Modeling as a Tool for Understanding of Changes in ECG Signals

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Abstract. *An updated program for modeling simplified heart geometry and simulation of action potentials propagation is presented. The implemented model allows simulation of geometry changes as well as changes in action potentials amplitude and duration. In propagation simulation real conduction velocities can be considered.*
Two different pathological situations were simulated using the model. First, only geometrical changes were applied simulating the left ventricular hypertrophy, second, only action potentials properties simulating activation propagation velocity were changed. The resulting ECG signals on the torso were very similar. It is shown that modeling and simulation is useful for explaining of discrepant observations in clinical diagnostics.

Keywords: Computational heart model, Simulation of activation propagation, Modeling of pathologies, ECG signals simulation

1. Introduction

Electrocardiography is considered a basic diagnostic method for different heart diseases providing essential information about the electrical properties of myocytes and activation wavefront propagation that results in systematic electro-mechanical work of the heart.

In this paper an updated programming tool for creation of simplified heart model and simulation of its normal and pathological activity is presented. The importance of using such model for interpretation of observed ECG signals is demonstrated on examples of two different pathologies.

2. Subject and Methods

Heart Model
A simplified geometry of ventricles consists of parts of eight ellipsoids as it is described in [1]. To model the physiological or pathological heterogeneity of action potentials durations, the walls of both ventricles are divided in five layers. For each layer an action potential amplitude and duration can be defined separately as well as the activation propagation velocity (Fig. 1). To mimic Purkynje fiber properties, the activation propagation velocity in the endocardial layer is defined three times higher than in other tissues. It is possible to change the properties of the model in a selected subvolume. The whole modeled volume is discretized using regular cubic grid. The properties of myocardial cells are assigned to each grid element.

The action potential propagation is started in selected starting points according Durrer's experimental findings [2] and later develops in spherical or ellipsoidal wavefronts using cellular automaton principle. The former algorithm computed the activation in discrete time

steps, the new algorithm allows set real values of conduction velocity in simulation and maximal time step in propagation is chosen by the formula:

$$max\_time\_step = model\_grid\_resolution/max\_model\_conduction\_velocity \qquad (1)$$

A realistic action potential shape is predefined according the experiments on canine wedge preparations [3],[4]. The resulting equivalent electrical heart generator is computed in the form of a multiple-dipole. The number/density of dipoles can be chosen as multiple/fold of the basic volumetric element of the model.
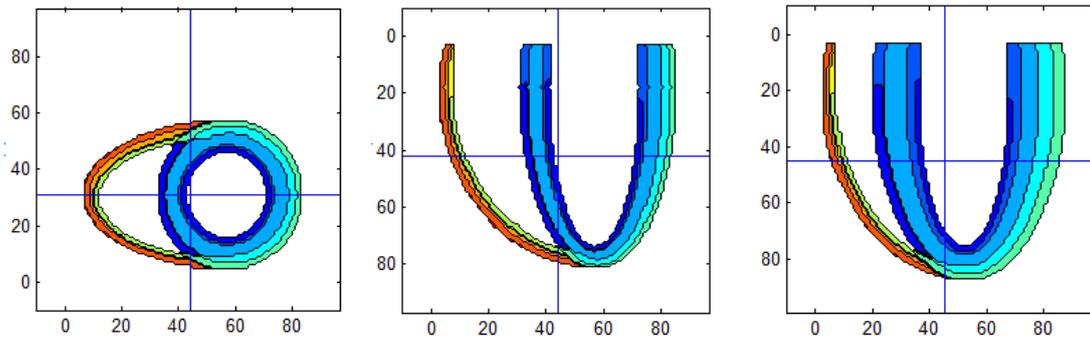

Fig. 1.   From left to right: Horizontal and vertical crosssection of the normal heart model. Vertical crossection of the model of left ventricular hypertrophy. The axes mark dimensions of the model in mm.

Software Implementation of the Model
The program for modeling the heart as an equivalent electric generator is designed as a modular system with graphical user interface. Each program module is represented by separate worksheet (Fig. 2):

M module – creates the geometrical model, allows individual detailed definition of the model size, thickness of anterior, lateral and posterior walls of each ventricle as well as the thickness of ventricular septum and apex. The extent of Pukynje fibers area can also be defined.

P module – allows the definition of a pathological area in the modeled myocardium as the intersection of the modeled heart volume with additionally defined ellipsoid representing the pathological area. In the pathological area the properties of action potentials and conduction velocities can be predefined. This process can be applied multiple times to model complex pathologies.

S module – in this worksheet the activation propagation is simulated in the defined volume. The additional input data are the file with positions and timing of starting points of activation and the file with definitions of action potential properties and conduction velocity.

I module – serves for inspecting the simulation results in the form of 3D visualization of the resulting heart vector and imaging of the propagation wave in chosen time instants. Time series of the x,y,z components of the heart vector are also depicted.

Simulations
Two types of pathologies were simulated using the model. First, anatomical enlargement of the left ventricle called left ventricular hypertrophy (LVH). The thickness of the left ventricular wall was increased by 50% without any other changes of the modeled myocardium. Second, structural changes were modeled, when the conduction velocity in the midwall layers of the left ventricle was slowed down by 50% without any changes in size of the heart model.

The resulting equivalent electrical generator of the heart (multiple-dipole) was inserted to realistically shaped torso model assuming the presence of main inhomogeneities such as the ventricular cavities filled with blood with three times higher conductivity than the mean conductivity of the torso and lung lobes with a conductivity four times lower than the mean conductivity of the torso. The ECG signals s(t) on the torso surface were computed using boundary element method [5] by the formula:

$$s(t) = Ad(t) \tag{2}$$

where A is the time-independent transfer matrix representing the geometrical properties of the torso as piece-wise homogeneous volume conductor and d(t) is the vector of dipole moments of equivalent heart generator in each time step.

The standard 12-leads ECG signals were computed on the surface of an inhomogeneous torso model for both simulated pathological situations and compared with normal reference simulation.
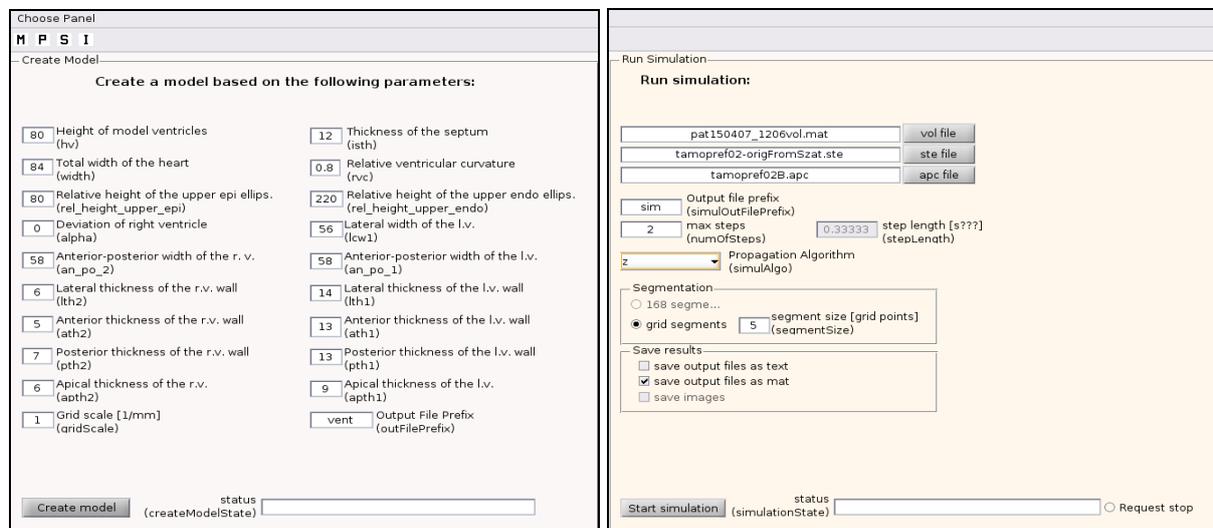

Fig. 2. Examples of worksheets for model creation (M module) and simulation (S module).

3. Results

The resulting ECG signals in precordial leads V1 – V6 computed from normal activation and two simulated pathologies are depicted in Fig 3.
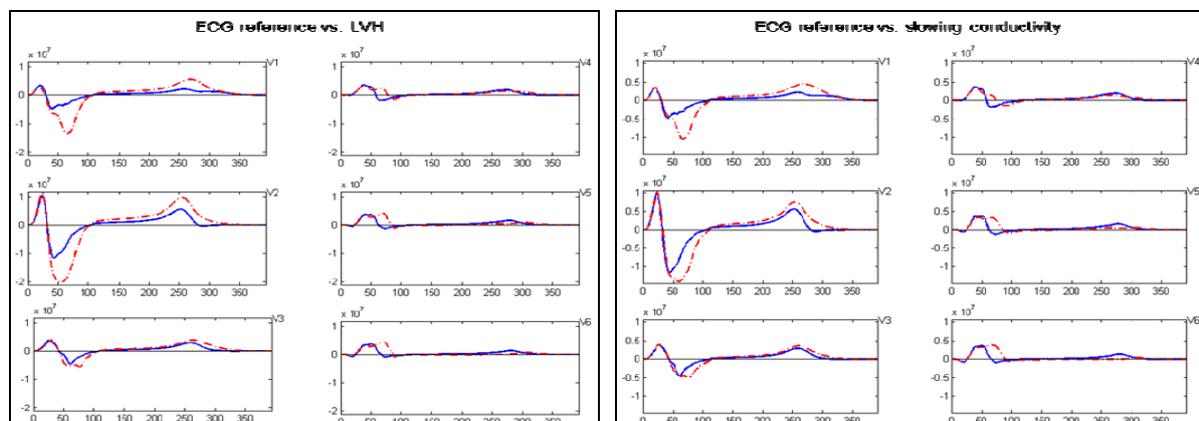

Fig. 3. Computed ECG signals in leads V1-V6. Full lines represent reference signals for normal activation. Dashed lines represent ECG signals for simulation of the left ventricular hypertrophy (left) and for the simulation of slowing down a conduction velocity in the mid-wall layers of the left ventricle (right).

4. Discussion and Conclusions

As it can be seen from the results, the both figures (Fig.3 left, right) are very similar. Dashed lines represent ECG signals of two different types of pathologies. The changes are apparent in the QRS complex as well as in the T wave. The increased signals amplitudes are one of the specific signs of the left ventricular hypertrophy and they were explained by the enlargement of the left ventricular mass [6]. However, in practice, using echocardiographic diagnostic methods in many cases the enlargement of left ventricular mass was not confirmed in spite of the presence of the ECG LVH markers [7]. Our second simulation in which only the conduction velocity was changed can provide an explanation for the contradiction mentioned above.

Although the presented heart model has numerous simplifications, it allows study of various factors influencing the resulting ECG signals, what is helpful in understanding ambiguous or questionable diagnostic results.

Acknowledgements

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