Physiology of the **Respiratory** system Lecture 3

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Gas transport in the blood

Transport of oxygen

- O2 is transport in the blood in 2 forms:-
- a) Dissolve in plasma (2%)
- b) Bound to haemoglobin (98%)

Dissolved oxygen

It equal to Solubility of $O_2 \times PO_2$

Solubility of $O_2 = 0.003 \text{ml}/100 \text{ ml blood/mmHg}$

- PO₂ =100 mmHg (in arterial blood)and 40 mmHg (in venous blood)
- $_{\odot}$ Therefore dissolved O_2 in 100 ml arterial blood =100×0.003=0.3 ml 0_2/100ml blood
- $_{\odot}$ Dissolved O_2 in 100 ml venous blood = 40×0.003=0.12 ml $_{0_2}$ /100ml blood

 Remember that PO2 in arteries (Pa02) is actually less than 100mmHg (=95mmHg) because of physiological shunt.

Oxygen bound to hemoglobin:-

- ✓ O₂ bind to Hb in a rapid reversible oxygenation reaction (iron remain in the ferrous state)
- ✓ The reaction takes less than 0.01 sec
- ✓ Each gram of Hb can carry up to 1.34ml O_2
- (if 100% saturated as in arteries)

O₂ bound to Hb in arterial blood can be calculated as follows: -

- O2=[Hb]×1.34×[% saturation of Hb with oxygen]
- [Hb]=15g/100ml blood

Therefore $O_2=15\times1.34\times100\%=20ml$ $O_2/100ml$ blood

Oxygen bound to Hb in venous blood can be calculate as follow:-

- O₂=[Hb]×1.34×[%saturation of Hb with O₂]
- Hb=15 g/dl
- Therefore $O_2=15\times1.34\times75\%=15$ ml $O_2/100$ ml blood

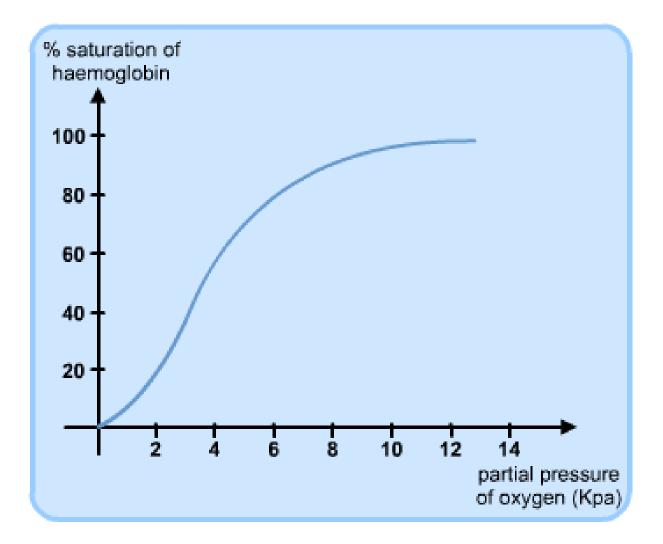
The relation between PO₂ and percent blood saturation of Hb with O₂ is explained by the *oxygen-hemoglobin dissociation curve*

Oxygen-hemoglobin dissociation curve

- This curve indicates direct relation between PO2 and % saturation of Hb with O2
- The sigmoid shaped (start slowly, become steep in the middle and then reaches the maximum)

The sigmoid shape is explained by: -

- 1)O2 bind to subunit of Hb successively (not all of them at the same time)
- 2)The binding with the first subunit facilitates binding with the second subunit and this facilitates binding with the third subunit and so on...
- 3)The facilitation occur due change in the configuration of Hb from tense (T) form to the relaxed (R) form.



Therefore binding:

i. Start slowly: indicating low affinity of Hb to oxygen (occurs when oxygen is binding to the first subunit).

ii.Becomes steep in the middle: indicating high affinity of Hb (occurs when oxygen is binding to other subunits) the affinity is increased up to 500 folds.

iii.Reach maximum at the end (indicating full saturation).

A Notes to remember from the curve

- ☑PO₂ of 40 mmHg gives oxygen saturation of 75 % (as in veins).
- PO₂ of 95 mmHg gives oxygen saturation of 97 %(as in arteries).
- ☑PO₂ of 97mmHg gives O₂ saturation of 97.5% (as in oxygenated blood in pulmonary venous capillaries). Less saturation in arteries (0.5%) is due to physiological shunt.
- $\blacksquare PO_2$ of 26mmHg gives O_2 saturation of 50%. (this is known as the P_{50})

 $\mathbf{\Sigma}\mathbf{P}_{50}$ is defined as the PO₂ when Hb is 50% saturated with O₂ It is used to describe the affinity of Hb to O_2 (e.g. high P₅₀ indicate low affinity of Hb to O_2 whereas low P_{50} indicates the reverse.)

Affinity of Hb to oxygen

-the affinity of Hb to oxygen is affected by certain factors ,these factors can shift the curve to the right or to the left.

-the P_{50} gives information about affinity of Hb to O_2 .Normal value=26mmHg ;higher values indicates shift to the right (low affinity) and lower values indicates shift to the left (high affinity).

Shift to the right

- Indicate lower affinity of Hb to O₂ (high P₅₀).
- Indicate increased release of O₂ to tissues.
- Caused by:-
- High carbon dioxide
- Low pH (high hydrogen ions)
- o High 2,3 DPG
- High temperature

2,3 diphosphoglycerate (2,3 DPG) is product of glycolysis, it is highly present in RBCs when metabolism is increased. It is also increased in exercise, high altitude and by some hormones like growth hormone, thyroid hormones and estrogens. Its binding to the beta chain of Hb decreases the binding of Hb to oxygen.

The ease with which haemoglobin releases oxygen to the tissues is controlled by erythrocytic **2,3-diphosphoglycerate** (**2,3-DPG**) such that an increase in the concentration of **2,3-DPG** decreases oxygen affinity and vice versa.

Shift to the left

- Indicate increased affinity of Hb to O₂ (low P₅₀).
- Indicate decrease release of oxygen to tissues.
- Caused by:-
- $_{\odot}$ Low carbon dioxide
- Low hydrogen ions(high PH)
- Low 2,3 DPG [e.g. due to acidosis(with high anion gap) or in stored blood]
- $\,\circ\,$ Low temperature
- Myoglobin and Haemoglobin F

Remember that: -

- ✓ Shift to the right occur in tissues, where CO₂, H⁺¹ and temperature are high whereas shift to the left occurs in the lung where these factors are decreased.
- ✓ Hb F and myoglobin have very high affinity to oxygen. They shift the curve to the left.
- ✓ Hb F binds less avidly to 2, 3 DPG this increase it's affinity to O₂.
- ✓ Anemia does not affect the shape of the curve because PO₂ is normal and therefore the% saturation of Hb is normal.

Bohr effect: -

- The affinity of Hb to O₂ is decreased when the PH of the blood decrease.
- That is why increase in CO_2 content of the blood decreases the affinity of Hb to O_2 causing shift of the curve to the right.

Transport of CO2:-

- CO₂ is transported in 3 forms:-
- As bicarbonate(the main form of transport)
- Bound to proteins(carbamino compound)
- Dissolved.



- The solubility of CO_2 is higher than O_2 (up to 20 times).
- The dissolved CO₂ constitutes about 5% of total CO₂ in arterial blood and 6% of total CO₂ in venous blood.
- Generally there is no reaction between CO₂ and water in the plasma (due to the absence of *carbonic anhydrase* enzyme in the plasma).

Transport of CO₂ as bicarbonate

- This is the main form of CO₂ transport in the blood.
- CO₂ diffuses inside RBCs and react with water in the presence of *carbonic anhydrase enzyme* to produce carbonic acid and then bicarbonate and hydrogen ion.
- $CO_2 + H_2O = H_2CO_3 = HCO_3^- + H^+$
 - Hydrogen ions are buffered by haemoglobin.
 - About 70% of bicarbonate diffuse to the plasma in exchange to chloride (=chloride shift).

Remember that:-

Due to diffusion of CO₂ into RBCs, the number of active osmatic particles in RBCs is increased by either HCO3⁻ or cl⁻.

So in venous blood, water enters RBCs by osmosis, increasing the size of RBCs. Then it passes out in the lung; when chloride leaves out and the RBCs return to their normal size in arteries.

That is why PCV of venous blood is higher than arterial blood by about 3%

CO2 bound to proteins

- CO₂ form carbamino compound by binding to proteins (plasma proteins in plasma and Hb in RBCs)
- About 11% of CO_2 in the blood is carried to the lungs as carbamino CO_2

Haldane effect

- DeoxyHb in venous blood binds CO₂ more readily than oxy Hb in arterial blood.
- Therefore binding of oxygen to Hb in the lungs facilitates release of CO₂ from Hb ,this is known as the Haldane effect
- for this reason arteries (containing oxygenated blood)carry less CO₂ than veins(containing deoxygenated blood).

In summery

CO₂ is transported in plasma as:-

- \circ Dissolved
- \circ Carbamino-CO₂
- \circ HCO₃
- CO₂ is transported in RBCs as:-
- Dissolved
- o Carbamino Hb
- \circ HCO₃

About 70% of HCO₃ enters the venous blood in exchange to chloride (chloride shift)

✓PCV of venous blood is higher than arterial blood by about 3%

To be continued once

again.....