PROGRESS TOWARDS AN ELECTROMECHANICAL MODEL OF THE HEART FOR CARDIAC IMAGE ANALYSIS

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ABSTRACT

We present a 3D numerical model of the heart ventricles which couples electrical and biomechanical activities. We have adopted a simpler formulation of previously published models to achieve a trade-off between biological accuracy and computational efficiency. To carry out computations, the FitzHugh-Nagumo equations are solved along with a constitutive law based on the Hill-Maxwell rheological law. Ultimately, the parameters of this generic model will be adjusted from the actual patient's ECG and from the cardiac motion measured in 4D cardiac images. By including biological and physical *a priori* knowledge, we expect to extract quantitative ventricular function parameters from time sequences of cardiac images[1].

1. INTRODUCTION

The clinical motivation of this work is the quantitative measurement of important ventricular function parameters from cardiac images, like the ejection fraction, the myocardium thickness and the local strain and stress. Those parameters are useful to detect ischemic zones, measure the pathology extent and control the therapy effectiveness.

The key idea of this paper is to build a "Beating Heart Model" which contracts under electrical excitation. By fitting this model to cardiac image sequences we intend to recover those ventricular function parameters. The knowledge of the heart function has greatly improved at the nanoscopic, microscopic and mesoscopic scales during the last decades, thus a global integrative work of this organ becomes conceivable [2]. Although this is a work in progress, we believe that the proposed framework should allow a better understanding of the heart behavior by measuring quantities such as stress that are beyond the pure geometric description currently achieved and it should help interpret cardiac images.

Our "Beating Heart Model" is based on mathematical systems of (non-linear) partial differential equations, set on a three dimensional domain, considering the ventricles as a continuum. Some of these equations are motivated by a realistic mechanical modeling of cardiac fibers occurring at the nanoscopic level. It is built in order to achieve a reasonable trade-off between the accuracy that is needed for the assessment of ventricular function and computational efficiency. This last quality is highly desirable to achieve a possible future clinical use for cardiac image analysis. We do not intend to simulate the detailed biological reality with this model, but to establish realistic and biologically based constraints for a segmentation process.

The long-term objective is to build a procedure that automatically adjusts all those electrical and mechanical parameters to the dataset of the patient, through a feedback loop (Fig. 1).

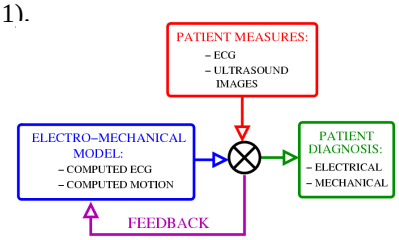

Fig. 1. Global scheme of the long term objective.

2. INTERACTION WITH CARDIAC IMAGES

Many different techniques have been used in cardiac image analysis with 3D models [3]. In the deformable model field, some of these ventricular function parameters can be efficiently extracted from the deformation of geometric surfaces. But these surfaces do not include any biological or physical *a priori* knowledge to guide their deformations where boundary data is missing. Moreover, only the apparent motion (ie. displacement along the normal direction) can be reconstructed. Recently, volumetric models were used [4, 5], they have a strong topology constraint and they can easily include *a priori* information [6]. In the heart motion, there is a twist during contraction, using biomechanical volumetric models could help recover this tangential displacement.

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In the deformable model framework, a model evolves under the influence of two energies: an *External Energy* which makes the model fit the images and an *Internal Energy* which acts as a regularization term and can include *a priori* information (shape, physical properties, motion,...)

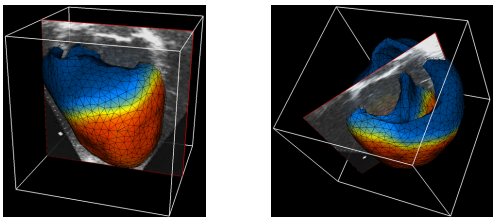

Fig. 2. Electromechanical model in a 4D ultrasound image.

In our approach, the computation of this *External Energy* at a surface vertex depends not only on the vertex location but also on its normal direction. Different type of forces may be applied depending on the image modality. We chose to combine intensity and gradient information with a region-based approach [7] applied to the intensity profile extracted at each vertex in its normal direction. It consists in defining a region with a range of intensity values and then finding its boundary by looking at the voxels of high gradient value. The extent of the intensity profile is decreased in the coarse-to-fine process. Then, we apply a force F_i which is proportional to the distance to the closest boundary point of the image from the considered point of the mesh surface.

The volumetric nature of our model strongly decreases the importance of the image outliers in the motion estimation since it strongly constrains the geometric (for instance the thickness of the myocardium wall) and physical behavior.

The *Internal Energy* corresponds to an electromechanical "Beating Heart Model" which is described in the next three sections: first, the anatomical data necessary to build the model, then the electrical model used to compute the action potential wave propagation and finally the mechanical model used to compute the contraction triggered by the action potential wave.

3. ANATOMICAL MODEL

To build our model we need data regarding both the 3D ventricular geometry and the muscle fiber directions. Indeed, the anisotropy created by those fibers intervenes in both the electrical wave propagation and the mechanical contraction. There are different ways to obtain those fiber directions. We are currently using data from a dissected canine heart available from the Bioengineering Research Group[2] of the University of Auckland, New Zealand and from reduced-encoding MR diffusion tensor imaging (dtMRI) [8].

In order to complete our anatomical model we also need data about the electrical network: the Purkinje network lo-

[2]http://www.bioeng.auckland.ac.nz/home/home.php

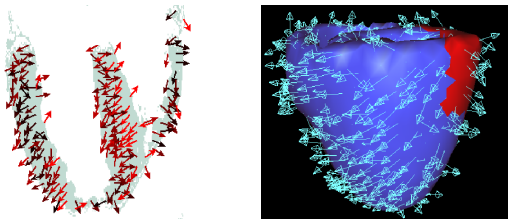

Fig. 3. dtMRI slice and tetrahedral myocardium mesh, with the fiber directions shown.

cations determine the electrical onset areas of the ventricular depolarization. But they are difficult to locate, both in dissection and cardiac images. In our model, they are currently approximated by a set of nodes near the apex.

4. ELECTRICAL MODEL

The electrical behavior of the heart, from the cell level to the global level of the muscle, has been extensively studied.

4.1. Cell Level

At the cell level, the main idea is to study the relationship between the transmembrane ionic currents and the ionic potentials inside and outside the cell. The models concerning this relation improve while the number of phenomena observed at the cell level increases [9]. At the beginning, we are only concerned with a model expected to account for the most important biological phenomena: a cell is activated only for a stimulus larger than a certain threshold; the shape of the action potential does not depend on the stimulus (it is only model-dependent); there is a refractory period during which the cell cannot be excited; a cell can act as a pacemaker.

A FitzHugh like model [10] seems to correctly capture these behaviors, and yields fast 3D computations. Here only the following set of differential equation is studied:

$$\begin{aligned} \partial_t u &= f(u) - z \\ \partial_t z &= \epsilon\,(ku - z) \end{aligned} \tag{1}$$

where $f(u) = u(1-u)(u-a)$. u is a normalized potential and z is a dynamic variable modeling the repolarization.

4.2. Whole Ventricle Level – Anisotropy

At the macroscopic scale, the ventricles are considered as a conducting continuum, where the local potentials are undergoing at the same time the diffusion and the reaction phenomena described by the models above.

Hence, (1) becomes:

$$\begin{aligned} \partial_t u &= \mathrm{div}\,(D\nabla(u)) + \lambda f(u) - z \\ \partial_t z &= \epsilon\,(ku - z). \end{aligned} \tag{2}$$

On a physiological point of view, these equations are understood either as a mathematical approximation of the dynamical system introduced by Hodgkin and Huxley [11],

as in [10], or as the result of some equilibrium equations that govern the conducting continuum, like in the so-called bidomain model [12].

The anisotropy of the ventricles is taken into account through the diffusion tensor D: $D = d_0.diag(1, \rho, \rho)$, in a local orthonormal basis $(\mathbf{i}, \mathbf{j}, \mathbf{k})$ where $\mathbf{i}$ is parallel to the fiber. d_0 is a scalar conductivity and ρ the anisotropy ratio between the transverse and the axial conductivities.

4.3. Results of the wave propagation

Simulated isochrones of activation are presented (Fig. 4), after a wave was initialized at the apex, using a crude approximation of the Purkinje network and a slightly anisotropic diffusion tensor.

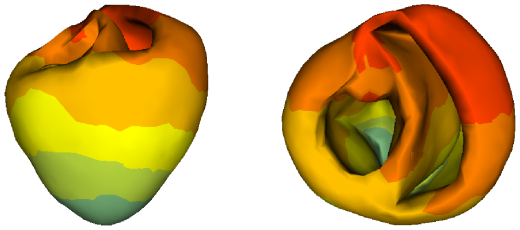

Fig. 4. Isochrones of activation (computed with a slightly anisotropic diffusion tensor: $\rho = 0.7$)

We can simulate different singularities that may correspond to pathologies by changing the conduction parameters (Fig. 5), for instance introducing a strong conductivity anisotropy.

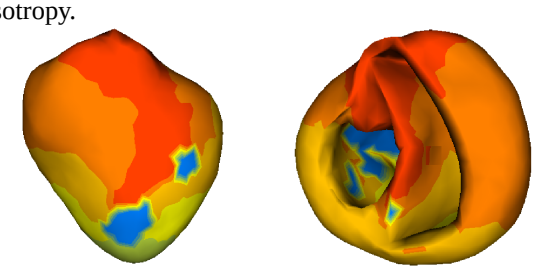

Fig. 5. Apparitions of activation singularities with a highly anisotropic diffusion tensor.

This time-dependent computed potential can then be used as an excitation entry to the system describing the mechanical behavior of the myocardium.

5. MECHANICAL MODEL

The myocardium is composed of muscle fiber bundles spiraling around the two ventricles. It is a nonlinear viscoelastic anisotropic active material. The qualitative behavior of the electromechanical coupling is a contraction for a positive action potential and an active relaxation for a negative one. Moreover, the action potential also modifies the stiffness of the material. The model introduced in [13, 14] by Bestel, Clément and Sorine captures this behavior. The global muscle model is based on the Hill-Maxwell rheological law which includes a contractile element E_c, a series element E_s and a parallel element E_p, as shown on Fig. 6 and is detailed in [15].

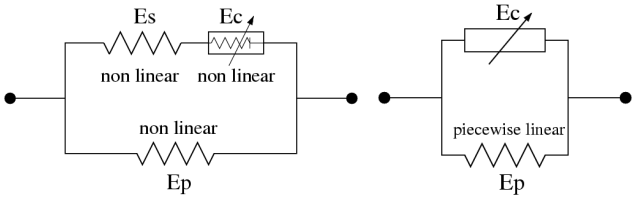

Fig. 6. (Left) Hill-Maxwell rheological model. (Right) Simplified rheological model.

In [14], Bestel, Clément and Sorine model the contractile element E_c, controlled by the action potential u, as follows:

$$\begin{cases} \frac{dk_c}{dt} &= -\left(|u| + \left|\frac{d\varepsilon}{dt}\right|\right) k_c + k_0 \left|u\right|_+ \\ \frac{d\sigma_c}{dt} &= -\left(|u| + \left|\frac{d\varepsilon}{dt}\right|\right) \sigma_c + k_c \frac{d\varepsilon}{dt} + \sigma_0 \left|u\right|_+ \end{cases}$$

with k_c the stiffness of the contractile element, u the action potential computed in part 4, ε the strain and σ_c the stress in the contractile element.

For computational efficiency, we simplified this physical model by using a piecewise-linear anisotropic material for the stiffness k, and only electrical command for contraction stress tensor σ_c (Fig. 6).

The simplified coupling equation only taking into account the electrical command writes $\partial_t \sigma_c = -|u|\,\sigma_c + \sigma_0 \left|u\right|_+$. With this simplified model, the contraction stress increases exponentially for positive action potential and decreases exponentially for negative ones, and the variation rate depends on the action potential value (Fig. 7).

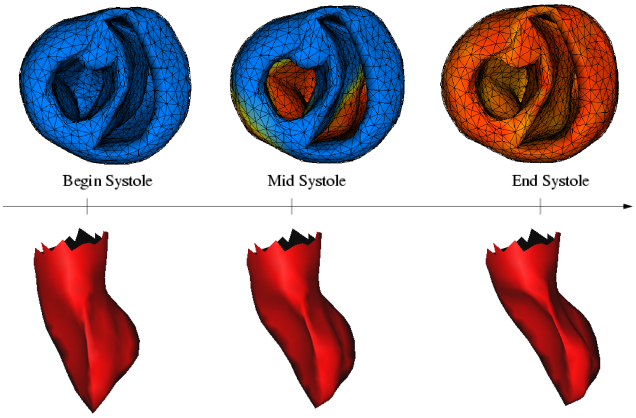

Fig. 7. Contraction of the fibers induced by the electromechanical coupling, and deformation of the left ventricle.

6. CONCLUSION AND PERSPECTIVES

We presented a model of the heart on which electrical wave propagation and mechanical contraction are computed. Currently, the total computation time for a full heart beat is

around 10 minutes whereas other existing electromechanical models take several hours of computation. We are investigating optimization and a parallel implementation to reduce the computation time to allow an interactive use of this model in time series of cardiac images.

The long term goal is to use this model to extract quantitative parameters of the ventricular function from cardiac images. We can also simulate electrical wave singularities that may correspond to pathologies or test different locations of electrical onset, useful for the implantation of pacemakers, for instance. Moreover, we will be able to simulate the influence of those electrical differences on the contraction. We believe that this beating heart model could help understand the consequences of local electrical or mechanical failures on the global motion. Additional images and videos are available on the web[3].

Globally, recent measurements of the electrical activity, fiber directions and motion reconstruction (from tagged MRI) on the same heart should help adjust the different parameters of the model [16]. We will work during the coming months on validating this method with an entire dataset coming from the same heart.

Finally, a more accurate model may be considered, and different numerical techniques exist: the complete simulation process can be improved, always keeping in mind the balance between the complexity of the models (more equations but more realistic) and the efficiency of the numerical methods (accuracy and speed).

7. ACKNOWLEDGEMENTS

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