Abstract – Experimental pulmonary embolism, pathophysiological aspects

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Theoretical part of the thesis deals with mechanisms of pulmonary hypertension after pulmonary embolism (PE) and summarizes the knowledge of literary sources in this issue. Peripherally deals with the mechanical obstruction of pulmonary vessels, particularly discusses vasoconstriction after the PE. In the experimental part are described three separate experiments. The first experiment tested effect of reactive oxygen species (ROS) and preventive administration their scavenger - superoxide dismutase inhibitor tempol on basal perfusion pressure, vasoconstriction, NO synthase activity and the production of NOx after acute PE. Results show that after PE grows NO synthase activity and the production of NOx, the administration of tempol reduces basal perfusion pressure and vasoconstriction after PE. In the second experiment, we measured basal perfusion pressure and vasoconstriction after PE in lungs exposed to 5 day chronic hypoxia. We tested influence of PDE-5 inhibitor sildenafil on the pulmonary vessels tone. Sildenafil decreased basal pressure after PE in chronic hypoxia, its administration does not affect the pulmonary vasoconstriction of pulmonary vessels, but increases their compliance. The last project tested effect of K^+ channels activator flupirtin in hypoxic pulmonary hypertension. Analysis of P/Q line, we found that flupirtin reduces pulmonary vascular resistance. The thesis summarizes existing knowledge in the field of pulmonary hypertension after PE and upgrades them by results of author´s experiments.