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Cardiac Conduction System: A Generalized Electrical Model

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A model of the cardiac conduction system is considered. The model provides an insight into the electrical processes in the myocardium under normal and pathological conditions. The model takes into account the involvement of the atria in generating the sinus rhythm of the heart and the sinus rhythm disorders in arrhythmia. The role of the atrioventricular node in reducing the risks associated with the cardiac conduction disorders in the sinoatrial region is considered. This includes frequency filtering of impulses transmitted from atria to ventricles and the activity of the second-order automatism node. The model can include units for simulating third- and fourth-order automatism nodes and a source of impulses that can cause premature ventricular contraction.

The mammalian myocardial conduction system provides a unique example of the natural pacemaker activity. Various mathematical, technical, and scientific approaches are used to study this system. Under conditions of physical and emotional rest in a healthy subject, the characteristics of the RR intervals are variable and tend to a normal distribution, while a "rigid" cardiac rhythm with virtually absent variability is a sign of gross pathology. According to the academician P. K. Anokhin, a leading figure of the Russian school of physiology, any functional system is a set of dynamic, self-regulating, and self-organizing components, whose interaction is aimed at achieving a useful adaptive result of the system's activity [1].

The activity of the cardiac conduction system (CCS) produces as its adaptive result a normal variable rhythm. This makes it possible to express this result mathematically. It also raises the possibility of developing generalized models of CCS in its normal state and in some types of pathology. For example, there is a mathematical self-oscillating model of a pacemaker based on nonlinear conductivity [2], which considers the combination of a slow depolarization of the sinoatrial node cell membranes with a nonlinear diffusion process.

The cardiovascular system is one of the most important systems of the body. Its rhythmic activity is largely determined by the quality of control [1], which, in turn, largely depends on the CCS activity. The CCS is a system of anatomical structures (nodes, bundles, and fibers) consisting of atypical cardiomyocytes, which ensure the coordinated work of the atria and ventricles aimed at providing normal hemodynamics. Thus, according to the theory of functional systems [1], an adaptive result that is optimal for the metabolic processes is maintained: a variable cardiac rhythm fitted to various functional states of the body.

The CCS consists of two interconnected parts: sinoatrial and atrioventricular. The sinoatrial part includes the sinoatrial (sinus) node (Keith–Flack node), three bundles providing fast internodal conduction between the sinoatrial (SA) and atrioventricular (AV) nodes, and the interatrial bundle providing fast conduction between the SA node and the left atrium. The atrioventricular part consists of the AV node (Aschoff–Tawara node) with an AV connection to the His bundle. The His bundle consists of a trunk and three branches (left anterior, left posterior, and right) leading to the Purkinje fibers.

The SA node is a first-order nomotopic automatism center containing true pacemaker cells (P cells), which automatically generate electrical impulses (action potentials) at a rate of 60-90 min^{-1}, setting thereby the normal rhythm of the heart.

The AV node located at the right edge of the interatrial septum below the oval fossa is one of the most important elements of the CCS. Its function is to conduct exci-

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tation from the sinus node to the ventricles. The AV node ensures stable work of the myocardium both under normal conditions and in many types of arrhythmias [3, 4], significantly reducing the risks from their possible consequences. This is achieved through the implementation of three main functions.

1. Delaying excitation waves traveling along the conduction system from the atria to the ventricles. The delay is achieved due to a significant slowdown in the speed of impulse conduction.

2. Frequency filtering of impulses transmitted from the atria to the ventricles.

3. Implementation of the function of the second-order automatism node.

The resulting delay in the excitation of the ventricles after a full atrial contraction can reach 0.1 s.

Due to the long refractory period of the depolarized cells, the AV node does not conduct signals with frequencies above a certain limit, thereby performing the role of a low-pass filter (LPF). As a result, even with atrial fibrillation, when the frequency of atrial impulses increases to 300 min^{-1} and higher, impulses reach the ventricles at a much lower frequency not exceeding, as a rule, the acceptable limit of 150 min^{-1}. When the function of the sinoatrial node is inhibited, the frequency of spontaneous action potentials of the AV node is about 40 min^{-1}. This is explained as follows. Impulses arising in the sinoatrial node spread to the atria and ventricles and cause their excitation and subsequent contraction. On their way, the impulses pass the second-order automatism center and each time cause a synchronous discharge (excitation) of the pacemaker cells of the center, which at that time undergo slow depolarization. The cycles are repeated until the disappearance of the impulses arriving at the input of the AV node. In this case, the stages of slow depolarization become "complete" and reach the critical threshold level on their own, generating thereby action potentials with a period of approximately 1.5 s. These action potentials are the impulses of the second-order pacemaker.

An electrical model of the AV node was proposed in [5]. The model takes into account the main functions of the node. Unlike mathematical models [6], it can easily be implemented in discrete-analog or digital form, or as a computer program. In [7], a more complete model of the AV connection was considered. It includes an additional unit that simulates the generation of impulses causing premature ventricular contraction. The model takes into account that, along with useful properties, the AV connection can serve as a source of certain arrhythmias in patients with pathological cardiac function. Among the possible arrhythmias generated by this part of the conduction system, premature ventricular contraction – premature excitation under the influence of impulses arriving from various parts of the ventricular conduction system– is the most common (in 60-65% of the adult population). The source of premature ventricular contraction in most cases is the branching of the His bundle and the Purkinje fibers.

Other common types of arrhythmias include atrial flutter and atrial fibrillation. Atrial tachycardias (microreentry and macroreentry, also known as global reentry [3]) are largely associated with the appearance of impulses caused by self-excitation of individual areas of

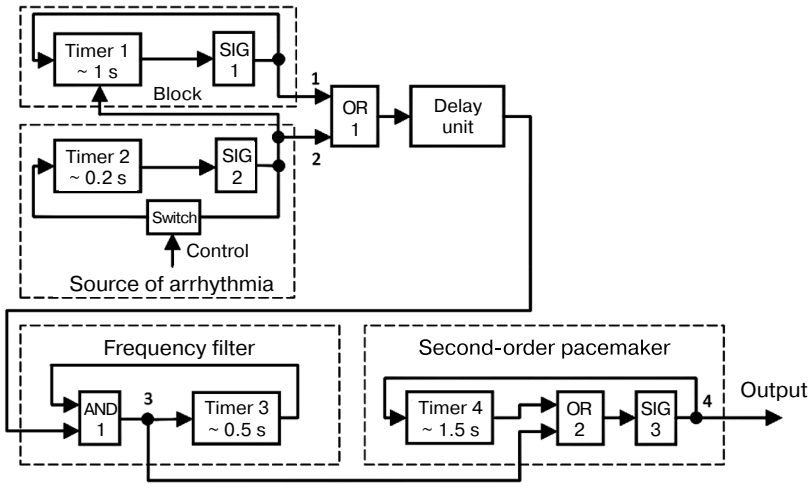

Fig. 1. Structural diagram of the equivalent electrical circuit of the CCS: AND, OR – logic gates; SIG – single impulse generator.

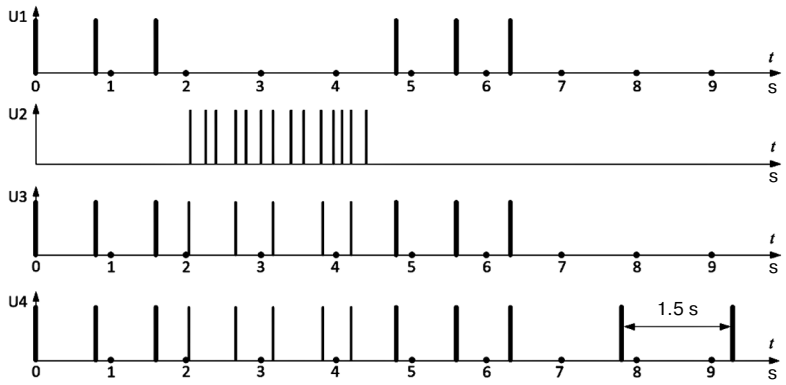

Fig. 2. Timing diagrams demonstrating the activation of the arrhythmia unit (U2), the impulse frequency decimation in the AV node (U3), and the activation of the second-order pacemaker at the eighth second. The delay is not shown.

the atrial myocardium. They emerge as autowave processes in a closed circuit. Despite certain differences, the microreentry and macroreentry mechanisms of arrhythmia have much in common: a high frequency (up to 300 min^{-1} and above) as well as suppression of the sinus rhythm and the normal functioning of the sinus node.

Thus, it is advisable to enhance the electrical model of the CCS by introducing two units simulating the sinus node and the occurrence of arrhythmias in the atria, and by connecting them with a model of the AV node (Fig. 1) [8].

The function of the first-order pacemaker (sinus node) is implemented by timer 1 and a single impulse generator (SIG 1). Its function can be inhibited by signals arriving from the arrhythmia unit (timer 2 and SIG 2). In the model, short rectangular impulses simulate cardiac impulses without loss of generality. The frequency of the sinus node impulses is chosen constant (without taking into account the influence of the sympathetic and parasympathetic systems). The frequency of the impulses of the arrhythmia unit can be set as random within a certain range by programming timer 2.

Time diagrams illustrating the operation of the circuit are shown in Fig. 2. In the absence of atrial arrhythmia, impulses U1 are transmitted from SIG 1 via the OR 1 gate to the delay unit of the AV node model. Here the impulses are delayed by up to 0.1 s (the times of delay and propagation of impulses in the atria are not shown in diagrams). Then, unchanged impulses are outputted by the frequency decimation filter and then by the AV node (4 in Fig. 1).

At the onset of arrhythmia, high-frequency impulses U2 from SIG 2 block timer 1 and are fed through the OR 1 gate to the delay unit and the input of the frequency decimation filter. The filter operates as follows. Before the arrival of the first impulse, the AND 1 gate is opened by applying to it a high potential from the timer 3 output. As a consequence, this impulse is transmitted to the third unit of the circuit. At the end of this impulse, timer 3 is set to zero for a preset time period (determined by the refractoriness of the conductive structures of the AV node; for example, 0.5 s). Only when that time period elapses, the next impulse is allowed to pass. In the case under consideration, it is every third impulse. In this manner, the required decrease in the frequency of impulses passing through the filter is achieved, simulating thereby the unique properties of excitable tissues to carry out frequency filtering of input processes [9]. When modeling, it was taken into account that frequency filtering in the AV node is carried out at very low frequencies (several hertz), so that it would be impractical to use a conventional low-pass filter.

The next unit activates an impulse generator simulating a second-order pacemaker. When impulses are applied to the input of this unit (3 in Fig. 1; U3 in Fig. 2), the generator (timer 4, OR 2, and SIG 3) is synchronized with the input impulses following at a frequency that, as a rule, exceeds 60 min^{-1}. If the input impulses cease, timer 4 counts down the preset time (for example, 1.5 s), after which it starts outputting impulses at a frequency of 40 min^{-1}. CCS model blocks simulating third and fourth-order pacemakers can be implemented in the same way.

As already noted, it is advisable to introduce a premature ventricular contraction simulation unit into the CCS model. Its structure and the principle of its operation are illustrated in Figs. 3 and 4 [7]. In this unit, a counter is used to count the number N of normal cardiac cycles followed by premature ventricular contraction. The latter becomes possible only if the corresponding cells of

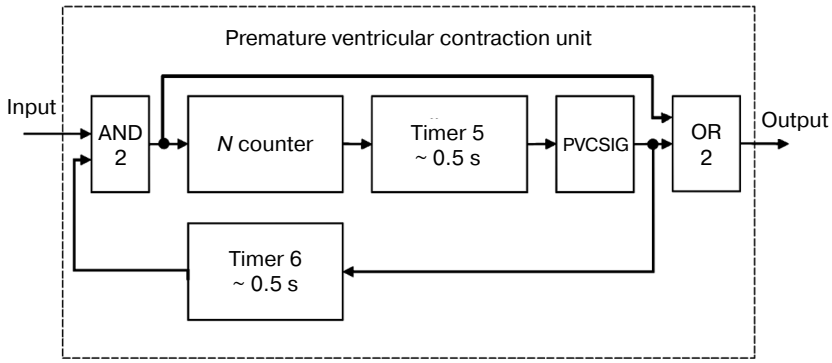

Fig. 3. Structural diagram of the equivalent electrical circuit of the premature ventricular contraction unit.

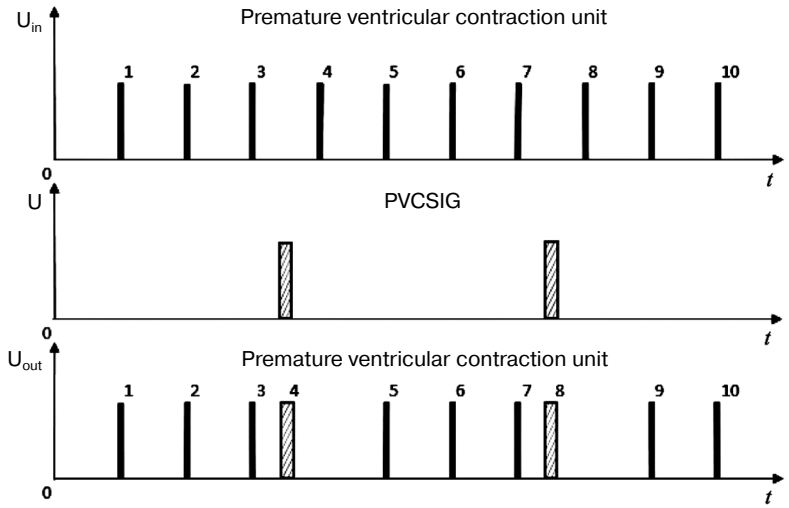

Fig. 4. Timing diagrams demonstrating the operation of the premature ventricular contraction unit.

the conduction system cease to be refractory. This delay in switching on the generator of single impulses simulating the premature ventricular contraction (PVCSIG) is provided by timer 5. After the impulse is generated by PVCSIG, timer 6 blocks the AND 2 gate for a time equal to the refractoriness of the cells of the conduction system in such a way that $N + 1$ impulses are blocked, i.e. each impulse is replaced using OR 3 with the preceding impulse from PVCSIG. In this case, there is a compensatory pause in the ECG between the premature ventricular contraction and the following normal QRS complex.

In Fig. 4, impulses simulating premature ventricular contraction (hatched columns) are shown as having a longer duration. It should be noted that, in practice, N can be random and vary widely. Here, for simplicity and clarity sake, we can take, without loss of generality, that $N = 3$; the refractory time in all cases is taken to be ~0.5 s. It can be seen from Fig. 4 that at $N = 3$ every fourth impulse of the normal rhythm is replaced with an impulse that causes premature ventricular contraction.

Conclusion

Modeling of systems is an essential tool for their study. The cardiac conduction system model presented in this work adequately reflects the main electrical processes both in healthy myocardium and in many pathologies. The model may prove to be of both theoretical and practical interest.

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