Analysis of the Optimization Model of Heart Function: Current Aspects

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***Abstract*—The paper examines the approach to mathematical modeling of the pumping function of the heart, which regulates blood circulation in humans. The analysis of the main parameters of the model confirmed the possibility of reproducing the fundamental physiological properties of the heart with this model.**

Keywords—cardiac activity, myocardial contractility, intraventricular pressure, non-invasive method, nonlinear dynamic system.

I. INTRODUCTION

Phase dynamics of intraventricular pressure is an important indicator of heart functioning. To understand this process, mathematical models are used, which take into account the main factors of the contractile activity of the myocardium [1,2].

One of the key components of such a model is consideration of blood dynamics in heart chambers. During the contraction of the myocardium, the volume of blood in the ventricles changes, which leads to a change in intraventricular pressure. The models allow us to predict these changes depending on various parameters, such as myocardial contractility and vascular resistance [3-7].

The purpose of this work is to analyze the mathematical model of the functioning of the heart and to investigate to what extent it allows to reproduce the main integral characteristics of the heart for the assessment of the functional state based on the measurements of the intraventricular pressure and its assessment by a non-invasive method.

II. DESCRIPTION OF THE MATHEMATICAL MODEL

One of the mathematical models used to study the phase dynamics of intraventricular pressure is a model based on the study of the heart as a pump. The proposed approach is based on the presentation of the cardiac function as a nonlinear dynamic system that formalizes the activity of the ventricles of the heart according to the phase structure of the cardiac cycle.

The main features of the proposed model [8]:

1. The model is based on a differential equation with a variable structure and impulse corrections.

2. The periodicity of cardiac activity and the influence of the previous cardiac cycle on the next one are reflected in the model.

3. The index of the model is the value of intraventricular pressure.

4. The volume-pressure dependence is not simply functional, but is presented in the form of a differential ratio that adequately reflects the physics of the process.

5. With this model, the Bowditch effect (dependency of heart contraction force on the heart rate), the Anrep phenomenon (when the pressure in the aorta increases, the heart contraction force automatically increases), the Frank-Starling law (the heart contraction force is greater, the greater the end-diastolic volume the chambers of the heart - but to a certain extent) are reproduced.

Each of these phenomena can be matched with the value of certain parameters of the model.

The use of such an approach allows, firstly, to provide a physical interpretation of the forces and factors that influence contractile activity, and, secondly, to use the apex cardiogram (the registration of the heartbeat determines the mechanocardiogram - the apex cardiogram), which is obtained by non-invasive methods in for diagnostic purposes.

The differential equation that we use to describe the change in blood pressure in the left ventricle is as follows:

$$\ddot{x} + a_i (x - p_{0i})^2 x - b_i \dot{x} + c_i x = 0, \qquad (1)$$

where $x = p - p_c$; $p : [0,\infty) \to R$;

$a_i, b_i, c_i, p_{0i}, p_c$ - certain constants, $i = 1,2$.

For the solution of equation (1), which is characterized by alternating fast and slow motion, secondly, it is an equation of the 2nd order, which allows us to give a physical interpretation of the constants of this equation later on the basis of Newton's second law. Let us give a physiological interpretation of equation (1).

The diameter of the ventricle $l(t)$ at a moment in time t we will call the diameter of the sphere of minimum size, containing a volume equal to the ventricle.

Let's consider the coefficients of equation (1) a_i, b_i, c_i, p_{0i}. One of them is related to aortic pressure (back pressure) and is characterized by a coefficient p_{0i}, and the second, which corresponds to the coefficient a_i prevents the stretching of the aorta. During diastole, the first factor, characterized by the coefficient p_{02}, is related to the venous pressure filling the ventricle, and the second, corresponding to the coefficient a_2, prevents the stretching of the heart muscle.

Note that $p_{01} > 0$, $p_{02} < 0$, $a_i < 0$, $i = 1,2$.

In steady motion, when the system oscillates, its energy must remain unchanged. At the same time, part of the energy is dissipated by the force of friction, so the system must continuously replenish energy from an external force source to maintain undamped oscillations. Such force must be proportional i. The role of the proportionality factor is played by a constant $b_i > 0$, $i = 1,2$. Thus, the third term of the equation represents the force that increases the contractile activity of the myocardium and ensures the rhythmicity of its work. This force is formed by a whole block of controlling factors. Coefficient $c_i > 0$, $i = 1,2$ characterizes the stiffness of the myocardium. In the systole phase, the model interprets this law as the Frank-Starling law, according to which the force of cardiac contraction is proportional to the length of cardiac muscle stretch.

To substantiate the physiological model, all but one of the constants of the model are fixed, and it is proved that the change in the unfixed constant, which corresponds to one of the factors affecting contractile activity, adequately reflects changes in the body. For example, we fix all coefficients except b_i. Research shows that as the ratios increase b_1 and b_2 the length of the cardiac cycle increases, so does the heart rate.

As soon as we look at the depth of the shock volume V and the magnitude of the active factors, the function $V(b_1)$ initially grows, reaches its maximum, and then tends to decline. These details show that the proposed model reflects the Bowditch effect. With growth b_1, the force of shortening $V(b_1)$ increases.

When tracking the stroke volume V, the systole period is seen. Remnants of that same volume (with stationary heart function) are responsible for the hour of diastasis, then it is possible to determine the correlation between the patients, which characterize systole and diastole.

To monitor the consistency of the impact volume V and the stiffness coefficient c_i, $i = 1,2$, we consider the relationship between the diastolic surfaces V and the vice p in the empty ram

$$p = \frac{1}{c_2}(V - U),\ V \geq U$$

where U - unstressed ventricular volume; c_2 - its diastolic distension. At constant p with a decrease c_2, the volume of blood entering the ventricle during diastole decreases. In stationary mode, the same volume will be displaced during systole. This regularity is preserved in the proposed model.

The proposed model allows taking into account pressure dynamics in heart chambers during myocardial contraction and other processes.

In addition to this formula, other parameters or more complex relationships can be added for more accurate modeling of the functioning of the heart.

III. Conclusions

In the proposed model of intraventricular pressure dynamics, the main factors and forces influencing the contractile activity of the heart are concentrated. This approach correlates with works that exemplify a geometric approach to modeling using differential equations.

The application of mathematical modeling and the systems approach in physiology is in good agreement with the main purpose of physiology as a science. Within the scope of this work is the definition of the general principles of functioning and quantitative description of the pumping function of the heart as a theoretical basis for optimizing the functional state and the processes of its correction.

The model is the theoretical basis for optimizing the processes of correcting the patient's functional state.

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