Numerical simulation of mathematical heart model in COMSOL

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Abstract

Electrical activity is essential for the cardiac cell to perform its function. Mathematical modeling of cardiac electrical activity is performed from the cell, tissue and organ levels through to the body surface level. The electrical activity of the cardiac as a whole is thus characterized by a complex multiscale structure. The most complete model of such a complex setting is the anisotropic bidomain model that consists of a system of two degenerate parabolic reaction diffusion equations describing the intra and extracellular potentials in the cardiac muscle, coupled with a system of ordinary deferential equations describing the ionic currents flowing through the cellular membrane. This study describes an anatomically realistic 3D Bidomain model of whole-heart electrical activity. The heart was embedded in a human torso, incorporating spontaneous activation with heterogeneous action potential (AP) morphologies throughout the heart. The aim of this study is the development of a geometrically simple and computationally efficient 3D model of heart. In this paper a finite element formulation, model and simulation of Bidomain equation has been conducted. The FitzHugh-Nagumo (FHN) equations were incorporated into Bidomain model of cardiac electrical activity, which was comprised of a simplified geometry of the whole heart with the torso as an extracellular volume conductor. Laplace equation for the torso also considered. Simplified 3D cardiac model was implemented using COMSOL Multiphysics 5.0 finite element software. Electrical potential at different point on torso is measured. Propagation of electrical excitation on heart surface is also observed. This study represents the first stage toward the development of an accurate computer model of heart activation.

Keywords: Bidomain Model; Electrical activity; FHN equation; Finite Element Method; Mathematical Model.

1. Introduction

Mathematical models of electrical activity in cardiac tissue are becoming increasingly powerful tools in the study of cardiac arrhythmias. Electrophysiological models of the heart describe how electricity flows through the heart, con-trolling its contraction [1]. In modeling the complex whole-heart within body system and also for accurate geometric reconstruction, there are several scale-specific biophysical modeling steps: cell modeling, tissue modeling, whole-heart modeling and organ in the body modeling [2, 3]. The most complete model of such a complex setting is the anisotropic Bidomain model [4] that consists of a system of two degenerate parabolic reaction diffusion equations describing the intra and extracellular potentials in the cardiac muscle, coupled with a system of ordinary deferential equations describing the ionic currents flowing through the cellular membrane [5]. Models of the electrophysiology of one cell are governed by systems of ordinary differential equations and one or more partial differential equations are governed for modeling the electrophysiology of more than one cell.

The modeling process consists of several distinct phases. The first stage involves the identification of the pertinent problem or problems. These problems are then described using the language of mathematics, important variables and features are identified, underlying assumptions are stated, and equations are formulated. The solutions of these equations are then obtained. Once a mathematical solution is obtained, it must be interpreted in the language used to describe the original problem, which again is a translation stage. When such a model has been extensively verified it can become a powerful tool for predicting function, including, for instance, the effect of interventions such as medical treatments [6]. The PDE system made of the heart and torso models is solved using a finite element method (FEM). The finite element method is a numerical method for solving problems of engineering and mathematical physics. Typical problem areas of interest in engineering and mathematical physics that are solvable by use of the finite element method include structural analysis, heat transfer, fluid flow, mass transport, and electromagnetic potential. The finite element formulation of the problem results in a system of simultaneous algebraic equations for solution, rather than requiring the solution of differential equations. These numerical methods yield approximate values of the unknowns at discrete numbers of points in the continuum. Hence this process of modeling a body by dividing it into an equivalent system of smaller bodies or units (finite elements) interconnected at points common to two or more elements (nodal points or nodes) and/or boundary lines and/or surfaces is called discretization. In the finite element method, instead of solving the problem for the entire body in one operation, we formulate the equations for each finite element and combine them to obtain the solution of the whole body [7].

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2. Methods and materials

Ordinary differential equations (ODEs) are used to model the behavior of electricity in a myocardial cell. We may couple a single cell model with a PDE model that describes how electricity flows across a network of cells [8]. One PDE model, called the bidomain model. The bidomain model was developed by Leslie Tung in 1978.

Outside the wall of the heart, the electric potential (V) is governed by

\[ \nabla \cdot (-\sigma_b \nabla V) = 0 \tag{1} \]

Where \( \sigma_b \) is the electrical conductivity of the torso.

Within the myocardial tissues the bidomain model was defined by three dependent variables: \( V_e \) the extracellular potential, \( V_i \) the intracellular potential and \( u \) an excitation variable. The bidomain equations employed utilize FHN type equation for cellular activation. For each region of the heart they are given by

\[ \beta C_m \left( \frac{\partial V_e}{\partial t} - \frac{\partial V_i}{\partial t} \right) + \nabla \cdot (-\sigma_e \nabla V_e) = \beta I_{\text{ion}} \tag{2} \]

\[ \beta C_m \left( \frac{\partial V_i}{\partial t} - \frac{\partial V_e}{\partial t} \right) + \nabla \cdot (-\sigma_i \nabla V_i) = -\beta I_{\text{ion}} \tag{3} \]

With,

\[ I_{\text{ion}} = c_1(V_m - a)(V_m - A)(V_m - B) + c_2 u(V_m - B) \]

\[ V_m = V_i - V_e \]

And,

\[ \frac{\partial u}{\partial t} = e(V_m - du - b) \]

Where \( \sigma_e \) and \( \sigma_i \) are the extracellular and intracellular electrical conductivities within the heart, \( \beta \) is the surface to volume ratio, \( C_m \) is the cell membrane capacitance per unit area, \( I_{\text{ion}} \) is the ionic current per unit cell membrane area and A, B, a, b, e, c_1, and c_2 are parameters describing the active electrical activity of the heart [9].

Parameters of the model are listed in Table 1. Initial values of all model variables are given in Table 2.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>A</td>
<td>55 mV</td>
</tr>
<tr>
<td>B</td>
<td>-85 mV</td>
</tr>
<tr>
<td>a</td>
<td>-66.8 mV</td>
</tr>
<tr>
<td>b</td>
<td>-85 mV</td>
</tr>
<tr>
<td>d</td>
<td>140 mV</td>
</tr>
<tr>
<td>c</td>
<td>285.1 V^{-2} s^{-1}</td>
</tr>
<tr>
<td>c_1</td>
<td>53 nSV^{-2} cm^{-2}</td>
</tr>
<tr>
<td>c_2</td>
<td>400 \mu S cm^{-2}</td>
</tr>
<tr>
<td>c_3</td>
<td>0.02 S cm^{-1}</td>
</tr>
<tr>
<td>C_m</td>
<td>1 \mu F</td>
</tr>
<tr>
<td>\sigma_e</td>
<td>0.008 S m^{-1}</td>
</tr>
<tr>
<td>\sigma_i</td>
<td>100 m^{-1}</td>
</tr>
<tr>
<td>\beta</td>
<td>0.2 Sm^{-1}</td>
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3. Results and discussion

In this simulation Bidomain model for excitation propagation and FitzHugh-Nagumo model for cardiac cell is used. Simplified 3D cardiac model was implemented using COMSOL Multiphysics finite element software.

3.1. Simulation of heart embedded in torso

A physical geometry of heart is built using COMSOL. In order to solve the Bidomain equations, the model which consider contain a highly simplified geometry of the atria consisting a thick spherical shell surfaces, and ventricle consisting an thick ellipsoidal shell surface within torso shown in Figure 1. In this study torso is presented as a continuous medium with higher conductivity. Then heart is embedded inside this medium. For this geometry Bidomain equation coupled with FitzHugh-Nagumo equation are solved to simulate electrical activity, potential at torso and also ionic current. Simulation results are compared with reference data. ECG signal is also simulated in this present study.

For the entire system, PDE (2) and (3) are solved using COMSOL Multiphysics finite-element software. Before simulation meshing for the geometry is done automatically with COMSOL.

<table>
<thead>
<tr>
<th>Parameter</th>
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<tbody>
<tr>
<td>( V_i ) [V]</td>
<td>-0.085</td>
</tr>
<tr>
<td>( V_e ) [V]</td>
<td>0</td>
</tr>
<tr>
<td>( u )</td>
<td>0</td>
</tr>
</tbody>
</table>
The finalized 3D cardiac geometry has 4 domains, 25 boundaries, 44 edges, and 28 vertices. Complete mesh consists of 10075 domain elements, 1556 boundary elements, and 216 edge elements.

Simulated torso potential $V$ at different times are shown in Figure 3 and Figure 4 respectively. Figure 5 and Figure 6 shows the simulated surface potential $V_m$ at different time. It was observed that excitation start at one point of heart and propagate from gradually from one point to next.
The electric potential at different points on the torso was observed. By placing electrode on the surface of the torso, ECG signal can be obtained. In this study, four probes at different points on the torso, as shown in Figure 2, are placed. For simplification, only two probe potentials are given in this paper. Simulated probe potentials are shown on Figure 7 and 8.
Simulation results are compared with reference value. And it was seen that our simulated results are closely similar with references shown in Figure 9.

3.2. Simulation with separating atrium and ventricle embedded within a torso

Atrium and ventricle is separated by a fat layer. So these two parts are electrically isolated. In this section simulation are done in two steps. First with only atrium in torso and next with ventricle in torso. Potential at any point is obtained by combining these two result with a delay for a second.

The effect of only atrium on electrical activity is studied. Figure 10 shown the geometry of atrium and torso. Simulated potential at point 2 of Figure 10 was shown graphically in Figure 11.
Similarly the effect of only ventricle on electrical activity is studied. Electric potential at different points on the torso marked by black dot as shown in Figure 12 were measured and the results is shown in Figure 13.
Then finally potential at same point on torso for atrium and ventricle are added. Manually 0.04 delays are set to propagate activation from atrium to ventricle and this simulation results is also shown in Figure 14. After addition, potential at torso which obtained is similar to the ECG signal.

![Figure 14: Sum of Potential at Point 2 on Torso with Delay.](image)

In practical the A-V node works like a delay station. When an electric impulse comes to the A-V node from atrium here is a little bit delayed before the electric impulse goes down to activate the ventricles. The A-V delays impulse by approximately 0.09s. But in this research atrium to ventricle delay is considered 0.04sec for obtaining ECG signal, others properties of heart are not considered. For getting accurate amplitude and duration as a healthy ECG we should take in account other properties of heart.

4. Conclusion

A fully PDE or ODE based mathematical model for the numerical simulation of ECG signals has been described. This approach has several limitations: I did not consider the AV node, Purkinj fiber and also Bundle of his. It cannot handle complex ionic interactions. The effect of the blood flow was neglected and the geometry of the atrium and ventricles were simplified. Despite the above mentioned limitations, I was able to compute action potential, membrane potential and ionic current. But I have not been able to compute a satisfactory healthy ECG. Numerical simulations on a three dimensional domain were simulated to show the behavior of the excitation spread and the repolarization phase for isotropic electric activity. The present developed code helps to calculate the intra-cellular and extra-cellular action potential of human cardiac tissues in the heart physical domain which can be used to predict the cardio electrocardiograph (ECG). The geometry and dynamics are modifiable with COMSOL Multiphysics based modeling system. Main concern during this study was to build a model to provide ECG and simple enough to be easily analyzed. Future extension of the work presented here is to conduct these studies using a minimally realistic fiber architecture model of the heart. The fibers anisotropy has an important effect on the ECG. The anisotropic structure of the cardiac muscle is to be applied to the modeling system and anisotropic myocardium tissue impedances are to be included in future impedance simulations. The heart model is planned to be modeled as a copy of the heart in a real experiment.

References


