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Title: Vasomotor activity of the aorta in rats with experimental COPD

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Body: The aim of the study was to evaluate the endotelium-dependent and endotelium-independent reactions of the aorta reproduced in vivo COPD model. The COPD model was reproduced by chronic smoking in rats within 6 months according to H.Zheng protocol (2009). The experimental group consisted of six male Wistar rats. The control group breathed only clean air. Assessment of the endothelial vasomotor reactions were carried out in 6 months after the experiment start and in 2 months after smoking cessation. Acetylcholine and nitroglycerin were injected in the rats body to assess endothelium-dependent (EDVD) and endothelium-independent (EIDVD) vasodilation, N-monomethyl-L-arginine (L-NMMA) to assess endothelium-dependent constriction (EDVC). Using magnetic resonance imaging the degree of the aorta diameter change in the tomograms before and after drug administration was evaluated. It was found that COPD model has insufficient EDVD and EIDVD (6,6 ± 0,76% and 3,7 ± 0,02% at a rate of 10 and 15% respectively). The test with L-NMMA showed the abnormal vasodilation in contrast to the control group which set the expected EDVC. In 2 months after smoking cessation in the acetylcholine test vasodilation was even less significant decreasing from 6,6 ± 0,76% to 3,07 ± 1,25%, while EIDVD was paradoxical leading to the vasoconstriction ($p < 0,05$). In response to the vasoconstrictor L-NMMA injection the pathological vasodilatation was preserved. Thus the COPD model revealed the violation of the aortic endothelial vasomotor function in the form of the insufficient activity of the vasodilating management and intensification of the vasoconstriction. After smoking cessation vasomotor disturbances are not only preserved but also exarcebated.