

A Finite-Element based Scheme of Cardiac Electromechanics

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Abstract

The propagation of electrochemical waves in the heart initiates the contraction of the cardiac cells. The organization and speed of the electrical waves synchronizes the different cardiac cells and enables the heart to contract as a single organ. Many cardiac diseases are related to the disorganization of the electric activation pattern, the so called cardiac arrhythmias. Among the cardiac arrhythmias, sudden cardiac death is the main cause of mortality in the industrialized world. In addition to the electromechanical coupling, mechanoelectric feedback is a complex issue that may have both pro-rhythmic and arrhythmogenic consequences. For example, mechanical deformation has been shown to alter the electrical properties of cardiac cells and play an important role in ventricular arrhythmias.

Computational modeling is a useful tool for the investigation and comprehension of the complex biophysical processes that underlie cardiac electromechanics. Unfortunately, the multi-physics and multi-scale complexity associated to the phenomenon of cardiac electromechanics translates mathematically to large non-linear systems of partial differential equations.

In a previous work [3] cardiac cell activity was modeled by Hodgkin-Huxley-like models and the non-linear propagation of the electrochemical waves was modeled by the anisotropic bidomain model. Time discretization was implemented via an operator splitting scheme and bi-linear finite-elements are used for the spatial discretization of the equations.

Our preliminary numerical results suggest that tissue mechanics significantly affects the dynamics of electrical propagation as depicted in Figure 1 (see [4] for further details).

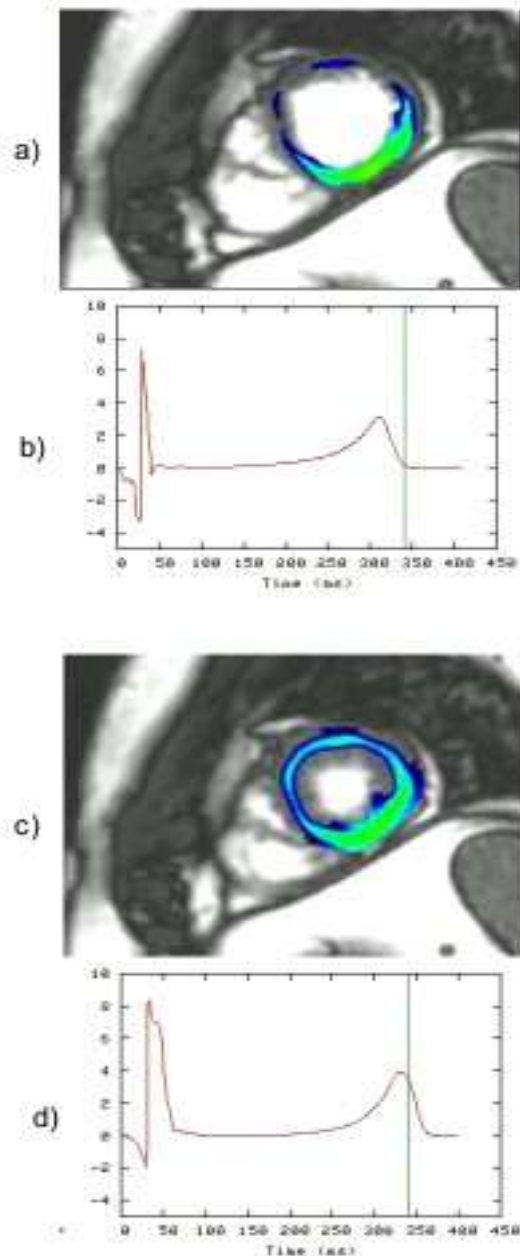


Figure 1: Electrical potential and the corresponding electrocardiograms simulated using different cardiac geometries: a) and b) diastole; c) and d) systole.

In this work, we propose a numerical scheme for the simulation of cardiac electromechanics. The numerical model here proposed accounts for the simulation of a two-dimensional cardiac tissue, the propagation of electric waves on this tissue and the corresponding tissue contraction. Furthermore, the mechanical deformations affect the electrical excitation properties of the tissue, i.e. the model accounts for the mechano-electric feedback.

As in reference [1] cardiac mechanics will be included via the Mooney-Rivlin hyper-elastic material model and the coupling of the electrical and mechanical models will be implemented by a qualitative ordinary differential equation. This equation models the electrically generated active stress (electro-mechanic coupling) and, by including cardiac tissue conductivity changes due to tissue deformation, the mechano-electric feedback. As the model develops large deformations the Newton method is used for the solution of the non-linear equilibrium equation that governs the mechanical activity.

We believe the numerical scheme here proposed for cardiac electromechanics is an important step towards the development of more realistic computer models of the heart.

References

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