Human, as all multicelullar organisms, is dependant on oxygen availability. Hypoxia is a stimulus which must the organism adapt from early development. In prenatal life fundaments of circulation originate separately and first than they interconnect in complete system of vessel tubes incorporating heart. Haemodynamic - as one of the factors - soon takes part in vessels system development. Restricted blood flow results in vascular bed regression; on the contrary higher flow promotes its development in prenatal life as well as in adulthood.

The aim of our study was to investigate the effect of hypoxia on coronary arterial tree formation and myocardial development and to explain embryonic lethality. Previous studies found ventricular dilatation and increased mortality with signs of heart failure.

We used quail embryos (Coturnix coturnix japonica). Eggs were incubated under hypoxic conditions (16% O_2). Controls were incubated in normal oxygen tension (21% O_2). The effect of hypoxia was analyzed 5 - 9 day of embryo development. Coronary system was visualized by injecting Indian ink. Proliferation was measured using BrdU labelling. For histological analysis we used standard haematoxylin and eosin labelling. For further analysis we used immunohistochemical staining with antibodies against sarcomeric actin (cardiomyocyte marker) and/or smooth muscle actin. As hypoxia marker we used Hypoxyprobe 1. We detected vascular system in section by staining with QH1 antibody (specific for quail endothelial cells).

Hypoxia reduced embryo survival versus controls. Maximal survival period in hypoxia was nine days. In the hearts of embryos developed in normoxic conditions there are present hypoxic areas of tissue. These areas enlarge under experimental conditions (16% O_2). Hypoxia increased capillarization in ventricular wall and in interventricular septum and increased myocardial trabecularization of ventricles and their dilatation. We found thinner ventricular wall (especially in the left ventricle) under hypoxic conditions. In this period myocardium relies on diffusion of oxygen from heart cavities and so these findings can be explained as a compensatory mechanism to minimize diffusion distance for oxygen. In hypoxic conditions coronary vessels are developed irregularly, often without connection to aorta.

Our findings demonstrate the cause of lethal effect of hypoxia. Ventricular wall thinning, coupled with insufficient coronary perfusion leads to dilatation and heart failure.