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Thermodynamically consistent orthotropic activation model capturing ventricular systolic wall thickening in cardiac electromechanics

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ABSTRACT

The complex phenomena underlying mechanical contraction of cardiac cells and their influence in the dynamics of ventricular contraction are extremely important in understanding the overall function of the heart. In this paper we generalize previous contributions on the active strain formulation and propose a new model for the excitation-contraction coupling process. We derive an evolution equation for the active fiber contraction based on configurational forces, which is thermodynamically consistent. Geometrically, we link microscopic and macroscopic deformations giving rise to an orthotropic contraction mechanism that is able to represent physiologically correct thickening of the ventricular wall. A series of numerical tests highlights the importance of considering orthotropic mechanical activation in the heart and illustrates the main features of the proposed model.

1. Introduction

Cardiac muscle is highly heterogeneous and features an anisotropic and overall nonlinear behavior. A helical arrangement of families of co-aligned cardiomyocytes supported by an extracellular fibrous collagen network defines the local macroscopic structure of the tissue and features a complex passive response of the material. During systole the tissue activates and the cardiomyocytes contract. Mechano-chemical activation is mainly governed by the binding of calcium to troponin C, exposing binding sites for myosin on actin filaments. This triggers sarcomere contraction, which can be also modeled as a process that depends on the local strain and strain rate. Despite numerous emerging studies on cardiac contraction mechanisms ranging from experimental observations to theoretical formalisms and mechanistic explanations, the underlying multiscale and multiphysics phenomena governing the excitation-contraction coupling are still far from being fully understood. One often needs to limit the study to a specific sub-aspect of the entire process, compounding all remaining effects into simplified descriptions.

In this work, we focus on the mathematical modeling of active strain generation at cellular and organ levels. The upscaling strategy is incorporated in the model following an anisotropic active strain formalism (Nardinocchi and Teresi, 2007; Ruiz-Baier et al., 2013a), where the force balance determining the motion of the tissue depends on local distortion of the microstructure followed by a macroscopic rearrangement of the material recovering compatibility of the deformation. Mathematically, this corresponds to a decomposition of strains. Dissipative effects taking place during ventricular contraction are introduced assuming that the energy is a function of an auxiliary internal state variable, which represents the level of mechanical tissue activation. Then, from classical laws of thermodynamics we derive an evolution equation for the active strain, which also depends on local stretch and ionic concentrations. The same theoretical derivation can also be used to define an evolution law for the active stress tensor in usual active stress formulations. Similar thermodynamically consistent models to the one

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presented herein have been derived in (Sharifimajd and Stålhand, 2013; Stålhand et al., 2008, 2011) for smooth and skeletal muscle and in Ruiz-Baier et al. (2013b) for isolated cardiomyocytes. We present here a phenomenological description of the excitation-contraction coupling, but an extension to more physiologically detailed models (Murtada et al., 2010; Negroni and Lascano, 2008; Rice et al., 2008; Washio et al., 2012) is conceptually straightforward. Our interest is more oriented to the development of subcellular activation mechanisms tailored for the study of macroscopic cardiac electromechanics. Many studies have focused on descriptions of the contraction in the "mean fiber direction" considering the contraction of the tissue as transversely isotropic. We propose a simple model that links the microscopic and macroscopic deformations explaining cross-fiber shortening.

The validity of our new interpretation is assessed by simulation of the entire cardiac electromechanical function. The passive response of the material is modeled using the orthotropic Holzapfel-Ogden model (Göktepe et al., 2011; Holzapfel and Ogden, 2009), including fiber and sheetlet directional anisotropy, whereas the tissue electrophysiology is represented by the monodomain equations endowed with the minimal membrane model for human ventricular electrophysiology from Bueno-Orovio et al. (2008). We choose a staggered algorithm to describe the interaction between the electrophysiology and soft tissue mechanics. This allows us to follow the intrinsic differences in the time scales of both phenomena and is computationally less involved than the so-called monolithic schemes (where all subproblems are solved simultaneously) that are, on the other hand, more stable (Dal et al., 2013; Göktepe and Kuhl, 2010; Pathmanathan et al., 2010).

For the proposed activation model, the knowledge of the directions of anisotropy is essential. In many cases the fiber reconstruction by DT-MRI is usually too noisy to be used in simulations, as such (Nagler et al., 2013). For this reason, a rule-based fiber field is constructed instead. Here we follow the example in Wong and Kuhl (2013) and build sheetlet and fiber fields using simple geometrical and physiological assumptions.

This paper is organized as follows. Section 2 outlines the theoretical settings of the formulation. Starting from the generalized dissipation inequality for isothermal processes, we introduce the active strain formulation and show how to link microscopic and macroscopic deformations. We use some thermodynamical restrictions to build an evolution law for the active strains and we show how the same theoretical setting could be applied to the more common active stress formulation. A model for the macroscopic electromechanical coupling, along with algorithmic considerations is briefly presented in Section 3. Here we also detail the procedure used to construct the fiber and sheetlet fields. Numerical results are collected in Section 4, where we present four test cases to asses the validity of the proposed model. Special emphasis is placed on demonstrating that with the new orthotropic activation model the contraction pattern of the ventricle exhibits the correct physiological amount of wall thickening, torsion, and longitudinal shortening. Finally, in Section 5, we discuss the implications and limitations of our model.

2. Theoretical setting

2.1. Energy balance and dissipation inequality

Consider a continuum embedded in a region Ω_t, relative to the natural (unloaded and stress-free) configuration Ω. The global form of energy balance in Ω_t reads

$$\dot{\mathcal{U}} + \dot{\mathcal{K}} = \mathcal{Q} + \mathcal{P}_{\text{ext}},$$

where $\mathcal{U}$ is the internal energy, $\mathcal{K}$ the kinetic energy, and $\mathcal{Q}$ the heat transfer rate. The total external power $\mathcal{P}_{\text{ext}}$ is the sum of the of work rate done by body forces on the material volume Ω_t and the work rate done by the surface forces on its boundary $\partial\Omega_t$. The power balance states that $\mathcal{P}_{\text{ext}}$ is balanced by the sum of the internal power $\mathcal{P}_{\text{int}}$ and the rate of change in the kinetic energy $\dot{\mathcal{K}}$, that is

$$\dot{\mathcal{U}} = \mathcal{Q} + \mathcal{P}_{\text{int}}. \tag{1}$$

In general, $\mathcal{P}_{\text{int}}$ can be represented by the set $\{\Lambda_1,\ldots,\Lambda_m\}$ of intensive variables describing the local kinematics of the continuum corresponding to the set $\{\lambda_1,\ldots,\lambda_m\}$ of extensive thermodynamic tensions work conjugate with the rates $\{\dot{\Lambda}_1,\ldots,\dot{\Lambda}_m\}$, such that $\mathcal{P}_{\text{int}} = \int_{\Omega_t} \sum_{i=1}^{m} \lambda_i \cdot \dot{\Lambda}_i$. In classical continuum mechanics, in the natural configuration Ω, the intensive and extensive variables sets only contain the strain tensor $\mathbf{E}$ and the symmetric stress tensor $\mathbf{S}$ work conjugate with the strain rate $\dot{\mathbf{E}}$ in such a way that $\mathcal{P}_{\text{int}} = \int_{\Omega} \mathbf{S} : \dot{\mathbf{E}}$. For this reason, from now on, we will assume the total internal power to be the sum of the conventional internal power and other possible additional contributions

$$\mathcal{P}_{\text{int}} = \int_{\Omega} \mathbf{S} : \dot{\mathbf{E}} + \int_{\Omega} \sum_{i=1}^{m} \lambda_i \cdot \dot{\Lambda}_i.$$

Denoting with u the internal energy per unit of mass, r the heat supply, $\mathbf{q}$ the heat flux vector and T the temperature, the general material form of equation (1) reads

$$\frac{\mathrm{D}}{\mathrm{D}t} \int_{\Omega} \rho u = \int_{\partial\Omega} \mathbf{q} \cdot \boldsymbol{\nu} + \int_{\Omega} \rho r + \int_{\Omega} \mathbf{S} : \dot{\mathbf{E}} + \int_{\Omega} \sum_{i=1}^{m} \lambda_i \cdot \dot{\Lambda}_i,$$

where ρ is the density of the material in the reference configuration.

The dissipation inequality in global form reads

$$\dot{\mathcal{S}} \geq \mathcal{J}, \tag{2}$$

where $\mathcal{S}$ is the internal entropy and $\mathcal{J}$ the entropy flux. Introducing the entropy per unit of mass η, inequality (2) (in the reference configuration) becomes

$$\frac{\mathrm{D}}{\mathrm{D}t} \int_{\Omega} \rho\eta \geq - \int_{\partial\Omega} \frac{\mathbf{q}}{T} \cdot \boldsymbol{\nu} + \int_{\Omega} \rho \frac{r}{T}.$$

In local form then the first and second law of thermodynamics in material coordinates are, respectively (Coleman and Noll, 1963; Epstein, 2012),

$$\rho\dot{u} = \mathbf{S} : \dot{\mathbf{E}} + \sum_{i=1}^{m} \lambda_i \cdot \dot{\Lambda}_i + \rho r - \nabla \cdot \mathbf{q}, \tag{3}$$

$$\rho\dot{\eta} \geq \rho \frac{r}{T} - \nabla \cdot \left(\frac{\mathbf{q}}{T}\right), \tag{4}$$

where gradients and divergences are operated with respect to the coordinates $\mathbf{X}$ in the reference configuration. It will be useful to consider the stress power $\mathbf{S} : \dot{\mathbf{E}}$ to be given in the form $\mathbf{P} : \dot{\mathbf{F}}$, where $\mathbf{P}$ is the mixed (two-point) first Piola-Kirchhoff stress tensor, conjugate with the rate of the deformation gradient tensor $\dot{\mathbf{F}}$ (see a summary of used notation in Table 1).

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