Секция «Тезисы на английском языке»

The role of voltage-gated potassium chanels in no-dependent vascular relaxation in pulmonary arteries of rats with unilateral common or external carotid artery ligation

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Abstract: Blood vessels are capable of NO-dependent relaxation through both soluble guanylate cyclase (sGC)-dependent and independent ways. In pulmonary arteries, sGC-independent way is predominantly involved, but in the case of pulmonary hypovolemia the ratio of contributions of sGC-dependent and sGC-independent ways can change and the expression of several types of potassium channels decreases. The model of hypervolemia was established by a superposition of the shunt between the common carotid artery (CCA) and the external jugular vein, which stopped the blood supply to the receptors in the CCA bifurcation and reduced the brain perfusion. It is necessary to clarify the role of reducing of the perfusion of the brain and peripherally located receptors - carotid sinus and carotid bodies - in the changing of NO-dependent dilation of the pulmonary arteries.

Object: The aim of the work is to determine the role of voltage-gated potassium channels in NO-dependent dilatation of the pulmonary arteries after common carotid arteria ligation.

Materials and methods: The ligation of left CCA was performed on white rats weighting 170-230 g. The level NO-dependent relaxation of isolated intrapulmonary arteries perfused in a constant stream was estimated a month later. Sodium nitroprusside (SNP) in concentrations from 10^{-11} to 10^{-7} M was used as NO donor. The evaluation of dilatation was conducted against tonic contraction in response to serotonin perfusion (5×10^{-6} M). sGC inhibitor (ODQ, 10 mM) and K^+ v-channel blocker - tetraethylammonium (TEA, 5 mmol) were used to suppress different ways of NO-dependent relaxation. Morphological characteristics of pulmonary arteries were evaluated the same way. The additional control group for histological study included rats with common carotid artery ligation with disorder of the perfusion only of the carotid bodies.

Results: Response to SNP using ODQ in the group with ligation of CCA is decreased more significantly than in the control group. Statistically significant ($p<0,05$) suppression of relaxation in response to SNP was observed in all ranges of the studied concentrations in the group with ligation of CCA, but in the control group the dilatation under the influence of ODQ changed only in conditions of perfusion of high concentrations of SNP: 10-8M and 10-7M. TEA significantly reduces ($p<0,05$) the efficiency of pulmonary arteries relaxation in the conditions of concentrations of SNP from 10-9M to 10-7M in the control group and from 10-10M to 10-7M in the group with ligation of CCA. Significant increase in the pulmonary arteries wall thickness was observed only in the group with ligation of CCA in comparison with the control group.

Conclusion: Probably the contribution of sGC-dependent vascular relaxation increases in rats with CCA ligation. This may be accompanied by a decrease of the contribution of the NO-induced but sGC-independent way - the direct activation of potassium channels by NO. The absence of perfusion in the carotid body does not affect the pulmonary arteries so strongly as brain ischemia and the lack of perfusion of CCA bifurcation receptors.