CELLULAR MECHANISMS OF HYPOXIA

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Tissue hypoxia, which plays a key role in the development of renal and vascular complications of cardiovascular diseases (CVD), might be considered a consequence of vascular remodeling and/or attenuated oxygen (O_2) delivery by erythrocytes. Using Raman spectroscopy (RS) and laser interference microscopy (LIM), we observed that erythrocytes at CVD exhibit changes in the conformation of haemoglobin (Hb) haemoporphyrin (HP), reflecting its lower O_2 transport capacity.The LIM method makes possible to estimate quantitatively a change in the cell optical density at different red blood cell states. This study examined the role of plasma lipids in the regulation of erythrocyte membrane viscosity, oxy-Hb content as well as Na^+/H^+ exchange and Ca^{2+}-ATPase, whose activities are altered in patients with CVD. Both oxy-Hb content and erythrocyte membrane fluidity were decreased in essential hypertension and coronary artery disease patients and negatively correlated with plasma cholesterol but not triglyceride content. This observation allows us to assume that decreased oxy-Hb content in patients with CVD is caused by high plasma cholesterol via attenuation of erythrocyte membrane fluidity and its permeability to O_2. Plasma cholesterol level correlated positively and negatively with erythrocyte Na^+/H^+ -exchange and Ca^{2+}-ATPase, respectively. However, in contrast to membrane fluidity, the impact of these ion transporters in oxy-Hb regulation under baseline conditions seems to be negligible. It was propose that decreased oxy-Hb content contributes to the reduced O_2 tissue supply seen in patients with CVD.