$$f(V)=\begin{cases}0,\ \varepsilon(V)<-\varepsilon_0\cup\varepsilon_0<\varepsilon(V)\\ 1-(\frac{\varepsilon(V)}{\varepsilon_0})^2,\ -\varepsilon_0\le\varepsilon(V)\le\varepsilon_0\end{cases},$$

where $\varepsilon(V)=\sqrt[3]{\frac{V}{V_0}}$, – elongation of the myocardium, V_0 – is the volume of the chamber in which myocardial contractility is maximal, ε_0 – is the value of elongation at which the myocardium loses its ability to contract.

To establish a relationship between the voltage of the myocardium and the pressure in the cavities of the heart let us consider a sphere of radius r surrounded by a wall of thickness h, consisting of muscle fibers that develop in each point of muscular tension *P* and associated with the elasticity of the passive voltage P_e in the direction of the fibers. Thus, at any point in the wall full tension in the direction of the fibers is equal to $P+P_e$, and the average value of the total stress in any direction perpendicular passing through the given point and the center of the sphere to direct equal t_0: $\frac{1}{\pi/2}\int_0^{\pi/2}(P+P_e)\cos\varphi d\varphi=\frac{2}{\pi}(P+P_e)$, where φ – the angle between the direction of the muscle fibers and the direction. Will rossicum the whole spherical layer through its center plane into two equal hemispheres. According to Newton's third law, the force of muscle tension acting between the hemispheres equal to the force acting on the inner surface of ventricle: $\frac{2}{\pi}(P+P_e)\left[\pi(r+h)^2-\pi r^2\right]=p\cdot\pi r^2$, where p – the pressure inside the chamber. Introducing the volume of the cavity $V=\frac{4}{3}\pi r^3$ and a lot of the myocardium $m=\rho\left[\frac{4}{3}\pi(r+h)^3-V\right]$, where ρ – the density of the myocardium results in: $P=\frac{3\pi\rho V}{4m}\rho-P_e$.

The dependence of passive tension of P_e from elongation λ on the basis of the work is determined by the equation $P_e(V)=\begin{cases}0,\ \lambda(V)<0\\ \alpha(e^{\beta\lambda(V)}-1),\ \lambda(V)\ge0\end{cases}$,

where $\lambda(V)=\frac{1}{1+\varepsilon_\lambda}\sqrt[3]{\frac{V}{V_0}}-1=\frac{\varepsilon(V)-\varepsilon_\lambda}{1+\varepsilon_\lambda}$ – elongation of the myocardium (in contrast to the relative elongation ε of the formula counted relative to V_0, λ is measured relative to $((1+\varepsilon_\lambda)^3V_0)$, ε_λ – the value of the relative elongation ε at which passive tension was missing, α and β – factors that determine the elasticity of the fabric.

Conclusions. Thus, the common mathematical model of the contractile function of the left ventricle and atrium can be on the use of cardiovascular systems in particular, such a common problem, as the mitral valve.

MATHEMATICAL MODELING OF PATHOLOGICAL CONDITIONS OF THE MITRAL VALVE

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Introduction. Mitral valve prolapse (MVP) is one of the manifestations syndrome of connective tissue dysplasia of heart (VDS). Close attention researchers to MVP is associated with a high frequency of symptoms of the problem, in addition, the risk of development of complications such as rhythm

disturbances and conductivity of the heart, the various vessels thromboembolism and sudden death. Timely diagnosis of MVP and evaluation of the clinical significance of this pathology is an actual problem for patients of a cardiological profile, especially among children and adolescents. The detection rate of MVP in children ranges from 2 to 16% and depends on the methods of its diagnosis. The pathogenesis of idiopathic MVP is genetically determined disorders of different components connective tissue (increase in the content of hyaluronic acid, sulfated proteoglycans, collagen types I and III). That leads to «weakness» of the valves mitral valve and to extend into the cavity of the left atrium. Important practical and theoretical importance to study intracardiac hemodynamics at different degrees of this disease.

Aim. The purpose of this work is mathematical modeling of transmitral hemodynamics in MVP, depending on the degree of mitral regurgitation, the contractile function of the left ventricle and atrium. **Materials and methods.** For describing the dynamics of reduction of ventricles and Atria was used the hill equation. Other parameters of heart function were determined according to ECG and hemodynamics of the heart. The proposed mathematical model allows to investigate the influence of the degree of MVP on change of intracardiac hemodynamics. The simulation results are compared with the data of clinical-physiological studies of intracardiac hemodynamics.

Results and discussion. To derive hemodynamic equations consider a sphere of radius *r* surrounded by a wall of thickness *h,* consisting of muscle fibers, developing in each point of muscular tension *P* and associated with the elasticity passive voltage P_e in the direction of the fibers. Based on the equations for maximum isometric tension and muscle tension in the moment time *t* at each point of the muscles of the heart sphere and neglecting the change of the cavity shape, changing the volume has the form: $\frac{dV}{dt} = \frac{dV}{dL}\frac{dL}{dt} = \frac{3V}{L}\frac{dL}{dt} = -3Vv,$ where L – is the linear size of the cavity.

Besides it,
$$\begin{aligned} \frac{dV_{1v}(t)}{dt} &= q_m(\rho_{1v}(t), \rho_{1a}(t)) - q_{ao}(\rho_{1v}(t)), \\ \frac{dV_{1a}(t)}{dt} &= q_p(\rho_{1a}(t)) - q_m(\rho_{1v}(t), \rho_{1a}(t)), \end{aligned} \quad (1)$$

where the cost of blood in the pulmonary circulation $q_\rho(\rho_{1a})$, through the mitral $q_m(\rho_{1v}, \rho_{1a})$ and aortic valves $q_{ao}(\rho_{1v})$ in accordance with Poiseuille's formula are respectively:

$$\begin{aligned} q_\rho(\rho_{1a}) &= \frac{\overline{\rho}_{pa} - \rho_{1a}}{R_\rho}, \\ q_m(\rho_{1v}, \rho_{1a}) &= \begin{cases} \frac{\rho_{1a} - \rho_{1v}}{R_m}, \rho_{1a} \geq \rho_{1v} \\ 0, \rho_{1a} < \rho_{1v} \end{cases} \\ q_{ao}(\rho_{1v}) &= \begin{cases} \frac{\rho_{1v} - \overline{\rho}_{ao}}{R_{ao}}, \rho_{1v} \geq \overline{\rho}_{ao} \\ 0, \rho_{1v} \leq \overline{\rho}_{ao} \end{cases} \end{aligned} \quad (2)$$

Here $\overline{\rho}_{pa}$ and $\overline{\rho}_{ao}$ — the average pressure in the pulmonary artery and the aorta, R_ρ, R_m and R_{ao} — the hydrodynamic resistance of the pulmonary circulation, the mitral and aortic valves. The flow resistance of the pulmonary circulation is equal to the ratio of the difference in average pressure in the pulmonary artery and the PL to the minute volume of blood: R_ρ = (15 – 7) мм рт.ст.·с: 7 л/мин ~ 0,07 мм рт.ст.·с/мл.

Transmitral hemodynamics. Given the equations of the voltage P_{max}, muscular tension at the moment of time *t* at each point of the muscles of the heart sphere and equations volume changes, the relationship between muscle tension and speed reduction takes the following form:

$$\begin{aligned} &\left(\left(\frac{3\Pi p}{4m_{1v}} V_{1v}(t)\rho_{1v}(t) - \rho_e(V_{1v}(t))\right) + c\rho_0 f(V_{1v}(t))n_{1v}(t)\right) \times \\ &\times\left(\frac{1}{3V_{1v}(t)}(q_{ao}(\rho_{1v}(t)) - q_m(\rho_{1v}(t), \rho_{1a}(t))) + cv_0 f(V_{1v}(t))\right) = \\ &= (1+c)c\rho_0 v_0 f^2(V_{1v}(t))n_{1v}(t), \end{aligned} \quad (3)$$

$$\left(\left(\frac{3\Pi p}{4m_{1a}}V_{1a}(t)\rho_{1a}(t)-\mathrm{P}_e(V_{1a}(t))\right)+c\mathrm{P}_0\,f(V_{1a}(t))n_{1a}(t)\right)\times$$
$$\times\left(\frac{1}{3V_{1a}(t)}(q_m(\rho_{1v}(t),\rho_{1a}(t))-q_p(\rho_{1a}(t)))+cv_0\,f(V_{1a}(t))\right)=$$
$$=(1+c)c\rho_0 v_0\,f^2(V_{1a}(t))n_{1a}(t)$$

Equations of the above system of equations contains the amount $V_{1v}(t), V_{1a}(t)$ and pressure $\rho_{1v}(t), \rho_{1a}(t)$ as unknown functions, as well as seven functions $\left(n_{1v}(t), n_{1a}(t), f(V), \rho_e(V), q_\rho(\rho_{1a}), q_m(\rho_{1v,}\rho_{1a}), q_{ao}(\rho_{1v})\right)$, the expressed equations according to the relative number of depolarized cells as a function of the number of depolarized cells, the dependence of passive tension from the elongation and of the system of equations (1). Unknown function is found by integrating the differential equations (1) by Euler's method with step-by-step numerical solution of algebraic equations (3) using conjugate gradient, calculated using the initial values of the form: $\rho_0=\frac{4m}{3\Pi\rho V}(\mathrm{P}_0\,fn+\mathrm{P}_e)$. The initial conditions of the differential equations (1) obtained from the requirement of periodicity: $V_{1v}(0)=V_{1v}(T)$ and $V_{1a}(0)=V_{1a}(T)$. The model calculated the main hemodynamic parameters of the normal heart.

Transmitral hemodynamics with mitral valve prolapse with regurgitation. Mitral regurgitation blood flow will no longer be described by equation (2b). For σ denoting the ratio of the area of the orifice of the mitral valve during regurgitation to the maximum area of its hole, and given that the flow resistance is inversely proportional to the square of the square hole, consider the formula (2b) as follows:

$$q_m(\rho_{1v},\rho_{1a})=\begin{cases}\frac{\rho_{1a}-\rho_{1v}}{R_m}, \rho_{1a}\geq\rho_{1v}\\ \sigma^2\frac{\rho_{1a}-\rho_{1v}}{R_m}, \rho_{1a}<\rho_{1v}\end{cases}$$

In the work carried out comparison of clinical and physiological data with the results of mathematical modeling. To solve the set tasks were examined in 76 children with MVP, 45 girls and 31 boys. Depending on the degree of connective tissue dysplasia of heart, all the children were divided into 2 groups: I – 37 children with MVP 1 degrees and moderate mitral regurgitation (MR) fraction regulierung volume (RF) <30%), II – 39 patients with MVP 2 degree MR and sever (30 % <RF<45%). Echocardiography (EchoCG) was performed according to the standard technique in a one-dimensional and two-dimensional modes on the device "Vivid 7" (GE). MVP criterion was systolic displacement of one or both cusps of the mitral valve into the cavity of the left atrium by 2 mm or more from the access transthoracic parasternal position along the long axis. During EchoCG determined by the diameter and volume of the left and right Atria, end-systolic and diastolic cavity dimensions of the left and right ventricles. We also evaluated the type of hemodynamics, shock volume, ejection fraction, thickness of the LV myocardium. According to color Doppler mapping were determined by the degree MR. Calculation of regulierung volume fraction (RF) was performed using the continuity equation. The mathematical model describes transmitral hemodynamics with account of the pathology of the valve. As follows from the data, PMK complicated with regurgitation, leads to an increase of all indicators, characterizing the size of the heart, such as end-diastolic and end-systolic volumes of LV and LP, shock volume. Due to a compensatory increase in heart size and mass of the myocardium, moderate mitral regurgitation there is an increase in blood volume in the LV, which is determined by the effective shock volume. However, the efficiency of cardiac activity, determined in this case, the effective ejection fraction, is significantly reduced.

Summary: 1. The mathematical model of transmitral hemodynamics in mitral valve prolapse taking into account various degrees of regurgitation. 2. The model allows to determine the dynamics of contractile function of the left heart, hemodynamic parameters including stroke volume, ejection fraction, fluctuations of pressure and transmitral blood flow. 3. The proposed mathematical model allows to give a physical interpretation of intracardiac processes and to evaluate the effectiveness of management of patients with MVP.