11th World Congress on Computational Mechanics (WCCM XI)
5th European Conference on Computational Mechanics (ECCM V)
6th European Conference on Computational Fluid Dynamics (ECFD VI)
July 20–25, 2014, Barcelona, Spain

A CONTINUUM MODEL FOR ACTIVE CARDIAC MUSCLE

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Key words: *Cardiac muscle, contraction, thermodynamics, visco-elasticity*

Dynamic models of cardiac muscle contraction are commonly derived by an additive decomposition of the stress tensor, giving the total stress $\boldsymbol{\sigma}$ as the sum of an active and a passive part,

$$\boldsymbol{\sigma} = \boldsymbol{\sigma}_a + \boldsymbol{\sigma}_p.$$

The passive contribution σ_p is normally derived in a continuum framework, and assuming a hyper-elastic stress-strain response. The active part σ_a results from mechano-chemical reactions in the cells, which are typically modeled in a single-cell framework where tension is a function of numerous dynamic state variables, see for instance [2]. The active tension is converted to a three dimensional stress tensor to be applied in a continuum setting. This modeling framework is commonly referred to as the active stress approach.

An alternative model for coupled active and passive tissue mechanics is obtained by introducing the notion of an active strain or active deformation, [1]. This leads to a multiplicative decomposition of the deformation gradient,

$$\boldsymbol{F} = \boldsymbol{F}_e \boldsymbol{F}_a, \tag{1}$$

where $\boldsymbol{F}$ represents the total (visible) deformation, $\boldsymbol{F}_a$ the active deformation, and $\boldsymbol{F}_e$ the elastic deformation. In the active strain formulation, the total stress in the tissue is equal to the passive elastic stress.

We present a modeling framework that differs from these assumptions in two ways. First, we extend the standard hyper-elastic models for the passive tissue response to consider visco-elastic effects, that give a better match with experimental data. Second, we adapt a mechano-chemical by Stålhand et al [3, 4], originally derived for smooth muscle, to describe the active-passive mechanical coupling in cardiac tissue.

We use (1) to introduce two right Cauchy-Green tensors, $\boldsymbol{C} = \boldsymbol{F}^T\boldsymbol{F}$ describing total deformation, and $\boldsymbol{C}_e = \boldsymbol{F}_e^T\boldsymbol{F}_e$ describing the elastic deformation. Introducing a (pseudo)

strain energy function Ψ, and following the steps outlined in [4], we get the following expression for the first Piola-Kirchoff stress tensor.

$$\boldsymbol{P} = 2\boldsymbol{F}\frac{\partial \Psi}{\partial \boldsymbol{C}} + 2\boldsymbol{F}\boldsymbol{F}_a^{-1}\frac{\partial \Psi}{\partial \boldsymbol{C}_e}\boldsymbol{F}_a^{-T}.$$

We proceed to assume an additive split of the strain energy, $\Psi = \Psi_1 + \Psi_2$, with

$$\Psi_1 = \Psi_{1,vol}(J) + \Psi_{1,iso}(\overline{\boldsymbol{C}}, \boldsymbol{f}_0, \boldsymbol{s}_0, \boldsymbol{n}_0) + \sum_{s=1}^{n} \Upsilon_s(\overline{\boldsymbol{C}}, \boldsymbol{\Gamma}_s)$$

and

$$\Psi_2 = \mathcal{N}(\lambda_a)\Psi_2(\boldsymbol{C}_e, \boldsymbol{f}_0, \boldsymbol{\alpha}) + \Psi_3(\boldsymbol{\alpha}) + \Psi_4(\beta).$$

Here, $\boldsymbol{f}_0, \boldsymbol{s}_0, \boldsymbol{n}_0$ are unit vectors aligned with the local structure of the tissue. We have decomposed the deformation into isochoric and volumetric parts by introducing the tensor $\overline{\boldsymbol{C}} = J^{-\frac{2}{3}}\boldsymbol{C}$, with $J = \det \boldsymbol{F}$, and assumed that the energy Ψ_1 associated with passive deformations can be decomposed into a volumetric part $\Psi_{1,vol}$, which behaves in a purely elastic manner, an elastic isovolumic contribution $\Psi_{1,iso}$ and a set of time dependent terms $\Upsilon_s(\overline{\boldsymbol{C}}, \boldsymbol{\Gamma}_s), s = 1, \ldots, n$. The energy Ψ_2 is associated with active deformation of the tissue, and is a function of the mechano-chemical states of the cardiac cells, represented by states $\boldsymbol{\alpha}$ and β, see for instance [4] for details.

In the presentation we will specify the terms in the strain energy functions, and compare model results to experimental data and existing models from the litterature, such as [2].

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