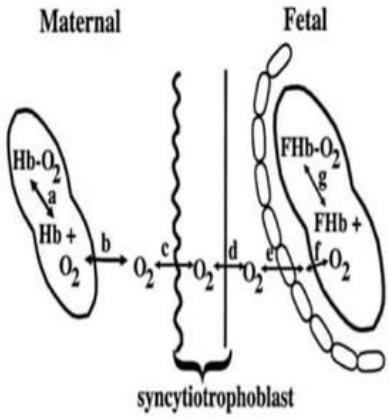
Fetal Blood Oxygenation

The fetus breathes through the placenta; it is the "lung" of the fetus (no external respiration with air). When fetal blood reaches the placenta, PO2 of the interstitium there is only 40mmHg (vs. 100mmHg in alveoli in external respiration).



So, in order to grab the oxygen from the interstitium despite the low PO2, fetal blood contains HbF instead of HbA. HbF has γ chains instead of β chains; so HbF does not bind to 2,3-DPG as avidly as does HbA; so the dissociation curve is shifted to the left, resulting with saturation of hemoglobin at lower PO2.

After birth, the baby's respiration starts, and so, HbF production from the bone marrow decreases, shifting the production towards HbA (shifting the curve back to normal). Normally, HbF levels should be ≤2% of total hemoglobin (more than that may be associated with problems).

CO and Hb

All the allosteric regulators of Hb are noncompetitive (H+, CO2, and 2,3-DPG)(they bind to a different site on Hb)

While the CO is a competitive inhibitor which binds to the same site as the O2, and its binding is 250X of O2 binding(more affinity).

In a blood sample with PO2 of 100(100/250=0.4⇒ same as CO tendency to bind) and PCO of 0.4 the HB will be 50% in a oxy-Hb and 50% in a carboxy-Hb(notice, it is not carbamino-hb)

-if the PCO becomes 0.8 the CO will displace more O2 from Hb (the doctor said that the Hb will be 100% in carboxy form)

Under the effect of CO poisoning:.

PO2 will be normal and equal to 100
Hb saturation of O2 will decrease
And the O2 content of the blood will decrease also

Under the effect of anemia:.
PO2 will be normal (it is affected by the alveolar PO2!)
Hb saturation will be normal (100%)
But the O2 content of the blood will decrease

Why shouldn't we exceed our maximum heart rate?

-When the heart rate is 75 the cardiac cycle time will be equal to 0.8 seconds.
-And we said also that only one third(0.3 s) of the cardiac cycle time(1/3 of 0.8) is more than enough to fully oxygenate the blood.
- During exercise, the heart rate may become 150 Bpm, the cardiac cycle time will become 0.4 s, which is also more than enough for full oxygenation (That's why ABG's remain normal during exercise).
- But if Heart rate jumps to 300 Bpm , Cardiac cycle time will decrease to 0.2 which is not enough for full oxygenation.
- If the heart rate increases above the maximum heart rate, the stroke volume and the cardiac output will decrease. This is due to the reduction in the filling time.
HR increases above the maximum limits⇒diastolic time↓⇒filling time↓⇒SV& CO ↓
- This means that the heart pumps less amounts of blood during a very short cardiac cycle time (the cardiac cycle time now is less than 0.3 which is the required time to fully oxygenate the blood)
- Under these conditions, the person faces 2 bad complication:.
  1-less volumes of blood is pumped
  2-no enough time to fully oxygenate the little amount of blood that reaches capillaries around alveoli.
- These 2 mentioned complications cause **ischemic patches** in the ventricles(especially in those how are elderly)
- Ischemia causes multiple foci of ectopic pacemakers (other than the SA node and the latent pacemakers*) within the ventricular muscle.
*(normally the SA node is the pacemaker, if it is destructed, latent pacemakers (AV node, bundle of His, or the Purkinje fibers) will drive the heart rate).
-Now we know why we shouldn't exceed our maximum heart rate, which can be calculated as follows
Maximum heart rate of the normal people=(220-age)*75%
Maximum heart rate of the athletes =(220-age)*85%
Maximum heart rate of the elderly and those with heart problems =(220-age)65%
