A Coupled Heart-Torso Framework for Cardiac Electrocardiographic Simulation

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Abstract

The existing works of electrocardiogram (ECG) simulation generally use a static or so called "one-way" electromechanical coupled heart model. However, electrical and mechanical activities of the heart are inter-dependent, and realistic ECG simulation can only be achieved when such coupled relationship is considered. In this paper, we propose an electromechanical coupled heart model that includes both electromechanical coupling and mechanoelectrical feedback. The model contains four components: cardiac electrophysiological model, cardiac electromechanical coupling, cardiac mechanics model and cardiac mechanoelectrical feedback. For ECG simulation, the model is incorporated into a coupled heart-torso framework, under which the heart is represented by meshfree nodes and the torso is represented by boundary elements. A direct projection from volumetric transmembrane potential maps (TMPs) to body surface potential maps (BSPs) can be established through meshfree-BEM method. We calculate ECG signals by the proposed framework and compare them with simulation results of a static heart model, and find the amplitude of T-wave is increased and action potential duration is slightly shortened due to the electromechanical coupled property.

1. Introduction

ECG signals remotely reflect electrical activity of the heart, which has been considered as one of the most widely used clinical tools for heart disease detection and diagnosis. Noninvasive imaging electrical activity of the heart from ECG/BSPs has been an active research subject within cardiac electrophysiology community [1]. To achieve the goal, ECG simulation, the forward problem of cardiac electrophysiology, plays a critical role. ECG simulation mainly relies on two ingredients: a heart model and TMPs-to-BSPs projection model.

A heart model describes the mechanisms behind healthy and aberrant heart behaviors. In the existing works, heart models generally only consider electrical activity with the assumption that the heart has no deformation during the cardiac cycle [2]. However, cardiac electrical sources are moving constantly due to the mechanical activity of the heart, thus leads the ECG simulation by a static heart model unrealistic. There are some other works using so-called one-way electromechanical coupling heart models for ECG simulation [3, 4], in which the electrical activity is first determined by an electrophysiological model and then be treated as an input for mechanical activity. Although electrical and mechanical activities are partially coupled, the effect of mechanoelectrical feedback is ignored. The inter-dependent relationship between electrical and mechanical activities determines that realistic ECG simulation can only be achieved when such relationship is included in the heart model.

A TMPs-to-BSPs projection model establishes the relationship between electrical source within the myocardium and electrical potential on the torso, and is usually based on bidomain theory. Based on a heart-torso representation approach, a linear relationship can be established between epicardial or myocardial TMPs and BSPs [2].

This paper aims to propose an electromechanical-coupled heart model for ECG simulation. The model contains both electromechanical coupling and mechanoelectrical feedback. To numerically solve electrical activity within the myocardium and TMPs-to-BSPs projection, we adopt a meshfree-BEM strategy for representing the heart-torso structure [2]. Under this strategy, the heart is represented by a group of unstructured meshfree nodes, while the torso is represented by boundary element method, as showed in Figure 1. ECG singles are calculated and compared to simulation results of a static heart model, and we find the amplitude of T-wave is increased and action potential duration is slightly shortened due to the electromechanical coupled property.

2. Methodology

In this section, we will introduce heart modeling and TMPs-to-BSPs projection model in details.

2.1. Heart modeling

2.1.1. Cardiac electrophysiological model

Cardiac electrophysiological model provides *a prior* knowledge of electrical wave propagation within the myocardium. Although there are various models from cellular to organ level [5], we have selected a monodomain two-variable Aliev-Panfilov model [6] to keep a balance of computational feasibility and physiological plausibility. This model has been widely used for cardiac electrophysiology (EP) simulation [4]

$$\begin{cases} \frac{\partial u}{\partial t} = \nabla \cdot (\mathbf{D}\nabla u) + su(u-a)(1-u) - uv \\ \frac{\partial v}{\partial t} = -e(v + su(u-a-1)) \end{cases} \quad (1)$$

where the variable u stands for normalized transmembrane (TMP), and v is a recovery variable taking charge of the inward and outward ionic currents. $\mathbf{D}$ is conductivity tensor reflecting the anisotropic properties of the myocardium tissue. Parameters a, e, and s determine the shape of TMP, and they are constants in time but not necessary in space.

2.1.2. Cardiac electromechanical coupling

Cardiac electromechanical coupling determines active contraction stresses resulting in electrical excitation. Various models have been proposed in the literature, from realistic complex cellular models [7] to ordinary differential equation (ODE) based phenomenological model [4, 8]. To keep a balance between computational cost and physiology plausibility, we have selected an ODE-based phenomenological model from [4]

$$\dot{\sigma_c} + \sigma_c = u\sigma_0 \quad (2)$$

where σ_c is a scalar related to active contraction stress, and $\dot{\sigma_c}$ is the time derivative of σ_c. σ_0 controls the magnitude of active stress. u is the normalized TMP from cardiac electrophysiological model. Through equation (2), electrical activity is coupled to mechanical activity. Further, we can get the contraction Cauchy stress tensor $\boldsymbol{\sigma}$ by :

$$\boldsymbol{\sigma} = -\sigma_c f \otimes f \quad (3)$$

where f is the fiber orientation of a point inside the computational domain, and $\otimes$ represents the tensor product.

2.1.3. Cardiac mechanics

Cardiac mechanics model describes the passive material properties of the myocardium, which relates the active stresses generated by electromechanical coupling to the heart deformation through equation (6). Since the length of a single cardiac cell changes up to 20% during a heart beat [8], the mechanical analysis should follow finite deformation elasticity theory. With the assumption that the myocardium is elastic, we can establish the stress-strain relation by Hooke's Law:

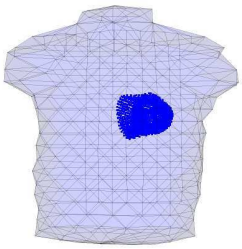

Figure 1. Heart-torso structure represented by Meshfree-BEM method

$$\mathbf{S} = \mathbf{C}\epsilon \quad (4)$$

Here $\mathbf{S}$ is the second Piola-Kirchhoff stress tensor and ϵ the Green-Lagrangian strain tensor. $\mathbf{C}$ is the stiffness matrix accounts for the materials properties of the tissue, which refers to Young's modulus and Poisson's ratio in this paper.

2.1.4. Cardiac mechanoelectrical feedback

Many previous works assumed the effect of mechanical activity to electrical activity can be ignored [1, 3]. However, experimental and clinical research has demonstrated that mechanical activity of the heart affects cardiac electrophysiology [9]. Mechanical activity affects cardiac electrophysiology mainly in two ways: firstly, the position of electrical source inside the myocardium will be changed when the geometry of the heart changes; secondly, stretch-activated ion channels in the cell membrane will be activated when the heart deforms [9]. We only consider the effect of heart deformation to electrical activity to make the problem computational trackable. Following this mechanoelectrical feedback, cardiac electrophysiological model will be solved in the deformed geometry. Finally, we can modify the original electrophysiological model in equation (1) as follows

$$\begin{cases} \frac{\partial u}{\partial t} = \nabla \cdot (\mathbf{D}(\mathbf{F})\nabla u) + su(u-a)(1-u) - uv \\ \frac{\partial v}{\partial t} = -e(v + su(u-a-1)) \end{cases} \quad (5)$$

The variable $\mathbf{F}$ is deformation gradient tensor. The conductivity tensor $\mathbf{D}$ is depended on $\mathbf{F}$, thus mechanoelectrical feedback is naturally introduced into the model.

2.1.5. Cardiac system dynamics

Using principle of virtual work, we can put the first three components into the same framework by using total-

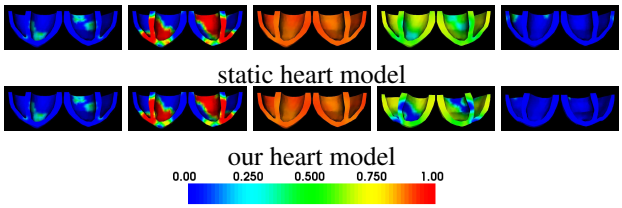

Figure 2. Simulation results comparison between static heart model and proposed heart model. left to right with time, 1 ms, 18 ms, 72 ms, 255 ms, and 288ms. Different color indicates different normalized TMP values

Lagrangian formulation, whose matrix formulation can be represented as follows:

$$ {}^{t}\mathbf{M}\,{}^{t+\Delta t}\ddot{U} + {}^{t}\mathbf{C}\,{}^{t+\Delta t}\dot{U} + {}^{t}\,\mathbf{K}\Delta U = {}^{t+\Delta t}R - {}^{t}R_I \quad (6) $$

Variables with superscript t are measured at time t, and variables with superscript $t+\Delta t$ are measured at time $t+\Delta t$. With ${}^{t}\mathbf{M}$ the mass matrix, ${}^{t}\mathbf{C}$ is the damping matrix, ${}^{t}\mathbf{K}$ the stiffness matrix, and ${}^{t+\Delta t}\ddot{U}$, ${}^{t+\Delta t}\dot{U}$, ΔU are acceleration, velocity and incremental displacement vectors. The vector ${}^{t+\Delta t}R$ is active force from electromechanical coupling component, and ${}^{t}R_I$ is an internal energy term.

By solving equation (6), we can calculate deformation of the heart for each time step. The deformation will be further used to solve the electrical activity in equation (5).

2.2. TMPs-to-BSPs model

Due to the relatively low frequencies in ECG, the quasi-static approximation of Maxwell's equations describes how active cardiac sources determine potential distributions within the torso [2]. The relationship between volumetric TMPs distribution and BSPs distribution can be represented as follows

$$ \nabla \cdot (\sigma \nabla \phi) = \nabla \cdot (-\mathbf{D}_h \nabla u) \quad (7) $$

where ϕ and u stand for BSP and TMP respectively, while σ and $\mathbf{D}_h$ represent torso and intercellular conductivity.

With meshfree-BEM representation of the heart-torso structure as in Figure 1, equation (7) can be further converted into a linear equation

$$ \mathbf{\Phi} = \mathbf{H}(t)\mathbf{U} \quad (8) $$

where $\mathbf{\Phi}$ and $\mathbf{U}$ represent BSP and TMP respectively. Here the transfer matrix $\mathbf{H}(t)$ is depended on time t, since the heart is moving constantly. $\mathbf{H}(t)$ encodes all the structural and electrophysiological information of the heart-torso structure.

3. Experiments

3.1. Heart simulation

To show the electromechanical coupled behavior of our heart model, simulation has been performed on a biventricular heart. The anatomy of the heart is extracted from MRI images, which can be publicly downloaded [10]. After image segmentation and 3D surface mesh generation, the heart is represented by 2017 meshfree nodes bounded by the surface mesh. Since the ventricular conduction system is not available for the current heart, the initial activation sites are selected according to the experimental study from [11].

In Figure 2, we make a comparison between simulation results of our model and a static heart model. We find there are two main differences: first, our model presents both electrical and mechanical behaviors, while static heart model only shows electrical behavior. Second, the action potential duration (APD) of our model is slightly shorter than that of static heart model. As we can see the results at time 255 ms, TMP values of some parts on the endocardium are close to zero by using our model, while the TMP values are larger by using static heart model. This indicates that our heart model repolarizes earlier than static heart model. Considering the depolarization time of both models are almost the same, thus the APD of our model is shorter than static heart model. The main reason of APD shortening is the fiber shortening during heart contraction. Actually, this result is consistent with what found in the work [12], in which the authors used a cellular level electromechanical coupled heart model to simulate ECG in two dimensional space and found that electromechanical coupled heart model can shorten APD compared to a static heart model.

3.2. ECG simulation

For ECG simulation, the anatomy of both heart and torso are downloaded from [10]. By using meshfree-BEM method, the heart-torso structure can be represented as in Figure 1. Again, we select the initial activation sites of the heart according to the experimental study from [11].

Figure 3 depicts normal 12-lead ECG simulation of both static heart model and our model. Compared to a real ECG, we can find that the waves of our model (in red) have a correct orientation in each of the 12 leads. (Currently, we are only interested in the pattern rather than quantitation of simulated ECG). We also compare them with the results of static heart model, and find the results of these two models are pretty much the same except the amplitude of T-wave. The T-wave amplitude of our model is larger than a static heart model, because the heart has the maximum contraction at the T-wave, and the relative distance between heart

and torso is also maximized at T-wave.

4. Conclusion and discussion

In this paper, we present an electromechanical coupled heart model incorporated in a heart-torso framework for ECG simulation. Through heart simulation, we find the APD of our heart model is slightly shorter than that of static heart model. Through normal 12-lead ECG simulation comparison, we can observe the amplitude of T-wave is increased by using our model. The main reason for these phenomena is the heart has the maximum contraction at the T-wave, and thus the geometrical difference between our model and a static model is maximized at that point.

By coupling electrical and mechanical activities, ECG simulation result by our model is more realistic than by static heart model. Nevertheless, there are still some limitations of our work. First, the parameters of our heart model are not personalized, which can be personalized through data assimilation approaches [1]. Second, each component of our model may not be the best model for describing the heart behaviors. Looking for the best model for each component is underway. Third, we assume the mechanoelectrical feedback is only based on heart deformation by ignoring the effect of stretch-activated channels. We can incorporate the stretch-activated channels by adding a term in the cardiac electrophysiological model.

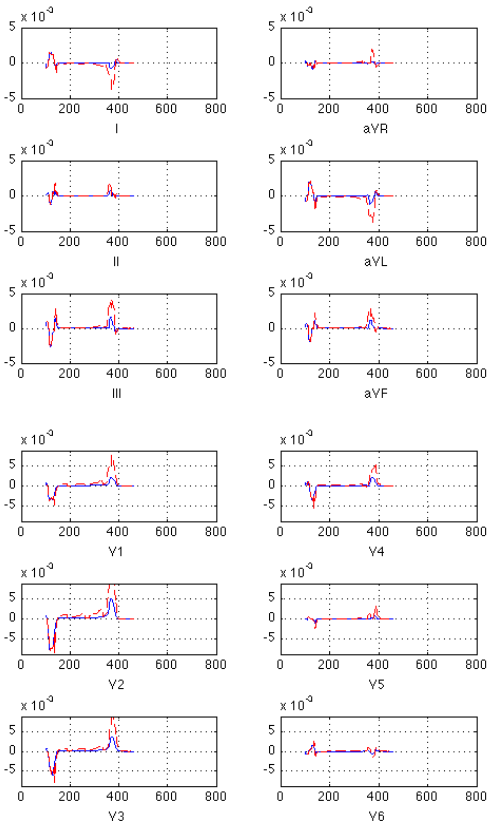

Figure 3. Normal 12-lead ECG simulation, blue: static heart model; red: our proposed heart model

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