MATHEMATICAL MODEL OF THE DESCRIPTION OF CARDIAC CONTRACTIONS

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Introduction. Cardiovascular disease (CVD), according to statistics, consistently rank first among the most common causes of morbidity and high mortality. Every year heart disease kills about 17 million people, representing approximately 29% of deaths. CVD has always been and will be in the attention of many medical scientists and are a relevant topic today.

The most common cardiovascular diseases include infectious endocarditis, myocardial infarction, ischemic heart disease, hypertrophic obstructive cardiomyopathy.

Aim. The aim of the presented robots is a theoretical substantiation of mathematical model of the contractile function of the left ventricle and atrium. For describing the dynamics we used the hill equation. Other parameters of the robots (the elasticity of the myocardium, the characteristic times of the process) was determined according to ECG and hemodynamics of the heart.

Materials and methods. The were used some theoretical methods of research for solving the problem (studying and analysis of scientific at literature to define the condition of development and theoretical basis of the research).

Results and discussion. Consider a mathematical model describing cardiovascular reduction.

1. The dynamics of the polarization of the myocardium. Even in conduction system the rate of depolarization is proportional to the number of unexcited cells of M. Then the equation that determines the dynamics of the depolarization, is: $\frac{dN}{dt} = \frac{ln10}{\tau_d} M$, where N − is the number of active cells, τ_d − the time of depolarization of the myocardium, equal to the period of time for which the number of unexcited cells of the myocardium decreases in ten times. The dynamics of repolarization of the myocardium is described by a similar equation.

Denoting the relative number of depolarized cells $n = \frac{N}{N+M}$, t − time from beginning of depolarization, T, T_d, and T_r = T-T_d, the duration of the cardiac cycle, the period of depolarization and repolarization period, respectively, and proindeksirovat the above equation, obtain the dependence of the relative number of cells depolarized from time to time in the form:

$$n(t) = \begin{cases} 1 - C_1 \cdot 10^{-\frac{1}{\tau_d}} \text{ in the period of depolarization,} \\ C_2 \cdot 10^{-\frac{1-T_d}{\tau_r}} \text{ in the period of repolarization.} \end{cases}$$

where the constants $C_1 = \frac{1-10^{-\frac{T_r}{\tau_r}}}{1-10^{-\frac{T_d}{t_d}-\frac{T_r}{t_r}}}$ and $C_2 = \frac{1-10^{-\frac{T_d}{t_d}}}{1-10^{-\frac{T_d}{t_d}-\frac{T_\gamma}{t_\gamma}}}$ are determined from conditions of continuity and periodicity.

2. The equations describing the dynamics of myocardial contraction. We describe the dynamics of myocardial contraction using the equations of hill, it establishes a link between muscle tension P and a speed reduction v: $(P + cP_{max})(V + cV_{max}) = (1+c)cP_{max}V_{max}$, where P_{max} − the maximum isometric stress, V_{max} is the maximum velocity of isotonic contractions, c − dimensionless coefficient ranging between 0,25 to 0,4. In this case, the voltage of P_{max} and the rate of reduction of V_{max} of the cardiac muscle are functions of the volume of the chamber, and P_{max} is a function of the number of depolarized cells: $P_{max}(t) = Pof(V(t))n(t)$, $V_{max}(t) = Vof(V(t))$.

Approximarely these works a parabola, write $f(V)$ as:

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

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