UDC 621.38:577.3 (045)

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CARDIAC ELECTRIC SIGNALS FORMATION MODELING

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Abstract*—*Mathematical modeling method of cardiac electric signals formation in linear and nonlinear formulation is proposed. Mathematical model proposed in paper is quite similar to the initial real object. The processes of electric potentials creation in myocardium cells, namely cardiomyocytes, are investigated. The dynamic characteristics of this system are considered. Numerical modeling of cardiac electric signals formation is provided.

Index terms—Mathematical modeling; cardiomyocyte; heart; myocardium; cell; differential equation.

I. INTRODUCTION

Biological processes which occur in living organisms are the most complicated for understanding and investigation. The chance to investigate these complex processes is to use and create mathematical models. The biocybernetic models of heart which in micro details copies the real heart are very useful for practical medicine. There is a wide area for diagnostics of different heart conditions also.

There are a lot of different mathematical models describing the functioning of heart. But most of them adopt linear approximation and have a limited realization. Various numerical techniques have been used namely: methods of finite elements, methods of finite differences and finite volumes, hybrid finite element difference methods, the interconnected cable method. Description of these methods and numerical techniques are provided, for example, in [1], [2].

Nonlinear mathematical model presented here describes the cardiomyocyte cells activity as a complex system. It can be easily installed into any hardware as it done in programming software MathCAD. The nonlinear dynamics is a useful methodology for a complex system modeling such as heart under normal and pathological conditions. Here we focus on cardiomyocytes because they cause contraction and relaxation of the heart. This is the most valuable function of hear.

II. PROBLEM STATEMENT

Cardiomyocyte is the basic cell of cardiac muscle ([3], [7]). Myocardial muscle fibers providing the contraction function of the heart consists of the muscle cells (cardiomyocytes) which are connected by inserted discs. Diameter of each cardiomyocyte is 10 … 20 mm and length – 50 … 120 micrometers. The excitability of cardiomyocyte arises in response to its stimulation by the electric action potential.

Cardiomyocytes of different species are roughly cylindrical in shape. In atrial muscle, their diameter is less than 10 microns and their length is about 20 microns. In ventricular muscle their diameter is about 17 … 25 mm and their length is about 60 … 140 microns [3], [4]. Cardiomyocytes compose around 30% of the total number of myocardial cells. The volume of cardiomyocytes is around 75 % of the myocardial volume, and mass of cardiomyocytes is more than 50 % of myocardial mass.

Contraction of cardiomyocyte is a specific effect of cardiomyocyte excitation. In other word, it is the process of cardiomyocyte length contraction. The mechanical stress decreasing of cardiomyocyte is accompanied by cardiomyocyte elongation. Excitation of cardiomyocyte and set of cardiomyocytes united in functional syncytia has the same features compare to the excitation of the muscle fibers of skeletal muscle and skeletal muscle as a whole. Contractions of cardiomyocytes and cardiac muscle start with outside action potential, or action potential coming from the pacemaker cells of the heart (e. g., sinoatrial node), or from cardiac conduction system cells (e. g., from the Purkinje fibers), or from neighboring cardiomyocytes.

The signals of the nervous system act through myocardium receptors of the ventricles, atria and conducting system of nodes and the smooth muscle of blood vessels. The regulation is carried out by the direct and feedback connection mechanism. Sensors are the baroreceptors.

The cardiac muscles are the functional syncytia. Each contraction of syncytium involves all cardiomyocytes and all fibers of the heart muscles combined in syncytia. This complex system demands corresponding mathematical description.

III. PROBLEM SOLUTION

The signals of myocardial muscle fibers give information about the state of the heart muscle and any pathological changes. Therefore, modeling of processes occurring in it has wide practical application.

Consider the mathematical model in the form of an equation of electrical state of the nonlinear resonator [5], [6], [8]. We also consider electrical resonator as an element of the circuit as a nonlinear capacitor. This model may be useful as a test of heart conditions. The mathematical model is carried out numerically and the results of calculations describe the heart activity qualitatively.

One-dimensional model of electrical charge q transfer in the nonlinear inertial system in the form of balance equations is

$$\frac{dq(t)}{dt}+\frac{1}{\tau_r(q)}q(t-\tau_0(q))=\sum_{k=1}^{n}\sigma_k(q)u_k(t), \quad (1)$$

where $q=\int_V \rho_{av}dV$, ρ_{av} is the average electric charge density, $\rho_{av}=1/V\int_V \rho dV$, τ_r is electric charge q relaxation time, $\tau_0=l/V$ is the time of electric charge transfer, l is the passing length, V is the drift velocity, σ_k is instantaneous input conductance of the investigated object, u_k is the input voltage, n is the number of inputs, t is time.

The equation of the proposed model is a nonlinear homogeneous differential equation of first order with deviating argument.

The solution of this equation is possible only by numerical methods. To obtain the analytical solutions (in linear approximation) we need to use the different kinds of asymptotic solutions and their approximations.

Let's use such approximation: $\tau_r=\text{const}$ and $\tau_0=\text{const}$. The last fact is justified when the sufficient value of the bias voltage U_0 define the electric field in which the drift velocity of charge reaches a saturation value, i. e. $V=\text{const}$.

In this case, the non-linearity is concentrated in the dependence $\sigma(q)$ in model and the initial equation takes the form

$$\frac{dq(t)}{dt}+\frac{1}{\tau_r}q\left(t-\tau_0\right)=\sum_{k=1}^{n}\sigma_k(q)u_k(t). \quad (2)$$

We can solve the equation (2) analytically using the small parameter method. The small parameter ε is introduced in the equation and the expansion of the parameter q and $\sigma(q)$ is implemented. In this case the equation for $n = 1$ takes the form:

$$\frac{dq(t)}{dt}+\frac{1}{\tau_r}q\left(t-\tau_0\right)=\sigma(q)\left(U_0+\varepsilon U(t)\right). \quad (3)$$

We seek a solution of equation (3) in the such form $q = q(t, \varepsilon)$

$$q=q(t,\varepsilon)=\sum_k q_k(t)\varepsilon^k, \quad (4)$$

with initial conditions

$$q\big|_{t=0}=q(t,\varepsilon)\big|_{t=0}=q_0. \quad (5)$$

Suppose that in a neighborhood of $q=q_0$, and $\sigma(q)$ can be expanded in a Taylor series

$$\sigma(q)=\sum_n \frac{\sigma^{(n)}(q_0)}{n!}(q-q_0)^n. \quad (6)$$

In its turn,

$$\frac{dq}{dt}=\sum_k \frac{dq_k}{dt}\varepsilon^k. \quad (7)$$

Substituting the expressions (4), (6) and (7) in equation (3) we obtain

$$\sum_k\left(\frac{dq_k(t)}{dt}+\frac{1}{\tau_r}q_k(t-\tau_0)\right)\varepsilon^k = \left(U_0+\varepsilon U(t)\right)\sum_n\frac{\sigma^{(n)}(q_0)}{n!}\left(\sum_k q_k(t)\varepsilon^k\right)^n. \quad (8)$$

As a result of equation (8) solution we obtain a system of linear differential equations.

The equation (8) in the first order approximation is:

$$\frac{dq_1(t)}{dt}+\frac{1}{\tau_r}q_1(t-\tau_0)-\frac{1}{\tau_r}\frac{d\sigma_i}{di}\bigg|_{I_0}U_0q_1(t)=\sigma_0U_m\sin(\omega t).$$

Taking into account the external effects in the form $u(t) = U_0 + U_m(t)$, we obtain from equation (8) the equation of the second approximation:

$$\frac{dq_2(t)}{dt}+\frac{1}{\tau_r}q_2(t-\tau_0)-\frac{d\sigma}{dq}\bigg|_{q_0}U_0q_2(t)-1/2\frac{d^2\sigma}{dq^2}\bigg|_{q_0}U_0q_1^2(t)=\frac{d\sigma}{dq}\bigg|_{q_0}q_1(t)U_m\sin(\omega t)$$

Analytical equations of higher order of approximation are even more cumbersome ones.

The dynamic characteristics of the myocardium fiber describing the equation (1) allows to determine only first component of the total current – the conduction current

$$i_c = \sigma(q)\left[U_0 + U(t)\right].$$

The second component of current (bias current) is given by the nonlinear capacity of myocardium fiber:

$$i_C = C(q_C)\left(dU(t)/dt\right),$$

myocardium fibers are characterized by the ambiguity function $\sigma(q)$, the relaxation time τ_0 leads to relaxation oscillations under constant bias value $U_0(I_0)$.

We analyze the relaxation oscillations of myocardium fibers by the model which is described above.

Given the ambiguity function $\sigma(q)$ and assuming $\tau_0 =$ const we present a mathematical model in the form:

$$\begin{cases} \dfrac{dq(t)}{dt} + \dfrac{1}{\tau_r(q)} q(t-\tau_0) = \sigma U_0; \\ G(q,\sigma) = 0. \end{cases}$$

Thus, the analysis of the model is carried out numerically. Dependence $\tau_r(q)$ is given in the form of

$$\tau_r(q) = \tau_0 \Big/ 1 + \left(q/q_0\right)^4.$$

The results of the mathematical equation numerical solution in the form of current pulses i with duration time t qualitatively comparable with experimental results are presented in Figs. 1 and 2.

The results of the offered model show that the form of the received relaxation oscillations qualitatively corresponds to the experimental results. The offered model of charge transfer allows to receive the important dynamic characteristics of a myocardium fiber in the simple ways.

Solving the equation of this model at the different values of the amplitude and frequency of the input signal it is possible to investigate the effect of model parameters on the output signal. The results of the simulation are presented in the form of an output signal waveform and the phase portraits at the different input signal modes (Figs. 3, 4, 5). They show the transition possibility from the periodic oscillations regime (normal heart muscle condition) to the regime of aperiodic oscillations (fibrillation of the heart muscle) at the input signal changing.

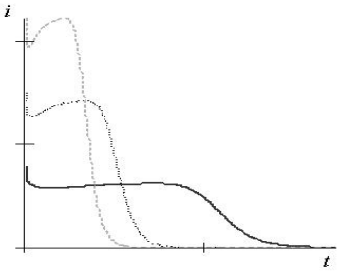

Fig. 1. Simulation of the nonspecific processes (action potentials) in cardiomyocytes during excitation ($U_1 < U_2 < U_3$): —— U_1; • • • • • • U_2; – – – U_3

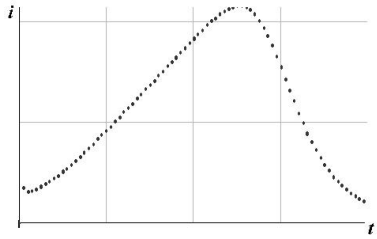

Fig. 2. Simulation of the specific processes in cardiomyocytes during excitation

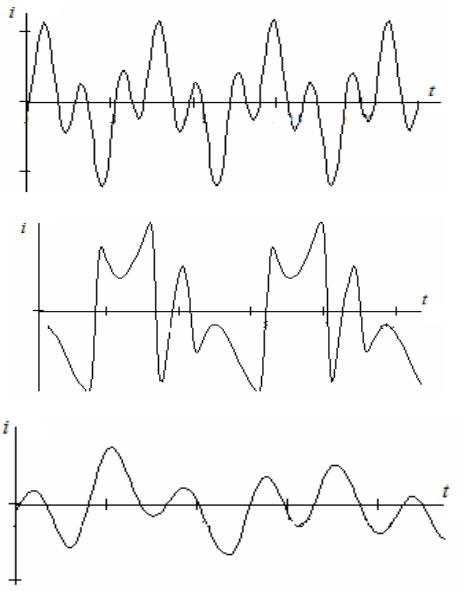

Fig. 3. Dependence of the electrical current i from time t in cardiac myocytes for different values of input voltage

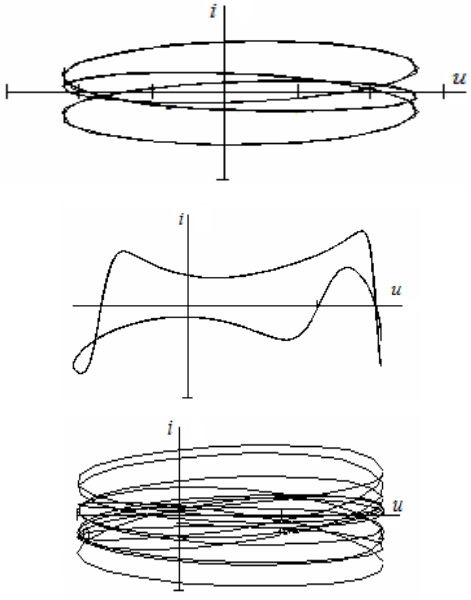

Fig. 4. Dependence of the electric current i from voltage u in cardiac myocytes at the different values of input signal

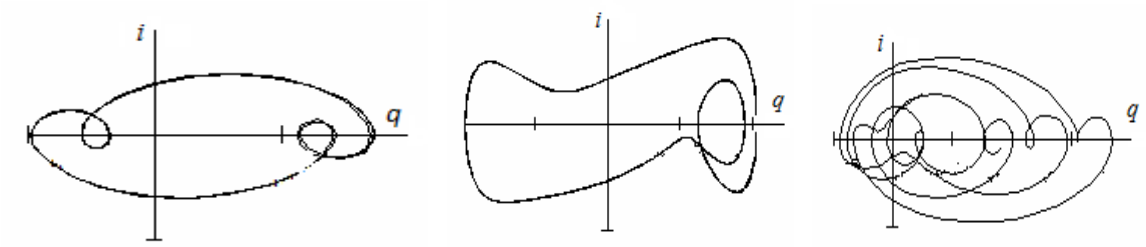

Fig. 5. Dependence of the electric current *i* from electric charge *q* in cardiac myocytes at the different values of input signal

CONCLUSIONS

The aim of research describes how the electric potential of different charges was to present the electronic system for investigation of cardiac electric signals formation in myocardium cells, namely, cardiomyocites of the bioobjects. We have reached the aim through performing certain task. We have created the mathematical model using software MathCAD, which appears in the same cell.

The use of this method allows to simplify the analysis of the cardiac signals because it does not require the implementation of complex algorithms for image analysis [7]. The mechanism of electromechanical conversion in the heart muscle is similar to the mechanism in skeletal muscle.

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Received 10 February 2015.

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Д. Є. Мельников, Д. Е. Азнакаєва, Е. Г. Азнакаєв. Моделювання формування електричних сигналів серця
Запропоновано метод математичного моделювання формування електричних кардіологічних сигналів у лінійному та нелінійному формулюванні. Проведено чисельне обчислення запропонованої моделі.
Ключові слова: математичне моделювання; кардіоміоцит; серце; міокард; клітина; диференційне рівняння.

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Д. Е. Мельников, Д. Э. Азнакаева, Э. Г. Азнакаев. Моделирование формирования электрических сигналов сердца
Предложен метод математического моделирования формирования электрических кардиологических сигналов в линейной и нелинейной формулировке. Проведены численные расчеты для предложенной модели.
Ключевые слова: математическое моделирование; кардиомиоцит; сердце; миокард; клетка; дифференциальное уравнение.

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