Commun. Comput. Phys.
doi: 10.4208/cicp.OA-2018-0253

Vol. **27**, No. 1, pp. 87-115
January 2020

Modelling Thermo-Electro-Mechanical Effects in Orthotropic Cardiac Tissue

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Received 25 September 2018; Accepted (in revised version) 15 January 2019

Abstract. In this paper we introduce a new mathematical model for the active contraction of cardiac muscle, featuring different thermo-electric and nonlinear conductivity properties. The passive hyperelastic response of the tissue is described by an orthotropic exponential model, whereas the ionic activity dictates active contraction incorporated through the concept of orthotropic active strain. We use a fully incompressible formulation, and the generated strain modifies directly the conductivity mechanisms in the medium through the pull-back transformation. We also investigate the influence of thermo-electric effects in the onset of multiphysics emergent spatiotemporal dynamics, using nonlinear diffusion. It turns out that these ingredients have a key role in reproducing pathological chaotic dynamics such as ventricular fibrillation during inflammatory events, for instance. The specific structure of the governing equations suggests to cast the problem in mixed-primal form and we write it in terms of Kirchhoff stress, displacements, solid pressure, dimensionless electric potential, activation generation, and ionic variables. We also advance a new mixed-primal finite element method for its numerical approximation, and we use it to explore the properties of the model and to assess the importance of coupling terms, by means of a few computational experiments in 3D.

AMS subject classifications: 92C10, 74S05, 65M60, 74F25

Key words: Cardiac electromechanics, orthotropic active strain, thermo-electric coupling, scroll wave propagation, numerical simulations.

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1 Introduction

Temperature variations may have a direct impact on many of the fundamental mechanisms in the cardiac function [74]. Substantial differences have been reported in the conduction velocity and spiral drift of chaotic electric potential propagation in a number of modelling and computationally-oriented studies [25], and several experimental tests confirm that this is the case not only for cardiac tissue, but for other excitable systems [28, 40]. The phenomenon is however not restricted to electrochemical interactions, but it also might affect mechanical properties [33, 45, 68]. Indeed, cardiac muscle is quite sensitive to mechanical stimulation and deformation patterns can be very susceptive to external agents such as temperature. For instance, enhanced tissue heterogeneities can be observed when the medium is exposed to altered thermal states, and in turn these can give rise to irregular mechano-chemical dynamics. A few examples that relate to experimental observations from epicardial and endocardial activity on canine right ventricles at different temperatures, as well as tachycardia and other fibrillation mechanisms occurring due to thermal unbalance, can be found in e.g. [25]. These scenarios can be related to extreme conditions encountered during heat strokes and sports-induced fatigue (easily reaching 41°C), and localisation of other thermal sources such as ablation devices; but also to surgery or therapeutical procedures (in open-chest surgery tissues might be exposed to cold air in the operating theatre at 25°C), or due to extended periods of exposure to even lower temperatures that can occur during shipwrecks or avalanches. It is not striking that temperature effects might affect the behaviour of normal electromechanical heart activity. However the precise form that these mechanisms manifest themselves is not at all obvious. This is, in part, a consequence of the nonlinear character of the thermo-electro-mechanical coupling. For instance, one can show that localised thermal gradients might destabilise the expected propagation of the electric wave, as well as change the mechanical behaviour of anisotropic contraction. Our goal is to investigate the role of the aforementioned effects in the development and sustainability of cardiac arrhythmias. These complex emerging phenomena originate from multifactorial and multiphysical interactions [57], and they are responsible for a large number of cases of pathological dysfunction and casualties. The model we propose here has potential therefore in the investigation of mechanisms provoking such complex dynamics, in particular those arising during atrial and ventricular fibrillation.

Even if computational models for the electromechanics of the heart are increasingly complex and account for many multiphysics and multiscale effects (see e.g. [11, 58, 70]), we are only aware of one recent study [10] that addresses similar questions to the ones analysed here. However that study is restricted to one-dimensional domains, it uses the two-variable model from [51], and it assumes an active stress approach for a simplified neo-Hookean material in the absence of an explicit stretch state. Our phenomenological framework also uses a minimal temperature-based two-variable model, but in contrast, it additionally includes a nonlinear conductivity representing a generalised diffusion mechanism intrinsic to porous-medium electrophysiology [36]. We postulate then an