Model-Based Approach to the Localization of Infarction

D Farina, O Dössel

Universität Karlsruhe (TH), Karlsruhe, Germany

Abstract

A model-based approach to noninvasively determine the location and size of the infarction scar is proposed, that in addition helps to estimate the risk of arrhythmias.

The approach is based on the optimization of an electrophysiological heart model with an introduced infarction scar to fit the multichannel ECG measured on the surface of the patient's thorax. This model delivers the distributions of transmembrane voltages (TMV) within the ventricles during a single heart cycle.

The forward problem of electrocardiography is solved in order to obtain the simulated ECG of the patient. This ECG is compared with the measured one, the difference is used as the criterion for optimization of model parameters, which include the site and size of infarction scar.

1. Introduction

Mortality of patients in the immediate postinfarction period, as well as during the first year after myocardial infarction, is most often due to sudden death from ventricular fibrillation [1]. Thus the development of a cardiac model, which would allow to estimate the probability of arrhythmia for a specific patient, must include a correct implementation of ischemia and/or infarction.

The aim of this work is to build a model of the patient's heart that contains infarction scar. It is optimized until the simulated multichannel ECG is as near as possible to the measured one. It is assumed that the location and size of infarction obtained in this manner corresponds to the real one. Taking part in the PhysioNet Challenge 2007 [2] should prove or disprove this assumption.

Within the scope of the Challenge, 4 data sets were proposed for patients suffering from different infarctions. Each of the data sets contained anatomical information about the heart and the locations of 352 electrodes on the surface of the patient's thorax. Corresponding 352-channel ECGs were also provided. The first two data sets were considered as training and contained the information about the parameters of infarction scar. The main task of the Challenge consisted in the estimation of location and size of infarction scar for the latter two patients based on the provided data.

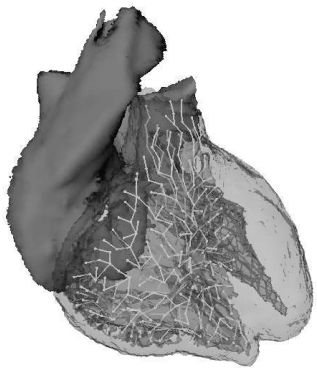

Figure 1. Model of the heart used in this work. Atria, ventricles and excitation conduction system are shown.

2. Methods

2.1. Cellular automaton

A cellular automaton based model of the heart is employed in this work. The anatomy of the heart has been taken from the Visible Male Project [3]. The cardiac geometry is defined on a regular mesh with the resolution of $1 \times 1 \times 1\ mm^3$. This model contains ventricles and atria, sinus and AV-nodes. An excitation conduction system is generated as a tree-shaped structure, with two branches going from the AV-node towards the apex of both ventricles, afterwards giving rise to multiple branches of Purkinje fibers connected to the ventricular endocardium through a large amount of junctions (see figure 1).

The cellular automaton is implemented as follows. If an excitation appears in some voxel, it is transferred to all the neighboring voxels containing excitable tissue. The velocity of excitation conduction for anisotropic ventricular myocardium has been previously determined from the ten Tusscher cell model [4], with different values along and across the fiber orientation.

After a voxel gets excited, its transmembrane voltage (TMV) changes according to the corresponding action potential curve. A family of 97 action potential curves were

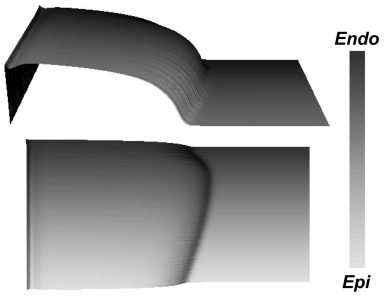

Figure 2. Family of 97 action potential curves employed by the cellular automaton heart model. The curves were generated using the ten Tusscher model of ventricular myocytes in humans [4]. The curve of choice is determined by the location of the voxel in endo-, epi- or midmyocardium.

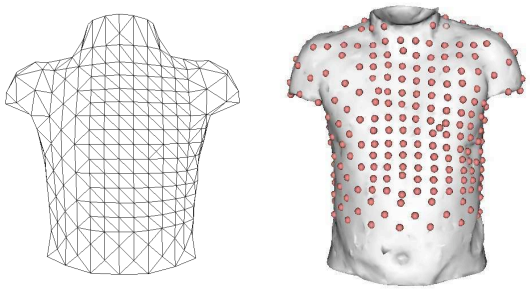

Figure 3. A mesh containing the electrode locations in its nodes provided within the scope of the PhysioNet Challenge (left); same electrode set projected on the thorax surface (right).

generated using endo-, epi- and midmyocardial cell models from [4] (see figure 2), in this way the transmural dispersion of action potential duration (APD) is implemented.

An infarction is introduced as a spherical region with predefined location and size, where the excitation conduction velocity, excitation amplitude and APD are significantly decreased, being zero around the center of the scar. The thickness of the ischemic border of the scar can also be varied.

Thus the heart model delivers the distributions of transmembrane voltages within the heart for a single sinus cycle. For the sake of simplicity only excitation in ventricles has been considered, which results in the absence of the P-wave in simulated ECG.

It takes about 3 min to compute the sequence of TMV distributions for a single heart beat for a model containing about 200,000 active voxels on a workstation with a PowerPC G5 processor and 2 GB of memory.

2.2. Forward problem of electrocardiography

The thorax of the patient is represented by a heterogeneous volume conductor defined on a finite element mesh. The average distance between nodes varies from about 4 mm within the heart to around 10-15 mm in the area distant from the cardiac sources. The overall number of nodes in the mesh is 50,000, forming around 300,000 tetrahedra.

The conductivity values for different tissue classes composing the thorax (heart, lungs, muscles, blood, fat, etc) are mostly taken from the measurements of Gabriel and Gabriel [5]. According to [6], the extracellular conductivity of infarcted myocardium is increased by a factor of 1.75, whereas the intracellular conductivity is fully suppressed due to the closure of gap junctions.

The bidomain model is used to compute the distributions of potentials within the volume conductor from the TMV distributions delivered by the cellular automaton. First, the impressed current density within the ventricular myocardium is calculated:

$$I = \nabla \cdot (\sigma_i \nabla V_m), \tag{1}$$

where σ_i represents the tensor of intracellular conductivity being non-zero only within the ventricular myocardium, and V_m is the distribution of transmembrane voltages within the heart. Afterwards the potentials within the volume conductor are found by the solution of Poisson problem:

$$\nabla \cdot ((\sigma_e + \sigma_i)\nabla\varphi) = -I \; in \; \Omega, \tag{2}$$

$$\varphi = \varphi_D \; on \; \Gamma_1, \tag{3}$$

$$(\sigma_e \nabla\varphi) \cdot \vec{n} = 0 \; on \; \Gamma_2, \tag{4}$$

where σ_e is the tensor of extracellular conductivity being defined within the whole thorax, Ω is the volume of the thorax, φ_D is the reference potential representing the Dirichlet boundary conditions on the surface Γ_1, and Γ_2 is the surface between the thorax and the air where the Neumann boundary conditions are defined.

The electrode locations given by the conditions of the Challenge are projected on the surface of the volume conductor (see figure 3). Potentials obtained from the problem (2)-(4) are recorded at the electrode locations, so the simulated ECG is created.

It has taken about 17 min on a single PowerPC G5 processor to compute a single simulated ECG from a given sequence of TMV distributions. As the cellular automaton and the forward computation were executed in parallel on a dual-processor workstation, this is a good estimation for the time needed for one optimization step.

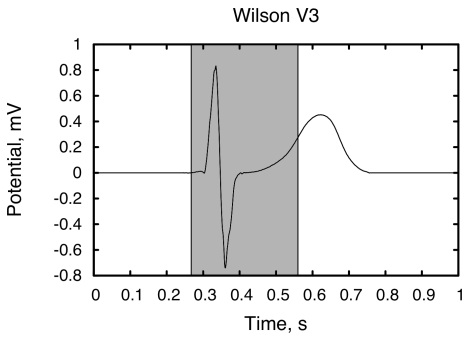

Figure 4. Wilson V3 lead measured for the patient 3. The interval selected for the optimization of the heart model is marked.

2.3. Optimization of the cardiac model

A set of 12 parameters is chosen to be optimized. Excitation conduction velocity is optimized within following tissues:

- Right and left ventricles;
- Right Tawara bundle branche;
- Left anterior and posterior fascicles;
- Right and left Purkinje fibers.

The location and size of infarction scar are described by following parameters:

- Azimuth and declination of the center of infarction scar in the own coordinate system of left ventricle;
- Distance between the infarction center and endocardial surface;
- Size of infarction;
- Size of the border zone between necrotic and healthy tissues.

Downhill simplex optimization method is used. The part of ECG selected for the optimization is shown in figure 4.

3. Results

The infarction scar found for the patient 3 of the Challenge is shown in figure 5. The infarction is located in the lateral wall, with the center in the 12th AHA segment. It covers about 10% of the ventricular myocardium.

In figure 6 the ECG measured on the patient 3 is compared with the simulated one.

Figure 7 depicts the location of the infarction scar found for the patient 4. This infarction covers only 0.2% of the ventricular myocardium. Measured and simulated ECG of the patient 4 are compared in figure 8.

The discrepancies between the obtained results and the real values are shown in table 3.

Table 1. Discrepancies between the estimation of the infarction site and size made in this paper and the real values. EPD: the percentage discrepancy of the extent of infarctions; SO: the overlap between the sets of segments affected by infarction (0-1, 1 is the perfect match); CED: the distance between the centroids of estimated and real infarctions (0-4, 0 is the perfect match).

	Case 3	Case 4
EPD	43	14
SO	0.400	0.167
CED	1	1

4. Discussion and conclusions

The localization error for each infarction did not exceed 1 AHA segment in each case. Still the error of the size estimation was quite large (see table 3). Following reasons might cause such an effect:

- The patient might suffer from ischemia instead of infarction;
- The assumption of spherical infarction scar might be wrong;
- Other modeling errors might affect the quality of solution, such as errors in body geometry, conductivity values etc.

Another problem encountered during the solution consists in a large amount of local minima of the optimization criterion in the space of model parameters. Several different values of parameters might lead to very similar modeling results. At least 3 different sets of initial values were used for each patient in order to find the global minimum.

The optimization-based method of infarction localization needs too much time to be used in clinical practice. But the main goal of this work was to estimate the quality of infarction *modeling*. If the model is correct, the simulation results might be used as *a priori* information in the solution of inverse problem of electrocardiography, for example, using the *maximum a posteriori* regularization [7, 8].

On the other hand, given a correct model of a heart with infarction, it is possible to estimate the probability of ventricular arrhythmia for the patient. This information can be very important for a cardiologist planning the further treatment of this patient.

An important topic of the future research consists in the development of ventricular cell models for ischemic tissue, in order to obtain the change of APD and the amplitude of excitation. This information must be also built into the cellular automaton in order to obtain the proper repolarization sequence for an infarcted heart. This research is currently performed at our institution.

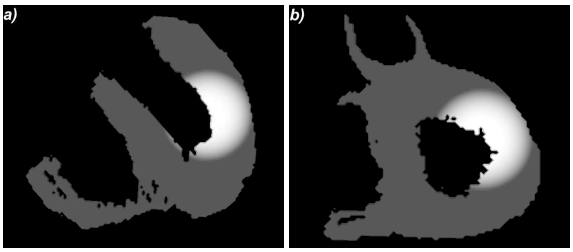

Figure 5. Infarcted area found for the patient 3 of the PhysioNet Challenge. Transverse (a) and frontal (b) slices are shown. Infarcted tissue is shown with white color.

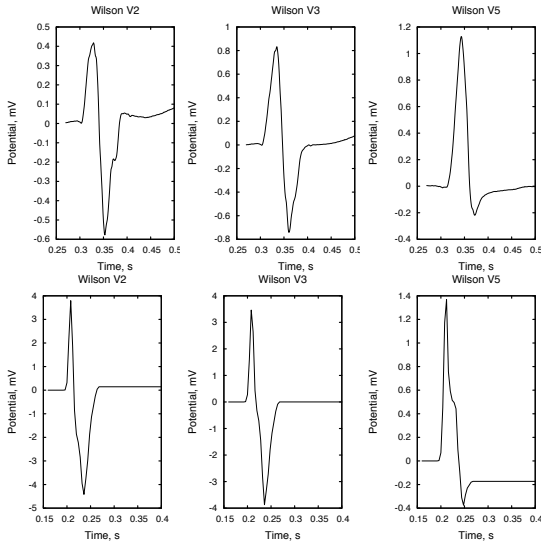

Figure 6. Wilson leads V2, V3 and V5, measured (top) and simulated (bottom) for the patient 3.

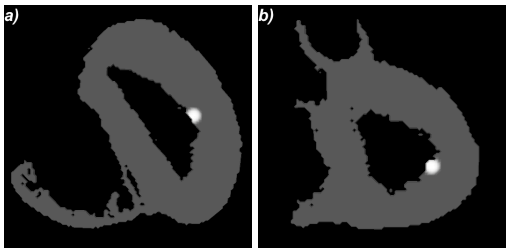

Figure 7. Infarcted area found for the patient 4 of the PhysioNet Challenge. Colors and views correspond to those in figure 5.

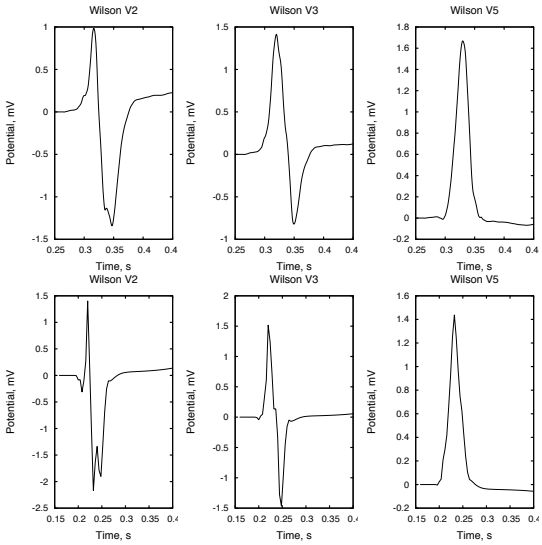

Figure 8. Wilson leads V2, V3 and V5, measured (top) and simulated (bottom) for the patient 4.

Acknowledgements

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Address for correspondence:

Dmytro Farina
Institut für Biomedizinische Technik
Universität Karlsruhe
Kaiserstraße 12
76131 Karlsruhe
Germany
df@ibt.uni-karlsruhe.de