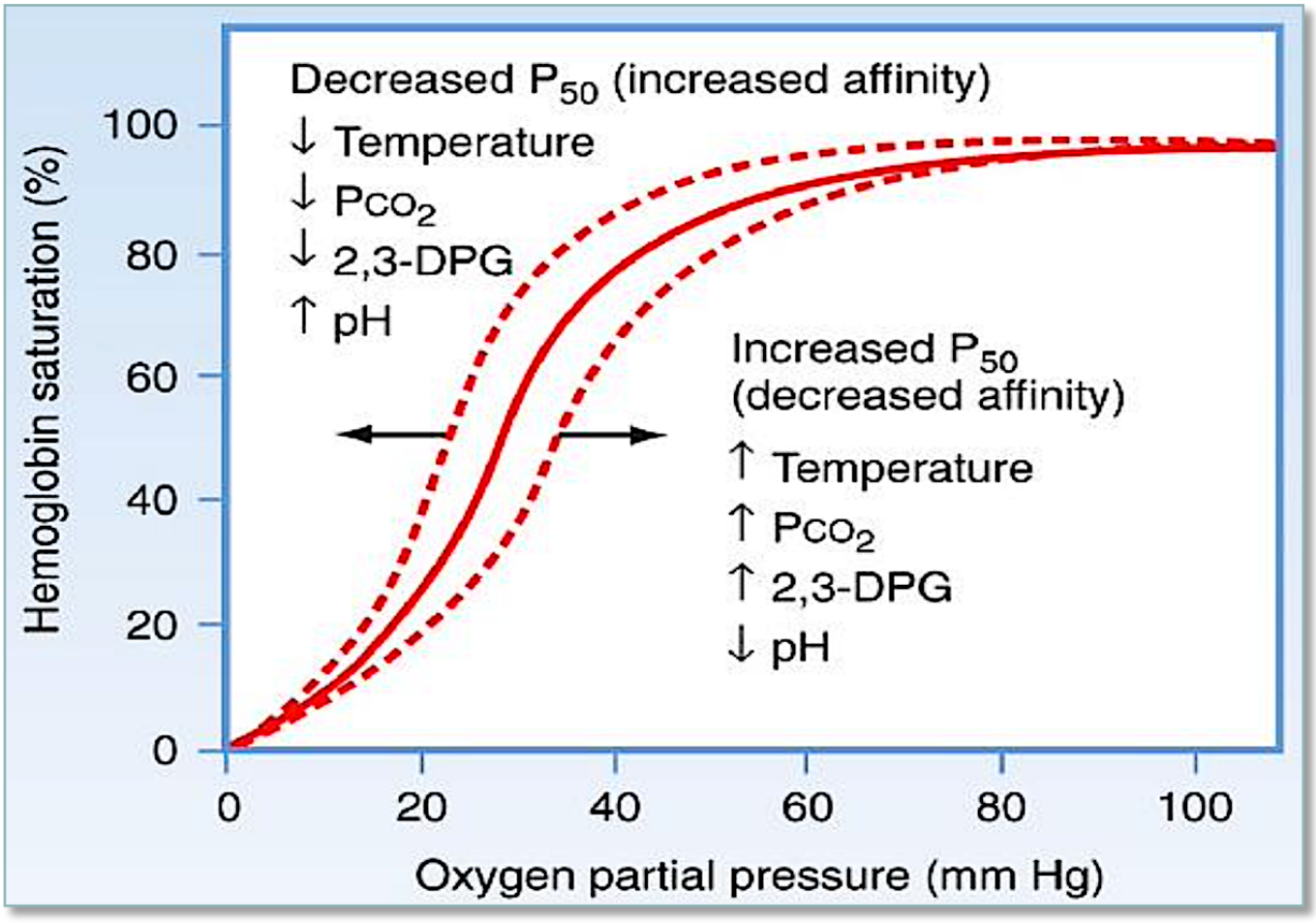
- **Because Hb contains 4 ferrous atoms and each one is saturated at certain O_2 tension and saturation of each one facilitates of the following one.**
- **Bohr Effect:**
- **When PH of the blood decreases (PCO_2 increases) → O_2 affinity to hemoglobin decreases.**



4. Shift of the O_2 dissociation curve:



	Shift to left	Shift to right
Meaning	At any PO_2, the Hb is **more** saturated with O_2 ⇨ **give less** O_2 to tissue. (⇧ **affinity** or loading).	At any PO_2, the Hb is **less** saturated with O_2 ⇨ **give more** O_2 to tissue. (⇩ **affinity** or unloading).
P 50	**Decreased.**	**Increased.**
	Shift of the curve has little effect on the horizontal part. But, greater effect on the **steep (vertical)** part. So, it is best measured by the **P50**.	
Causes	1) ⇧⇧ O_2. 2) ⇩⇩ CO_2 (Bohr effect). 3) ⇩⇩ H^+ (alkalosis = ⇧ pH). 4) ⇩⇩ Temperature. 5) ⇩⇩ 2,3 DPG. 6) Polycythemia. 7) Fetal Hb (Hb F). 8) Carboxy Hb (CO poisoning). 9) Met Hb (Strong oxidation).	1) ⇩⇩ O_2. 2) ⇧⇧ CO_2 (Bohr effect). 3) ⇧⇧ H^+ (acidosis = ⇩ pH). 4) ⇧⇧ Temperature (fever). 5) ⇧⇧ 2,3 DPG. 6) Anemia. 7) Pregnancy. 8) Exercise.
Significance	⇧ O_2 loading at the lung.	⇧ O_2 supply to active muscle during exercise.



CO_2 TRANSPORT

CO_2 content

- PCo_2 in arterial blood = 40 mmHg, Co_2 content of arterial blood = 48 ml/100 ml blood.
- PCo_2 in venous blood = 46 mmHg, Co_2 content of venous blood = 52 ml/100 ml blood.
- In normal resting conditions body produces 200 ml CO_2/minute.

CO_2 is carried in two forms in the arterial blood

1. In physical solution (3 ml):

- It is the free form of Co_2 present mainly in plasma but some diffuse to RBCs.
- Some combine with $H_2O \leftrightarrow H_2Co_3 \leftrightarrow H^+ + HCO_3^-$
- Its amount depends on Co_2 tension in blood.

2. In chemical combination (45 ml):

A. As bicarbonate (42 ml).

B. As carbamine compound (3 ml).



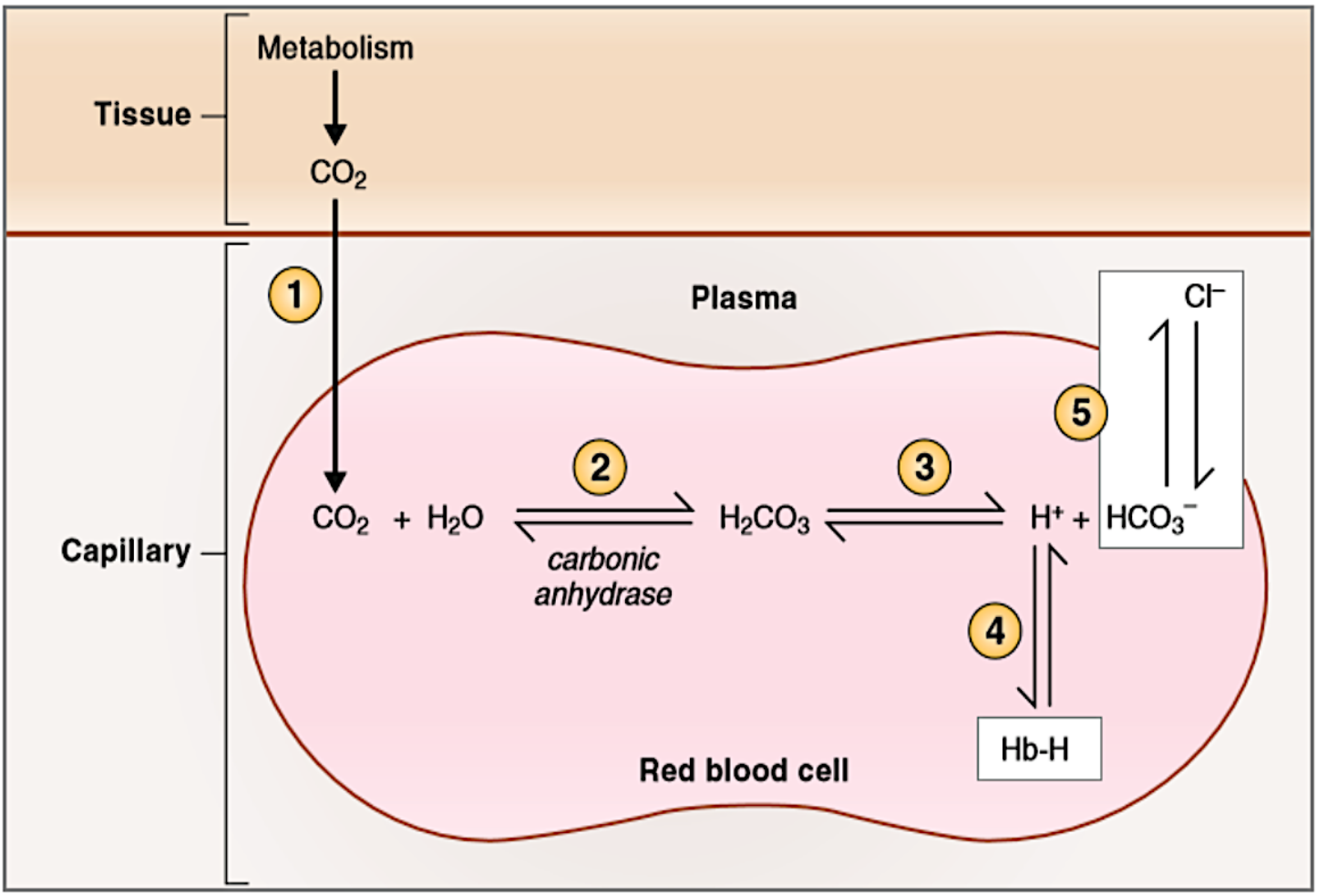
the tidal CO_2 (4 ml/100 ml blood)

- It is the Co_2 which given by tissue to the blood.
- It is about 4 ml which is buffered into 3 forms:

1. In physical solution 0.4 ml (10%).
2. As carbamine compound 1 ml (25%)
3. Buffered with Hb → HCO_3^- 2.6 ml (65%) via Cl^- shift phenomenon.

Chloride shift (Hamburger's) phenomenon

- It is responsible for carrying most of tidal Co_2 in the form of bicarbonate in the venous side of circulation.



A. At tissue side:

- **CO_2 is changed to $HCO3^-$.**
- Tidal CO_2 diffuses from tissue to RBCs and combine with H_2O.
- $CO_2 + H_2O \Leftrightarrow H_2CO3 \Leftrightarrow H^+ + HCO3^-$.
- **H^+** is buffered by reduced Hb (after Hb giving its O_2 to tissue).
- **$HCO3^-$** combines with K^+ released from reduced Hb.
- ⇧ Intracellular $HCO3^-$ ⇨ diffusion of $HCO3^-$ according its concentration gradient to plasma to be combined with Na^+ (Na HCO3).
- According to electrostatic gradient **Cl^-** passes from plasma to RBCs until:

$$\frac{Cl^- \text{ in plasma}}{Cl^- \text{ in RBCs}} = \frac{HCO_3^- \text{ in RBCs}}{HCO_3^- \text{ in plasma}}$$ (Donnan equilibrium).

B. The results of Cl^- shift:

- Changes in **venous** blood than arterial blood:

	$HCO3^-$	Cl^-	Osmotic pressure	pH
In RBCs	⇧	⇧	⇧ Cl^- ⇨ osmosis of H_2O inside RBCs ⇧ RBCs **size** in **venous** blood leads to: **a)** ⇧ Hematocrit value. **b)** ⇧ Osmotic fragility.	Constant.
In plasma	⇧	⇩	Constant.	Constant.

C. At lung side:

- The reverse occurs. **$HCO3^-$ is changed to CO_2.**
- $HCO3^-$ re-diffuse to RBCs and Cl^- to plasma.
- **$HCO3^-$** changed in RBCs into **CO_2** which is expired through the lung.

Other sites of carbonic anhydrase enzyme:

1. Stomach.
2. Pancreas.
3. Brain.
4. Eye (aqueous humor).
5. Kidney.

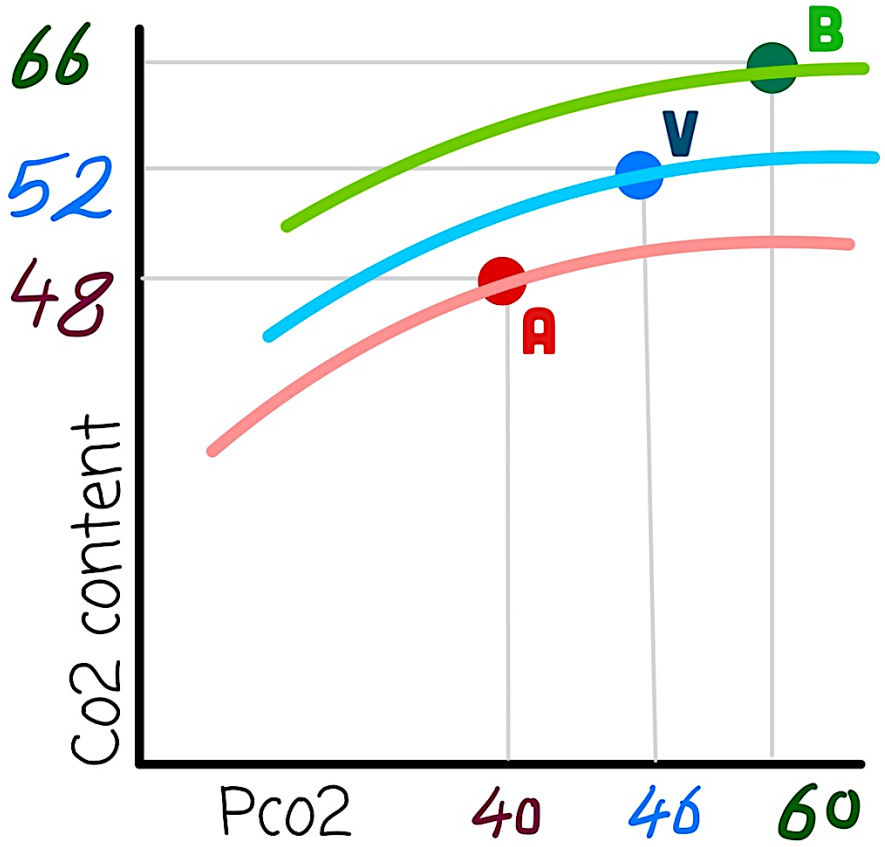


CO2 dissociation curves

- Three curves are made.

	Curve for fully Oxygenated blood	Curve for 30% Reduced blood	Curve for fully Reduced blood
Site	**A**rterial blood.	**V**enous blood.	Reduced blood.
CO_2 content	**48** ml/100 ml blood.	**52** ml/100 ml blood.	**66** ml/100 ml blood.
PCO_2	**40** mmHg.	**46** mmHg.	**60** mmHg.
Point	(Point **A**)	(Point **V**)	(Point **B**)

CO2 DISSOCIATION CURVE



ALKALI RESERVE

- **Definition:**

- It is the volume of CO2 present in chemical combination as HCO3- (NaHCO3) in arterial **plasma**.
- **Not in whole blood** as CO2 content differs according to hematocrit value.
- So, **CO2 content in anemic blood is more due to more plasma**.

- **Value:**

- **55 – 70 ml** CO2/100 ml of arterial plasma = 27 milli equivalent/L.

Significance:

1. Carriage of CO2 as bicarbonate.
2. Buffer for acids as the body has tendency to become acidic.
3. <u>It is an index of acid base balance:</u>

- If alkali reserve > 70 ml → indicates that alkalemia may occur.
- If alkali reserve < 55 ml → indicates that acidemia may occur.

REGULATION OF RESPIRATION

- Normal breathing (eupnea) is usually an unconscious act driven by the autonomic nervous system.
- The cyclic pattern is established by a respiratory control center within the brain stem.
- Sensors (Receptors) located within the central nervous system (CNS) and the periphery to monitor blood and tissue PCO2, PO2, and pH levels and feed this information back to the control center for processing.
- The center also receives information from mechanoreceptors located in the lungs and chest wall then adjusts ventilation as needed.

NERVOUS CONTROL OF RESPIRATION

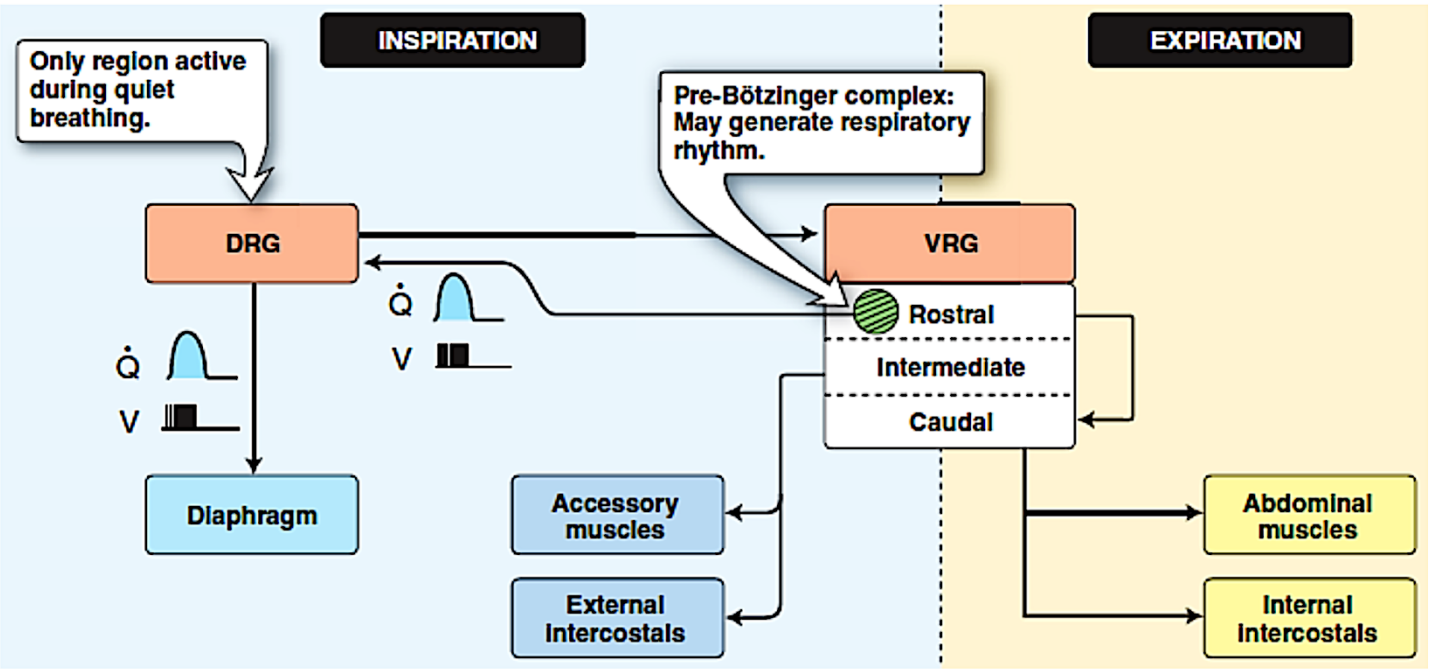
A. MEDULLARY CENTERS

1.Dorsal Respiratory Group (DRG):

- Function:
- DRG neurons are active primarily during tidal inspiration the active phase of quiet breathing.
- Input:
- The DRG receives sensory inputs from the thorax and abdomen and processes incoming signals using interneuron networks.
- Output:
- DRG neurons communicate with premotor neurons controlling the diaphragm.
- They also project to a control center area involved in forced inspiration and expiration (the VRG).

2.Ventral Respiratory Group (VRG):

- The VRG can be subdivided into rostral, intermediate, and caudal regions.
- Function:
- The VRG functions primarily to coordinate accessory muscles of inspiration and expiration.
- Central Pattern Generator (CPG):
- The VRG rostral region contains a small area known as the Pre-Bötzinger Complex composed of cells that exhibit pacemaker-like activity.
- Ablation of the complex abolishes rhythmic breathing, suggesting that it constitutes a respiratory central pattern generator (CPG).



B. PONTINE CENTERS

- The pons contains two areas that influence medullary output.

1.The apneustic center (lower pons):

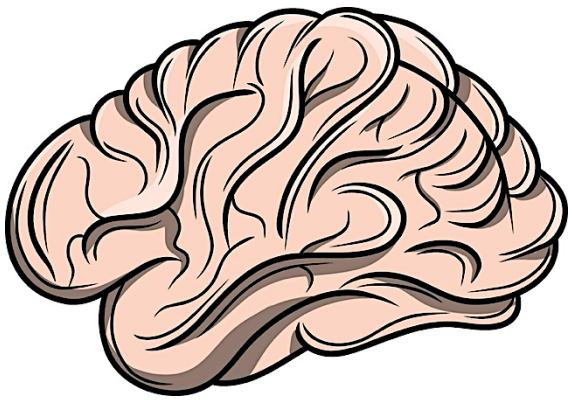
- It normally permits lung expansion.

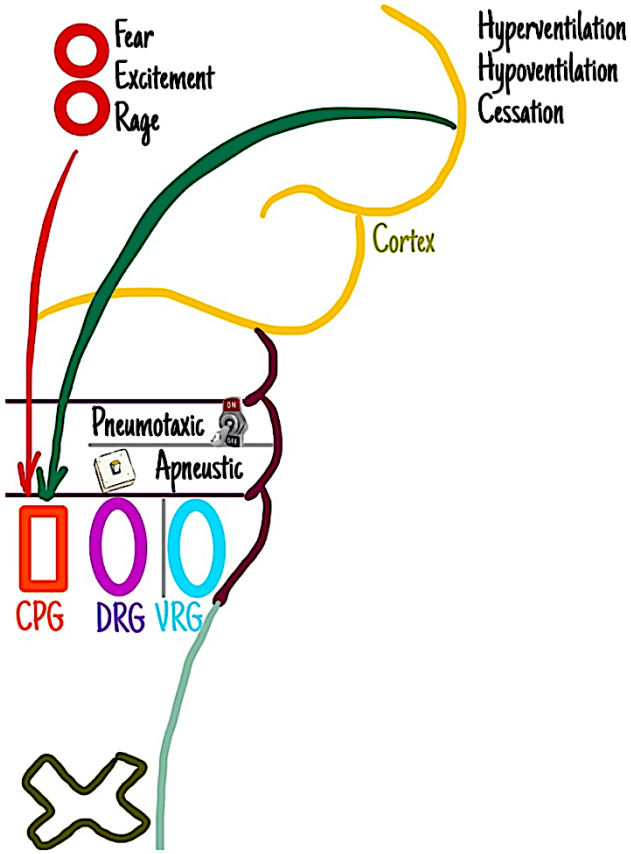
2.The pneumotaxic center (upper pons):

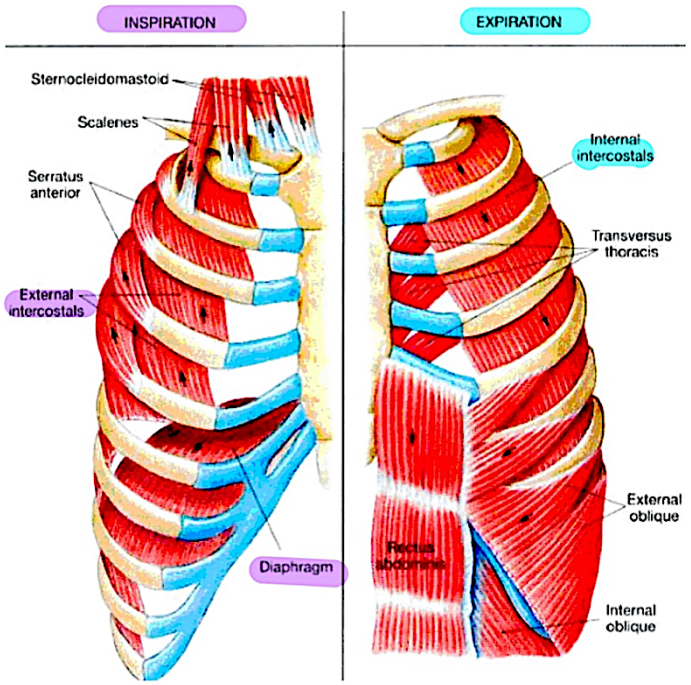
- Shortens inspiration and increases breathing rate.

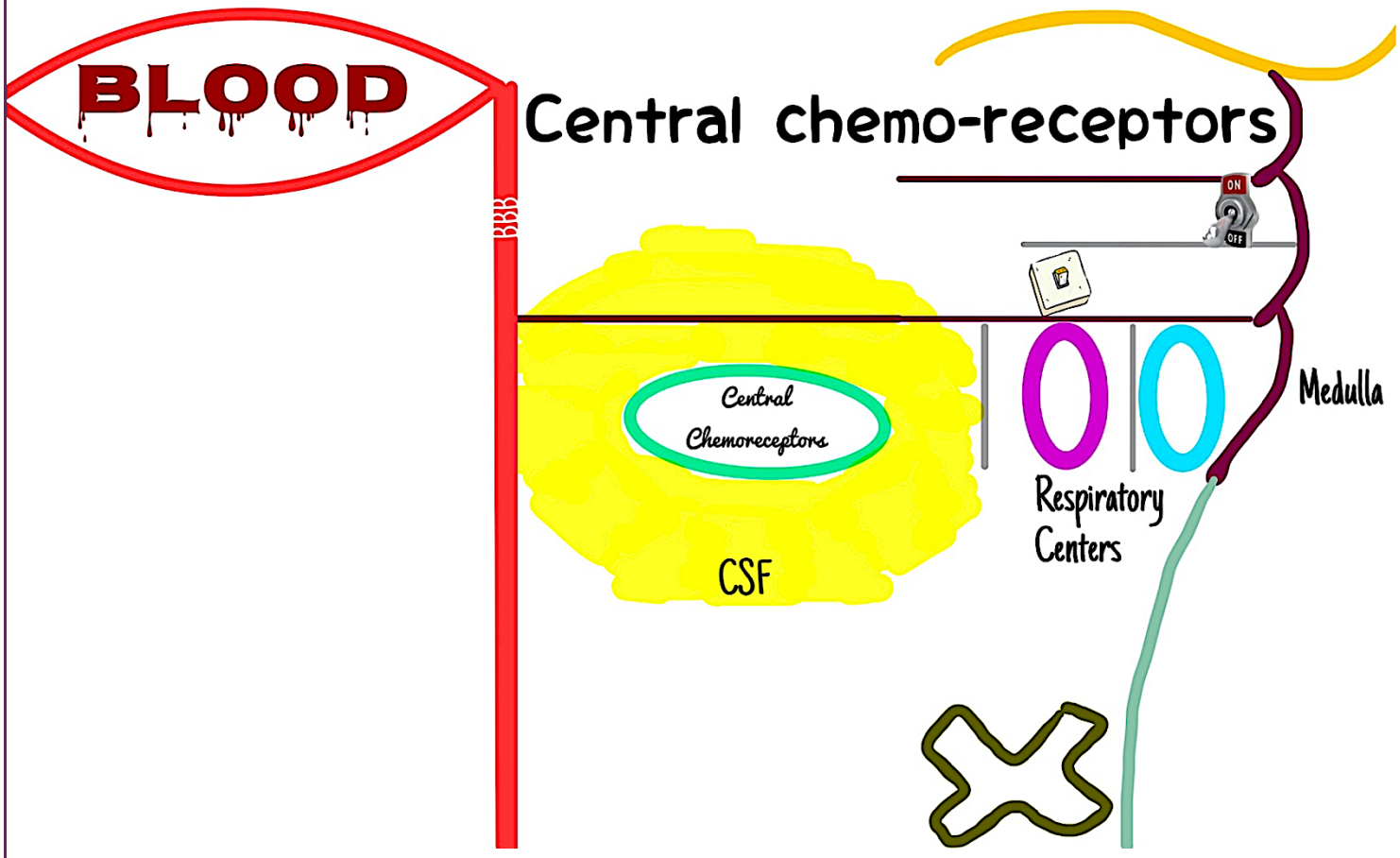
C. CEREBRAL CORTEX

- Emotions such as fear, excitement and rage can alter respiratory rate, reflecting the ability of the hypothalamus and limbic system to modulate the CPG.
- A person can also consciously override the CPG and hyperventilate, hypoventilate, or cease breathing.




Fear
Excitement
Rage
Hyperventilation
Hypoventilation
Cessation
Cortex
Pneumotaxic
Apneustic
CPG
DRG
VRG

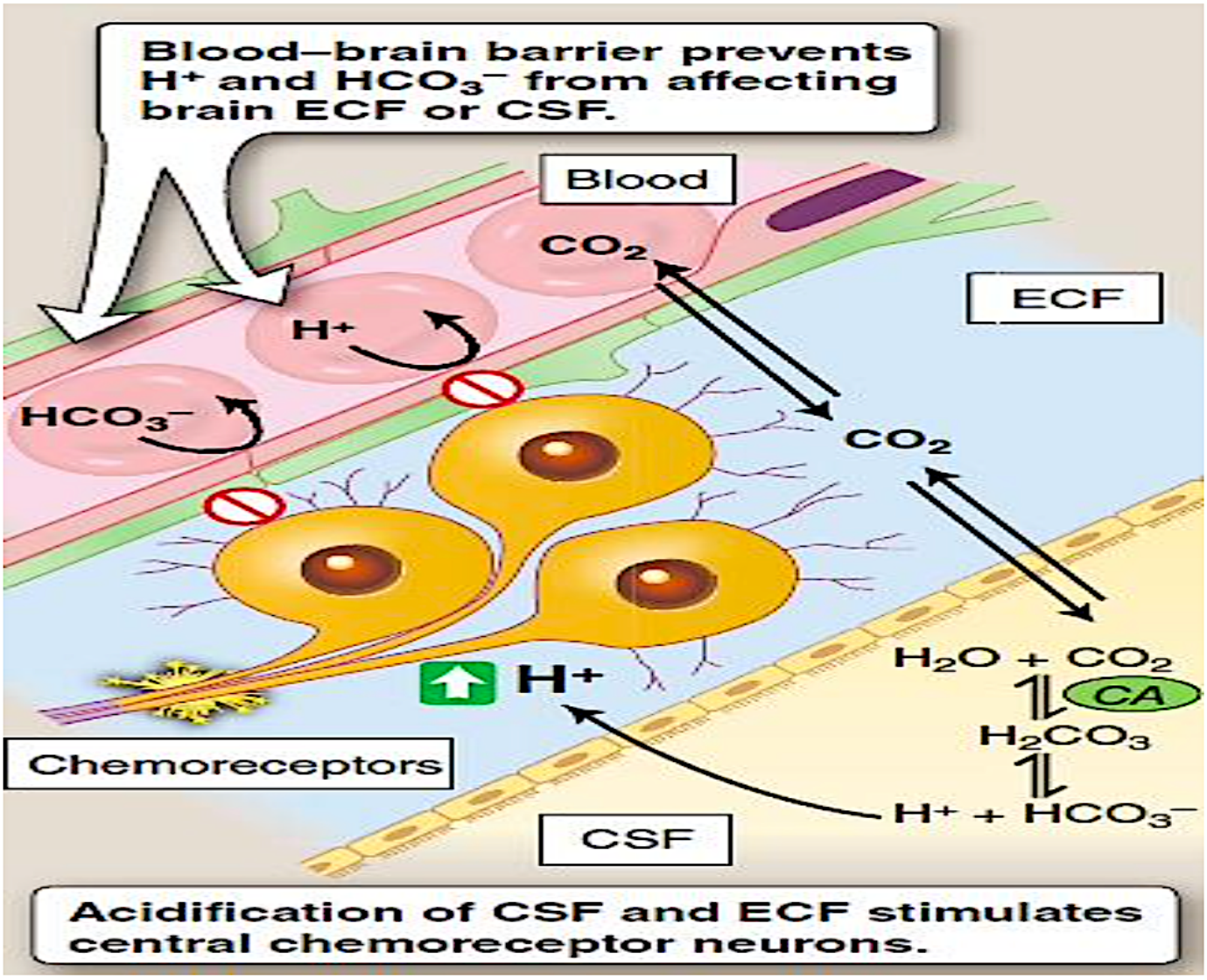

INSPIRATION
EXPIRATION
Sternocleidomastoid
Scalenes
Serratus anterior
External intercostals
Diaphragm
Internal intercostals
Transversus thoracis
External oblique
Rectus abdominis
Internal oblique


BLOOD
BBB
Central chemo-receptors
Central Chemoreceptors
CSF
Respiratory Centers
Medulla

CHEMICAL CONTROL OF RESPIRATION

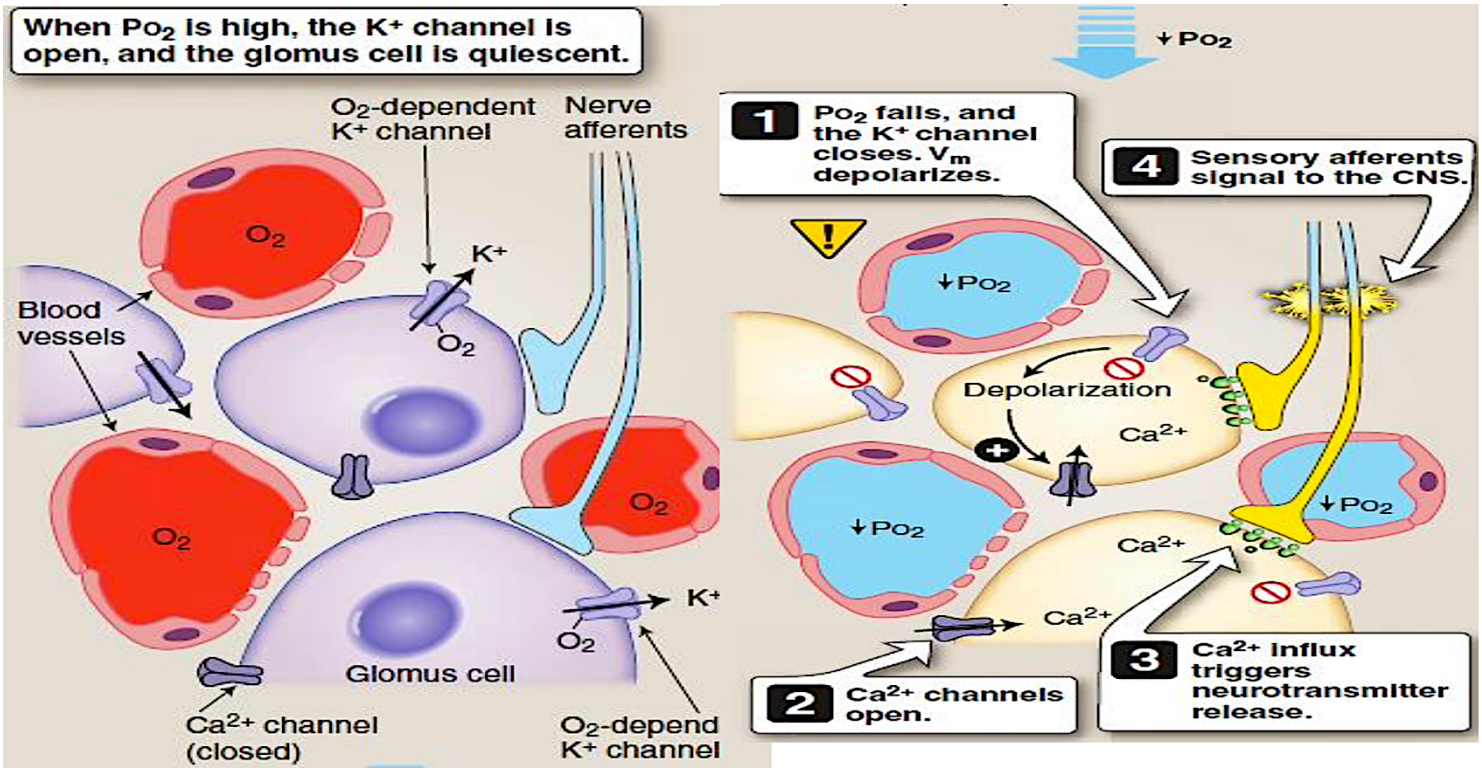
A. CENTRAL CHEMORECEPTORS

- Ventilatory control under resting conditions is dominated by the central chemoreceptors, which are responsive primarily to changes in PCO2.
- The chemoreceptors are CNS neurons located behind the blood–brain barrier (BBB) along the medullary surface.



B. PERIPHERAL CHEMORECEPTORS

- Located in carotid bodies and aortic bodies.
- Peripheral chemoreceptors monitor PO2, PCO2, and arterial PH.



C. VENTILATORY RESPONSES

- The respiratory control center processes information about PO2, PCO2, and arterial pH.

1. Changing carbon dioxide levels:

- The nature of control center response to a rise in PCO2 depends on whether the change is acute or chronic.

A. Acute rise in PCO_2:

- When PCO2 rises the respiratory center compensates by increasing alveolar ventilation.
- CO2 is an extremely potent ventilatory stimulus.
- The peripheral receptors are fast acting and trigger an immediate response.
- The central receptors take several minutes to activate fully, but their effects ultimately dominate the ventilatory response.
- Ventilation increases linearly with any rise in PCO2 above resting values, whereas ventilation decreases when PCO2 falls below 40 mm Hg.

B.Chronic PCO_2 rises:

- Patients with chronic pulmonary disease may not be able to ventilate at levels required to hold PCO_2 at 40 mm Hg.
- Ventilation increases initially when PCO2 begins to rise because the pH of the CSF falls, but the choroid plexus responds by secreting HCO_3^- into the CSF, which, over a period of 8 - 24 hours, that terminates the effect of the higher PCO2 on plasma pH.
- Thus, although PCO2 may remain high, the medullary chemoreceptors no longer register the change, and the respiratory control system adapts to a new, higher PCO2.

2. Changing oxygen levels:

- The peripheral chemoreceptors promote a rapid compensatory increase in ventilation if PO2 falls to dangerously low levels (below 60 mmHg).
- Control center output in response to changes in PO2 is influenced both by blood pH and PCO2.

A.PH:

- When PO2 falls, deoxyhemoglobin concentration rises, which binds more H^+ (the Bohr effect) and plasma H^+ level fall as a result.
- Rise in pH decreases chemoreceptor sensitivity to a fall in PO2.

B.CO2:

- When ventilation increases, PO2 increases, and PCO2 falls.
- Because the respiratory control centers are designed to optimize PCO2, the response to mild hypoxia is overridden in favor of a stable CO2 concentration.

PULMONARY RECEPTOR ROLE

A. IRRITANT RECEPTORS

- The epithelium of the larger conducting airways contains sensory nerve endings (rapidly adapting receptors) that respond to irritants and noxious stimuli, such as ammonia, smoke, pollen, dust, and cold air.
- The receptors trigger bronchoconstriction, mucus secretion, and coughing to prevent foreign materials from reaching the respiratory zone.
- This bronchoconstriction results from histamine release during an allergic asthma attack.

B. JUXTA PULMONARY CAPILLARY RECEPTORS

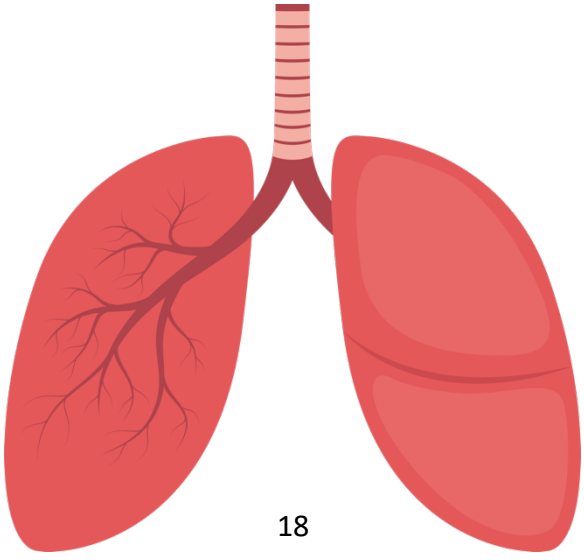
- The alveolar walls contain unmyelinated nerve fibers (C fibers).
- Also known as juxta pulmonary capillary receptors, or J receptors, they are sensitive to lung inflation, injury, pulmonary vascular congestion, and certain chemicals.
- When stimulated, they cause bronchoconstriction, mucus secretion, and shallow breathing.

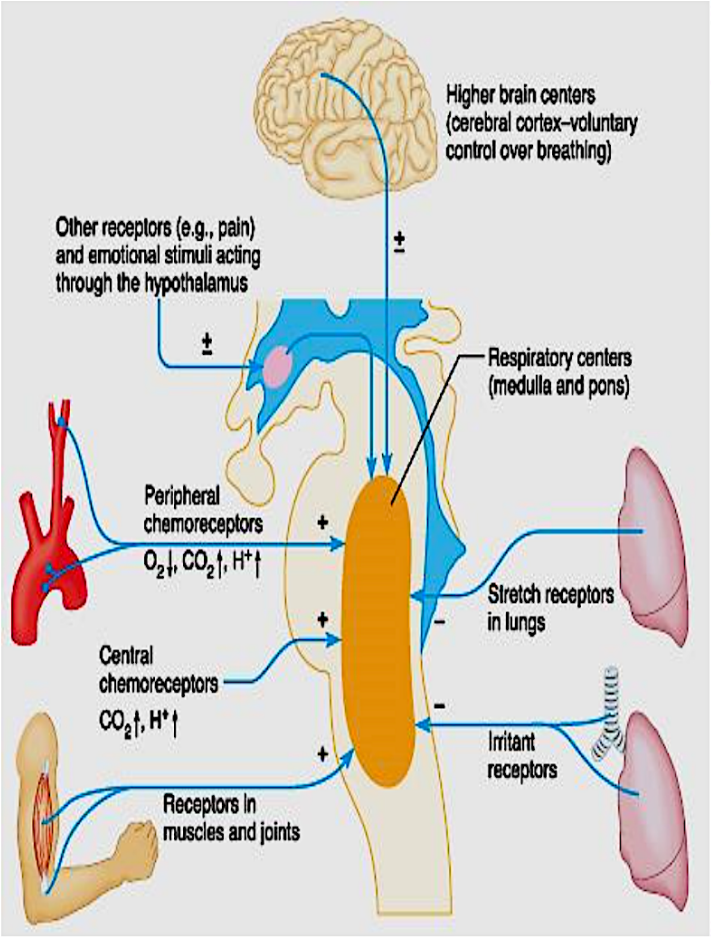
C. STRETCH RECEPTORS

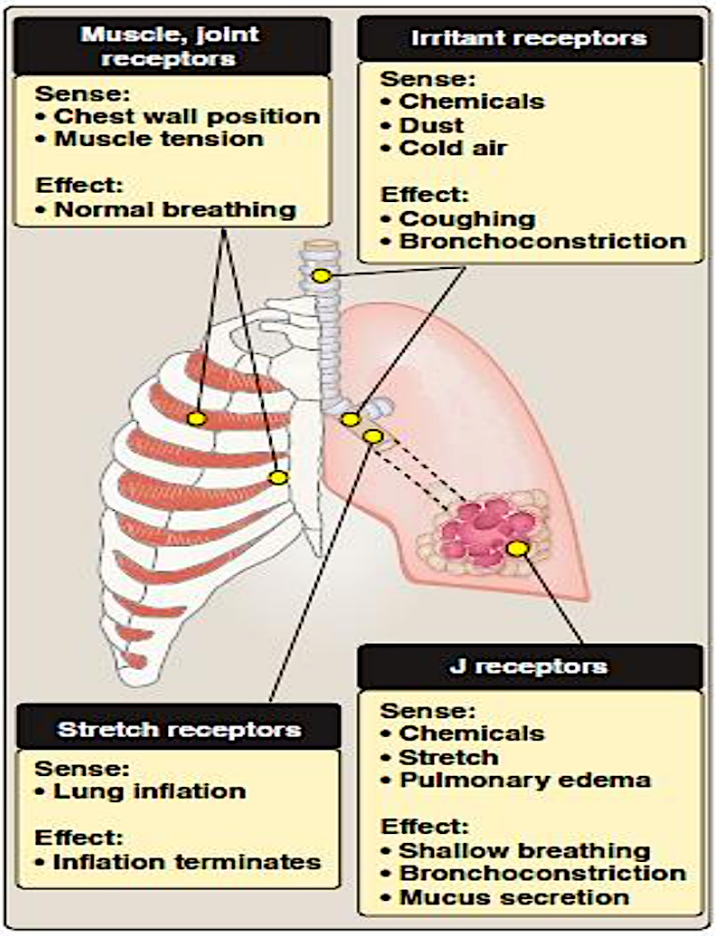
- Embedded within the smooth muscle layers of conducting airways are myelinated sensory fibers.
- They respond to stretch, with output intensity reflecting the extent of inflation.
- If tidal volume is sufficiently high, the stretch receptors can terminate inspiration and prolong the exhalation that follows (Hering-Breuer reflex).

D. JOINT AND MUSCLE RECEPTORS

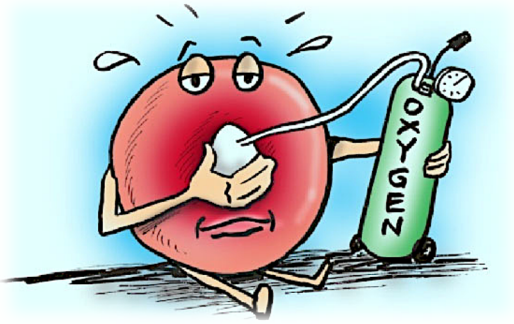
- Stretch and tension receptors located in the chest wall sense wall movement and the amount of effort involved in breathing.
- Output from these receptors allows for increased force of inspiration and expiration when wall movement is impeded.
- Limb joints contain similar receptors that contribute to increased ventilation during exercise.







HYPOXIA



- **Definition:**
- Hypoxia is O2 deficiency at the tissue level.
- Hypoxemia is O2 deficiency in the blood.
- **Types and causes:**

1. Hypoxic Hypoxia:

- Decrease in the available oxygen due to abnormal ventilation.

2. Anemic Hypoxia:

- These is deficiency of functioning hemoglobin capable of carrying O2.

3. Stagnant Hypoxia:

- In this type O2 and Hb content are normal but there is decreased blood flow through tissues leading to cyanosis in the area of stagnation.

4. Histotoxic Hypoxia:

- Due to inhibition of tissue oxidative processes.

- **Characters:**

	Hypoxic	Anemic	Stagnant	Histotoxic
Definition	Deficiency in **oxygenation** of blood.	Deficiency of **functioning Hb** capable of carrying O_2.	Decreased **blood flow** to tissues (**ischemia**).	Deficiency in **O_2 utilization** by tissues.
Arterial PO_2	⇩	Normal	Normal	Normal
Arterial O_2 content	⇩	⇩	Normal	Normal
Venous PO_2	⇩	⇩	⇩	⇧
Venous O_2 content	⇩	⇩	⇩	⇧
Polycythemia	Present.	No.	Present.	No.
Cyanosis	Present.	No.	Present.	No.
Benefit of O_2 therapy	Great.	Limited.	Limited.	No.

CYANOSIS

- **Definition:**
  - It is **bluish** discoloration of the skin & mucous membrane due to increase amount of **reduced hemoglobin more than 5 gm% reduced Hb** in the superficial Capillary blood. (Normally capillary blood contain 2 gm% reduced Hb).
- **Causes:**

A. Hypoxia:

- Hypoxic hypoxia & Stagnant hypoxia:
  As there is polycythemia with large amount of Hb in the blood.
  So, amount of O2 is not sufficient for oxygenation of all hemoglobin.
- **Cyanosis NOT occur in:**
  Anemic hypoxia: due to decrease total amount of Hb.
  Histotoxic hypoxia: as blood content of oxy Hb is normal.

B. Asphyxia:

Combined hypoxic hypoxia , hypercapnea & respiratory acidosis.

C. Cold cyanosis (physiological):

Due to decreased blood flow due to VC.
But, in severe cold whether there is no cyanosis due to ↓ tissue metabolism.





- **Types and causes:**

	Central	Peripheral
Causes	1) Hypoxic hypoxia. 2) Stagnant hypoxia (generalized). 3) Asphyxia.	1) Cold. 2) Stagnant hypoxia (localized).
Site	**All over the body**	**Peripheral (fingers)**
Hands	Warm.	Cold.
Effect of Warming of hands	Causes more blood flow with more cyanotic blood.	Correct the cyanosis.
Tongue	**Cyanosed**	**Normal**
Polycythemia	Present.	Absent.

DYSPNEA

- **Definition:**
  - Difficult breathing in which the person is conscious of shortness of breath.
- **Causes:**

1. Pulmonary causes:

- Obstructive lung disease (asthma and emphysema).
- Restrictive lung disease (pneumonia and fibrosis)

2. Cardiac causes:

- Mitral stenosis or left side heart failure.

3. Neurogenic:

- Psychic fear or emotions causes hyperventilation.

4. Anemic dyspnea:

- It appears during exertion, not during rest.



5. General causes:

A. Acidosis.
B. O2 lack in high altitude.
C. Thyrotoxicosis → ↑metabolism → ↑CO2 → ↑respiration.

6. Abdominal causes:

- As marked ascites or distension leading to decrease the descend of diaphragm.

Caisson's Disease (Decompression Sickness)

- When a diver ascends to the surface gradually, the decompression is gradual, no harmful effects occur as the excess N_2 is liberated through the lungs.
- If the person decompressed rapidly the deep see, the excess N_2 form bubbles in tissue → joint pain and itching and in blood causes block blood vessels.
- Block cerebral blood vessels → ischemia, paralysis and respiratory failure.
- Block pulmonary capillaries → dyspnea.
- Block coronary arteries → myocardial damage and angina pectoris.
- It is treated by recompression by special pressure chambers (to dissolve the N_2 bubbles) and slowly decompression.



RESISTANCE

$$\text{Flow} = \frac{\text{Pressure difference}}{\text{Resistance}}$$

Definition of Airway Resistance:

- It is the resistance of airway passages to flow of air through them.
- It reflects degree of friction between air molecules and walls of airways during air flow through them.

Poiseuille's Law States That:

$$R = \frac{8\eta l}{\pi r^4}$$

- Where L are the Length and r is the radius of the tube, R is the Resistance, and η is the viscosity of the gas (or liquid) in the tube.
- It is clear from these two equations that Resistance is **critically dependent on the radius** of the tube.

Types of Lung Resistance:

1. Airway Resistance (80%):

A. Nose, pharynx and larynx:

About one-third of total airway resistance arises from the nose, pharynx and larynx.

Mouth breathing (as in exercise) significantly **reduces this value**.

B. Uppermost parts of the bronchiolar tree (Trachea and large {medium} size bronchi) (Site of maximum resistance):

C. Lower bronchial tree (Small bronchioles and terminal bronchioles) (Site of minimum resistance).

2. Tissue Resistance (20%).

Factors Affecting Resistance

Structural Factors

$$R = \frac{8\eta L}{\pi r^4}$$

1. radius — INVERSE RELATION
2. lung volume
3. Cross sectional area
4. Airway length — DIRECT RELATION

Physical Factors

1. Cool Air — DIRECT RELATION
2. Air Density — DIRECT RELATION

Nervous Factors

1. Sympathetic — INVERSE RELATION
2. Parasympathetic — DIRECT RELATION

Chemical Factors

1. VIP — INVERSE RELATION
2. Histamine — DIRECT RELATION

Trans_Pulmonary P.

INVERSE RELATION

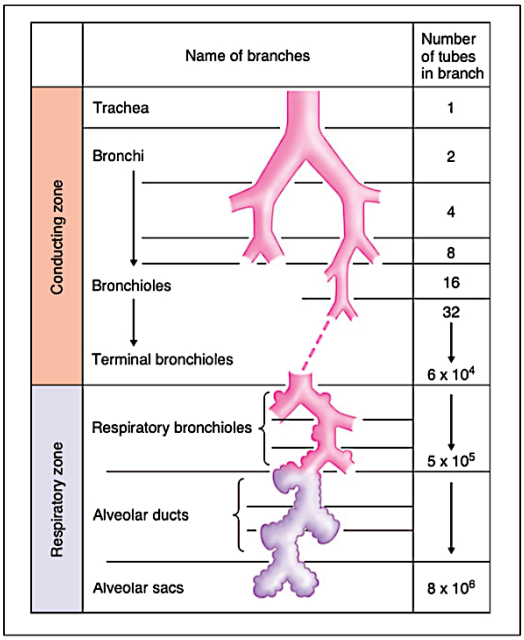
DEAD SPACE (DS)

- **Definition:**

  It is volume of air which **doesn't undergo gas exchange** in respiratory system.

- **Types:**

1. Anatomical DS:
   - Thick respiratory passages (nose → terminal bronchiole).
   - It is called: air conducting zone (16 divisions).
   - It is normally 150 ml.



2. Alveolar DS:
   - Non-functioning alveoli (normally absent).
   - It is normally absent (zero).

3. Physiological DS:
   - Anatomical DS + alveolar DS.
   - It is normally 150 ml (150 + 0).

- **Normal value of DS:**

  - Physiological DS = anatomical DS = 150 ml.
  - In a healthy person, inspiration through a tube leads to **increase** in the dead space and may lead to hypoxia

- **Functions of dead space:**

1. **P**revents marked changes in PO2 & PCO2 in the blood with each respiration.
2. **P**revents marked changes in inspired air temperature & humidity.
3. **P**rotective functions & non respiratory functions. (see before).
4. **It is responsible for the difference between:**

   Respiratory minute volume (**RMV**) & Effective ventilation volume (**EVV**).

	RR	TV	RMV	EVV
Normal breathing	12	500 ml	**6 L / min**	(500 – 150) x 12 = **4.2 L / min**
Shallow rapid breathing	30	200 ml	**6 L / min**	(200 – 150) x 30 = **1.5 L / min**