Active strain and activation models in cardiac electromechanics

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We present a model for mechanical activation of the cardiac tissue depending on the evolution of the transmembrane electrical potential and certain gating/ionic variables that are available in most of electrophysiological descriptions of the cardiac membrane. The basic idea consists in adding to the chosen ionic model one ordinary differential equation for the kinetics of the mechanical activation function. A relevant example illustrates the desired properties of the proposed model, such as delayed muscle contraction and correct magnitude of the muscle fibers' shortening.

1 Introduction

The modeling of the heart is essential to better understand several pathologies arising from cardiovascular disorders that are known to be the leading cause of deaths in industrialized countries [3]. We focus our attention on two important elements in cardiac function: the electrophysiological phenomena driving the propagation of electrical potentials through the tissue, and its interaction with the large deformations of the myocardium.

2 The active strain formulation for the electromechanical coupling

In the active strain formulation, a decomposition of the deformation gradient is performed into a passive and an active contribution $\mathbf{F} = \mathbf{F}_p\mathbf{F}_a$ [1]. Moreover, it is assumed that the mechanical activation depends on the electrical potential through the function γ, which acts on the active factor of the deformation gradient defined as $\mathbf{F}_a = \mathbf{I} + \gamma \boldsymbol{n}\otimes\boldsymbol{n} + \gamma_s \boldsymbol{s}\otimes\boldsymbol{s}$, where $\boldsymbol{n}$ and $\boldsymbol{s}$ denote the local direction of the fibers and sheets, respectively (Fig. 1). Since the cardiac cells are composed mainly of water, we impose the incompressibility condition $\det(\mathbf{F}_a) = 1$ which implies in particular, that $\gamma_s = -\gamma/(1+\gamma)$. A material-dependent energy function $\mathcal{W}$ will determine the specific constitutive relations to use. In this note we restrict ourselves to Neo-Hookean materials. The effect of the mechanical deformation is included in the equations that drive the electrophysiology by rewriting the balances from Eulerian to Lagrangian coordinates. Let $\Omega \subset \mathbb{R}^N$, $N = 2, 3$ be a bounded body in its reference undeformed configuration, and let $\mathbf{D}$ denote a tensor of constant electrical conductivities. Putting all together, the modified equations accounting for the coupling between active finite elasticity and the monodomain equations read as follows: Find $v, \boldsymbol{u}, p$ (transmembrane potential, displacements, and pressure, respectively) such that

$$\begin{aligned} -\nabla\cdot\left(\frac{\partial\mathcal{W}(\boldsymbol{u},p)}{\partial\mathbf{F}}\right) &= 0 \quad \text{in } \Omega,\\ \det\mathbf{F} &= 1 \quad \text{in } \Omega,\\ \partial_t v - \nabla\cdot\left(\mathbf{F}^{-1}\mathbf{D}\mathbf{F}^{-T}\nabla v\right) &= I_{\text{ion}}(v,\boldsymbol{w}) \quad \text{in } \Omega\times(0,T),\\ \partial_t\boldsymbol{w} - H(v,\boldsymbol{w}) &= 0 \quad \text{in } \Omega\times(0,T),\\ \partial_t\gamma &= G(v,\boldsymbol{w},\gamma) \quad \text{in } \Omega\times(0,T). \end{aligned} \tag{2.1}$$

3 Activation models and numerical examples

The ODE system modeling the kinetics of the membrane and of the activation γ is given by

$$\partial_t v = I_{\text{ion}}(v,\boldsymbol{w}), \quad \partial_t\boldsymbol{w} = H(v,\boldsymbol{w}), \quad \partial_t\gamma = G(v,\boldsymbol{w},\gamma). \tag{3.1}$$

For phenomenological description of the membrane kinetics according to the Rogers-McCulloch model [6], system (3.1) reads

$$\partial_t v = -c_1 v(v-a)(v-1) + c_2 vw, \quad \partial_t w = b(v-w), \quad \partial_t\gamma = d_1^{RM}(\beta w - d_2^{RM}\gamma),$$

where the parameters are chosen as in Table 1. The parameter $\beta = 0.3$ is included to model the maximum change of length experimented by the cardiac fibers in a normal heartbeat. Fig. 2 depicts the time evolution for this system computed on a

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Table 1 Values of membrane and activation parameters.

$a = 0.13, b = 0.013, c_1 = 0.26, c_2 = 0.1, d_1^{RM} = 0.025, d_2^{RM} = 0.42, \beta = -0.3, d_1^{LR} = 2.2, d_2^{LR} = 0.005.$

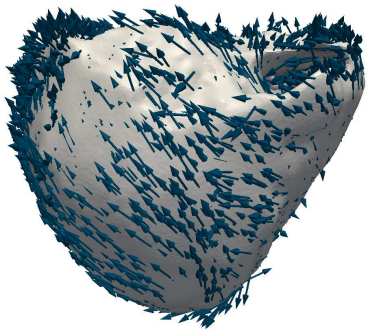

Fig. 1 Fibrous architecture of the cardiac tissue.

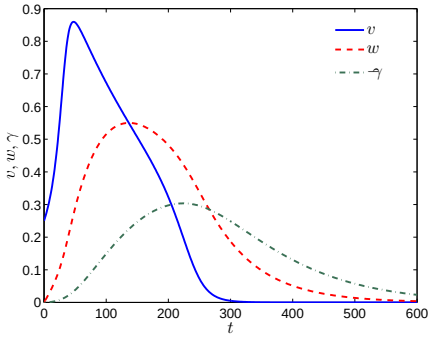

Fig. 2 Rogers-McCulloch model. Evolution of the action potential, recovery variable, and activation function in absolute value.

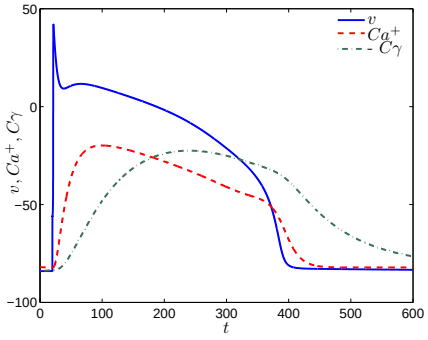

Fig. 3 Luo-Rudy model. Evolution of the action potential, rescaled calcium concentration, and rescaled activation function.

single cell. Analogously, in Fig. 3 we present the kinetics for the Luo-Rudy (phase I) model [2], which are obtained with the following specification for G in (3.1): $\partial_t \gamma = d_1^{LR}(\beta[Ca]_i^+ - d_2^{LR}\gamma)$. Here the amplitudes of both calcium and activation have been rescaled for visualization purposes. We see a qualitative accordance with the results provided in e.g. [5, Fig. 9]. Finally, in Fig. 4 we present a finite element simulation of (2.1) for a Neo-Hookean material with elastic modulus $\mu_1 = 4$. The computational meshes are a biventricular geometry consisting of 13'638 nodes forming 69'544 tetrahedra, and a truncated ellipsoid of 29'560 vertices and 155'770 elements. The time step is set to $\Delta t = 0.5\,\mathrm{ms}$ for the Rogers-McCulloch and $\Delta t = 0.05\,\mathrm{ms}$ for the Luo-Rudy examples. As in [4], Taylor-Hood finite elements are employed for the spatial discretization.

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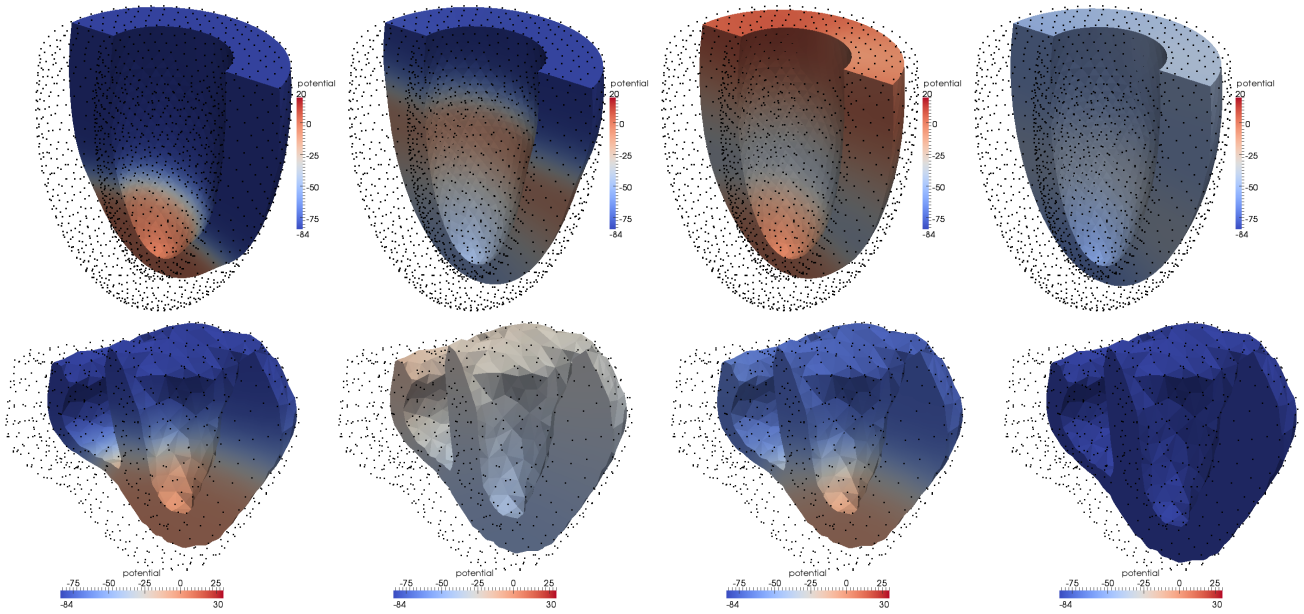

Fig. 4 Snapshots of the evolution of the transmembrane potential and corresponding slight movement of the mesh. Rogers-McCulloch Monodomain electro-mechanic model (top), and Luo-Rudy Monodomain electro-mechanic model (bottom) at time instants $t = 40, 180, 250, 400\,\mathrm{ms}$. In each snapshot, the undeformed uncutted domain is represented by a cloud of points.

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