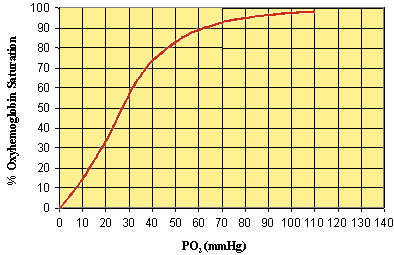
* As we said yesterday:  
  O2 in the blood is carried mostly with Hb and not in its dissolved form. However, each Hb molecule can maximally carry 4 O2 molecules (if it carries 4 then it's fully saturated and if carries 3 then it's 75% saturated >>and so on) but we don’t' have only one Hb molecule we have trillions of molecules in the blood and each Hb carries different number of O2 for example one Hb carries 3 and the other 1 molecule of oxygen and so on thus the average saturation can go from 0 to 100% we don't jump from 0 to 25 to 50 to 75 to 100% because we're not talking about one single molecule we have trillion Hemoglobin molecules.
* Usually oxygen in plasma is expressed as partial pressure, if oxygen is present in the plasma then there is a possibility that the oxygen will diffuse into the RBC and bind with Hb so the independent variable is oxygen in plasma and the dependent variable is oxygen in Hb, this allows us to draw a curve about relationship between Partial pressure of O2 (oxygen in plasma) with oxygen binding/saturation.
* Our aim is to saturate Hb with O2 and to do so O2 must be available in the plasma and the O2 in plasma is originally from alveoli (from the lung), when O2 diffuses from plasma to bind to Hb it's no longer an O2 molecule it becomes oxyhemoglobin, so that drives more O2 diffusion from the lung toward the plasma until Hb is fully saturated with O2   
  …to make it clear: if we have a glass of water containing HB&O2 .. if we add O2 saturation will increase.
* (PO2-O2 saturation)curve it will have three phases:  
  **Phase1:** we increase PO2 but we don't get that much binding with Hb  
  **Phase2** we increase PO2 and they can easily bind to Hb (steep)  
  **note**: steep curve means that any increase in PO2 is associated with an increase in the saturation &vice versa.  
  **Phase3** we increase PO2 but it's very hard for O2 to bind with Hb because it's hard to saturate an already saturated Hb!
* The pivotal point in the curve found in first page is PO2=60, the partial must **NOT** decrease further because after this the curve becomes steep,so this point is considered to be the "controller point" in this curve (it PO2 become less than 60 mmhg, respiratory centers will be activated to drive ventilation .. in hyper ventilation PCO2 in plasma will decrease)   
  example: on mount everest PO2 will decrease to 40 but if we increase the ventilation it might reach 50 but it will not reach 60
* We previously expressed O2 in 3 different ways 1) Saturation 2) Partial pressure 3) concentration, but remember that the first two don’t mean a thing why? Concerning saturation, even if the saturation of Hb is 100% and the Hb conc. is only 5 then the patient is in trouble however, the important one is the concentration and it should be 20ml 🡪Hb in males=15 g/dl…every g contains 1.34 ml O2 … so arterial blood contains 19.5 + 0.3(dissolved)=20 ml O2 in full saturation state(arterial blood) then the cells extract 5ml and the venous blood will have 15ml thus it's considered partially oxygenated.
* Other two important points on the curve are PO2=100 because it's arterial and PO2=40 because it's venous blood.
* If for example I have a patient with Hb=15g/dl(meaning that the blood is fine) and his respiratory system is also working fine but he has a problem with his appendix then there is no need for mask or nasal tube for oxygen supply, cause in supra-saturated blood instead of partial pressure100 in the plasma I have 200 then we are only going to increase dissolved form.

**According to henrys' low = PO2 \* solubility**

In normal situatuion 🡪100\*.003=0.3

And in the case of our patient the PO2 is 200 then 200\*.003=0.6…the increase is only 0.3 thus instead of giving the cell 20 ml it's receiving 20.3 ml and that's not imp. Because the cell is only consuming 5ml so extracting 5ml from 20 or from 20.3 is of no difference.

So as we said PO2=100 is an imp. Point in the graph .. there is no need to go above it.

* During exercise are we satisfied with 200-250ml of O2? No we might need 20X more (5L)

🡪O2 consumption at rest=250 ml   
🡪O2 consumption at maximal exercise = 5 L (20 times more)

Q) HOW CAN WE ACHIEVE THAT 20 TIMES MORE??  
1) By increasing cardiac output (20X) but this is not possible cause maximally we can only increase it 6 times more.

2) By extracting more O2 from the blood... if it was consuming 25% now it should consume 50 or 75🡪that means that the blood in this case starts releasing O2 and hate binding with O2 (so at any PO2 the O2 release is more in this case the curve in page 1 is shifted to the right)

The hemoglobin is made up of α2 β2 subunits and each one of them contains Hb that contains Fe that will bind reversibly with O2, O2 will either bind tightly or not at all either way is not desirable but this is compensated by making Hb hateO2 in lungs and love O2 in the tissues.In an exercising cell the cell is working harder so as a result CO2 is released and it goes and binds with Hb but in a diff. binding site than O2 when Co2 binds it will make the Hb release (hate)O2 and also these cells as a result of working hard they will produce lactic acid(hydrogen) which also bind reversibly to a third binding site causing the O2 to be released

This is called **Bohr's effect**: when CO2 or hydrogen binds to Hb... Hb releases its O2.

**Other factors that cause the release of O2**

🡪The local temp of the exercising muscle will increase and this change the shape of Hb causing it to release O2

🡪The presence of 2, 3-BPG (binds to the same binding site of CO2) will decrease the Hb binding affinity with O2

**Note**: CO competes with O2 at the same binding site(irreversibly) but Hb has 250 time more affinity towards CO than O2   
EXAMPLE: if we brought plasma with partial pressure equals 100 & PCO=100/250=0.4 … 50% of Hb will bind to O2 and 50% will bind to CO.

* **"All of the factors mentioned above shift the curve to the right and if the opposite happens the curve will be shifted to the left"** as in the case of:   
  1- Cold temperature: our nose, hand become red because the blood is keeping the O2 bounded to Hb  
  note: we sometimes need to shift the curve to the left to increase the O2 carried to the lung.. So to decrease CO2 binding affinity so the CO2 is released outside (Haldane effect/ Reverse Bohr's effect)

2- The fetus doesn't breathe alone... He gets O2 from his mother (he's exposed to PO2=40 while we're exposed to PO2=100) so the mother is considered to be the lung of her baby  
...how can the fetus gets the O2 from his mother?  
The fetus has fetal hemoglobin which doesn't bind to 2, 3-BPG... so the affinity of the fetus Hb to O2 is high... and thus the O2 travels easily to his blood.

After birth,fetal Hb is replaced by adult Hb because he's now capable to breathe on his own.

* arterial blood contains48 ml CO2 while the venous blood contains 52 because the tissues give 4 ml to the blood while taking 5ml O2 the 4 ml of CO2is carried in 3 forms:

1- Dissolved in plasma =0.4 (10%)

According to Henry's low:  
in arteries :   
40\*20\*.003=2.4   
in veins:  
45\*20\*.003=2.8   
>>the difference equals 0.4

0.4 Of 4ml🡪 10%

2- Carbaminohemoglobin (not carboxyhemoglobin🡪Hb bound ro CO)...20%  
  
3- The remaining 70% is carried as HCO3-

When CO2 reaches plasma it will immediatley diffuse into the RBCs and undergo this reaction with the help of an enzyme carbonic anhydrase (H2O+CO2🡪HCO3- + H+)... then HCO3- goes to the plasma inorder to compensate for the negative charge lost in the RBC chloride ion enters this is called chloride shift… when the blood reaches the lung the HCO3- enters the RBC again and binds to hydrogen to form carbonic acid which dissociate into water and CO2...then the CO2 goes outside  
\*basically HCO3- is CO2