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**Excess and deficiency of serotonin in embryogenesis affects the contractility of the right ventricle myocardium**

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Background Congenital heart disease (CHD) is one of the most common pathologies among congenital malformations in children. The incidence of CHD is currently more than 30% of all malformations. The most frequent and severe complication of CHD is pulmonary hypertension (PH). PH worsens the CHD prognosis, affects the survival rate of children, and increases the risk of disability and mortality. Experimental studies demonstrate a high correlation between the PH degree, concentrations of serotonin and its metabolites in the blood.

The purpose of our study was to investigate the effect of excess and lack of serotonin in the embryonic period on the functioning of the heart in early postnatal ontogenesis.

Methods Study was conducted according to "Principles of laboratory animal care". The study was carried out on pregnant female Wistar rats and their offspring at the age of 14 days. Myocardial contractility in vitro was studied on myocardial strips of right ventricle. The amplitude-time characteristics of the contraction were calculated by the method of S. Laer 1998. The following series of experiments were conducted: 1. Pregnant females, starting from the 11th day of pregnancy for 10 days were injected intraperitoneally: group 1 - serotonin synthesis inhibitor (p-chlorophenylalanine) at a dose of 100 mg / kg; group 2 - selective serotonin reuptake inhibitor (fluoxetine) at a dose of 50 mg / kg; group 3 - (control group) - saline solution. Inotropic myocardial function was studied in the offsprings of each group at the age of 14 days. The reactions of the contraction of myocardial stripes of rat pups were evaluated in 2 batches: first on epinephrine and then on the calcium channel inhibitor (verapamil) at concentrations of 2.5 µM and in the reverse order.

Results Our experiment demonstrates that the reaction of inotropic function of the right ventricle on epinephrine in 2 weeks of age rats is not affected by changes in serotonin metabolism in the embryonic period of development. Against the background of a pronounced positive effect from the use of epinephrine, verapamil was used to inhibit calcium channels. We observed a significant reduction in the force of contraction by 83.41 ± 3.14% in the control group, by 71.42 ± 2.95% in the group with an excess of serotonin and by 63.97 ± 7.93% in group with serotonin deficiency. In the next series of experiments, verapamil was first applied, resulting the decrease of contraction strength by 78.58 ± 1.71% in the control group, by 80.81 ± 7.33% in the myocardium of rats with an excess of serotonin and by 67.70 ± 4, 66% in the group with serotonin deficiency. Subsequent exposure of epinephrine led to an increase in myocardial contractility equally in all groups at 132-135%.

Conclusion Violation of serotonin metabolism during embryogenesis affects the regulation of contractility (force of contraction) of the right ventricle.