The Physics And Physiology Of Oxygen: An Interdisciplinary Approach To Explain Oxygen Transport To Ischemic / Hypoxic Tissue In Hyperbaric Oxygen Treatment

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BIOPHYSICAL APPLICATION

The transport of oxygen from capillary blood to surrounding tissue has been the subject of considerable analysis, dating from the Krogh tissue cylinder model [17]. We too begin with the Krogh model, where each blood capillary nourishes a surrounding co-axial cylinder of tissue. The radius of the tissue cylinder is obviously larger than the radius of the blood capillary. Within the Krogh model the oxygen transport is considered as a molecular diffusion from the blood into the tissue.

As the walls of blood capillaries constitute membrane, permeable for H_2O, $O_2$and N_2, but block- ing red cells and plasma proteins from entering the tissue, only the oxygen dissolved in plasma is capable to penetrate from blood to tissue. Hemoglobin acts as the oxygen storage, supplying plasma with oxygen and supporting oxygen concentration in plasma accordingly to the Henry law. At ambient conditions the concentration of O_2 in plasma should be expected to be about the same as concentrations of O_2 in sea water at equilibrium with air at 350°C. This value is tabulated and equal to 6 mg/L.

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At hyperbaric oxygen treatment conditions with pressure 2 ATA and with pure oxygen inhalation, the Henry law provides the value of oxygen concentration in blood plasma equal to 60 mg/L. This number is very close to the tabulated value of sea water in equilibrium with oxygen at 2 ATA. Thus, after pressurization in a hyperbaric chamber the blood plasma in the capillary and in the tissue cylinder are in non- equilibrium state in respect to O_2concentration. Not only will the molecular flux from blood to tissue through the wall membrane, but the osmotic flow also cause the transport of oxygen.

The process kinetics was demonstrated by the model experiments provided by Kylstra et al. [8] and supported by original calculations for time dependent and essentially non-steady state osmotic flow and transport, presented for the first time in this paper. At this point the direction of the osmotic flow should be indicated. As human tissue tends to adsorb and even to react chemically with oxygen, the molecular energy of oxygen is at a lower level within the tissue in respect to the energy level in blood plasma. As a consequence, all theoretical conditions are satisfied for anomalous osmosis, causing flow from higher oxygen concentration to the lower one, or from blood capillary into the tissue.

Certain amounts of oxygen enriched plasma will temporary enter the tissue and assist in the healing process. As tissue is elastic in nature, the influx of oxygen enriched plasma will be compensated by counter flow raised by pressure in plasma due to elasticity. From this point on, the molecular diffusion controls the oxygen transport and the osmotic pressure. Essentially it is similar to model experiments, with elastic forces acting instead of gravity head, as gravity is not a factor in this biophysical problem. The longer the treatment session in a hyperbaric chamber, the smaller is becomes the difference in the oxygen concentration between blood and tissue.

However, the transport effect is initially stronger, so the final time should be determined by other causes, medical in nature. It should be noted at this point, that the strong interaction between oxygen and tissue renders the direction of osmotic flow from higher to lower oxygen concentration, namely from blood to tissue. Were it otherwise, the flow direction from tissue to blood would oppose and prevent the oxygen transport from blood to tissue.

The temporarily small additional swelling of tissue by plasma will begin to relax after osmotic pressure reaches its maximum, which is on the time scale of 10 minutes in the model experiments. When the chamber is depressurized and pure oxygen inhaling is terminated, the oxygen concentration in the blood drops to the ambient level in a matter of minutes. Then again the system finds itself in a state far from equilibrium. This time the oxygen concentration in tissue exceeds the equilibrium level. As the process is reversed, the osmotic flow will be directed from the tissue into the blood capillary.

The drainage process will be assisted by forces of elasticity and no traces of additional swelling will be found. Here we can only hypothesize: the hyperbaric oxygen treatment session will contribute to restoration of elasticity of the initially damaged tissue. If this statement is correct, the amount of the liquid drained from the damaged tissue will exceed the volume of plasma which initially entered the tissue. The net effect observed is contraction of the swelling. On the assumption that the damaged tissue elasticity is being restored whilst breathing oxygen, the swelling reduces coherently.

The scenario presented here will not be complete, unless another component of air, namely nitrogen N_2, initially present in blood plasma and tissue as the second solute is examined for its impact on the oxygen O_2 transport. At the start of hyperbaric oxygen treatment the patient inhales pure oxygen and

exhales N_2 along with O_2 and CO_2. No new intake of nitrogen follows during the treatment in the hyperbaric chamber until the end of the session. This leads to the fast loss of N_2, initially dissolved in the blood. This process proceeds simultaneously and with a similar rate as the blood plasma saturation with additional oxygen.

In comparison with O_2, nitrogen is an inert gas, not chemically interacting with the tissue. This means that conditions for normal osmosis caused by N_2 as a solute in blood plasma are satisfied. As nitrogen concentration in the blood plasma becomes lower than its concentration in the tissue, the normal osmosis will cause an additional inflow of plasma into the tissue (from lower to higher concentration of N_2). Thus, the direction of osmotic flow caused by anomalous osmosis due to oxygen coincides with the direction of normal osmosis due to nitrogen concentration gradient. Both components of osmotic flow are directed from blood to tissue.

Therefore the presence of N_2 enhances the rate of O_2 transport. At the end of the hyperbaric oxygen treatment session, depressurization and air inhalation will reverse boundary conditions for osmosis and flow will be directed from tissue into the blood stream, draining plasma from the tissue. The presented mechanism for oxygen transport during hyperbaric oxygen treatment is based on solid physical-chemical grounds. Osmotic phenomena in leaking (sometime called capillary) membranes constitute a very fine chapter in Colloid and Surface Chemistry.

CONCLUSION

Using an increase in ambient pressure with an increase in the inspired oxygen fraction to improve oxygen transport to diseased or injured tissue is fully supported by the physicochemical and biophysical explanations presented here. However, it is recognized that these are not complete and the role of osmotic mechanisms should be the subject of further research.