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# MULTIPHYSICS SIMULATION OF HEART MUSCLE ELECTROMECHANICS

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## ABSTRACT

The progress of computing technology has enabled the development of sophisticated computational models of human organs to simulate various physiological conditions. Cardiac modelling can be applied to simulate invasive treatments and predict outcomes for various disease conditions. Cardiac function involves multiple physics interactions, such that electrical activation triggers mechanical contraction to eject the blood. Hence, disruption of electrical activity in the heart, such as in left bundle branch block (LBBB), can cause disruption to the heart contraction sequence. To restore contraction function, cardiac resynchronization therapy (CRT), which directly stimulates the late activate region, is often prescribed. However, clinical trials reveal that over 30% patients do not respond to this treatment. As such, heart modelling emerged as potential avenue to optimize such treatment. Over the past decade, heart modelling had matured to a multiphysics finite element problem, where electrical activity is coupled to trigger tissue mechanical deformation. In this article, we will provide a brief explanation of the methodology, using a simple cube, suitable for many beginners in the field. The cube model can be used as a benchmarking tool to test early framework development before proceeding with complex model.

## KEYWORD

Heart muscle, electromechanics, action potential, myocardial mechanics.

## INTRODUCTION

Since the nature of cardiac function involves multiple physics interactions, it is crucial to model all these physics together to ensure the coupled effect is captured by the model. A multiphysics model also enables a wide variety of potential model expansions. By incorporating sub-cellular phenomena, several electromechanics models of the heart have been successfully developed to simulate multiphysics interactions (Gőktepe and Kuhl, 2010; Bakir et al., 2018). As our understanding of cardiac electrophysiology and excitation-contraction coupling has greatly improved, mathematical models have become increasingly accurate, but far more complicated. The result of the experiments allows the physiological phenomena to be translated into usable differential equations, enabling it to be incorporated into finite element methods.

Mechanical activity of the heart is initiated by a sequence of electrical events within cardiomyocytes known as action potentials in which the cell membranes depolarise for a certain period and repolarise back due to the flux of ions into and out of the cells through various ion channels. This causes a momentary transient in membrane potential, as shown in Figure 1. The action potential triggers mechanical contraction of the cell, whilst simultaneously propagates across the entire heart tissues. (Guyton and Hall, 2006). The muscle cell contracts in a certain sequence, causing the entire organ to pump out the blood in the chambers, before subsequently relax and blood refills for the following cycle.

The micro-structure of mammalian heart is an important determinant of its unique kinematics. The cardiac muscle fibres are arranged in a helical fashion (Figure 1 (Right)) with the orientation angle varying depending on the transmural depth within the myocardium. The cardiomyocytes are also organised in sheet-like structures, making it essentially an orthotropic structure. These unique orientations yield distinct properties where the fibre direction is stiffer than the sheet direction and sheet-normal. The tissue electrical properties are also exhibiting similar orthotropic

nature where the fibre is the most conductive, followed by the sheet direction and normal-to-sheet. All these properties must be considered when modelling the muscle.

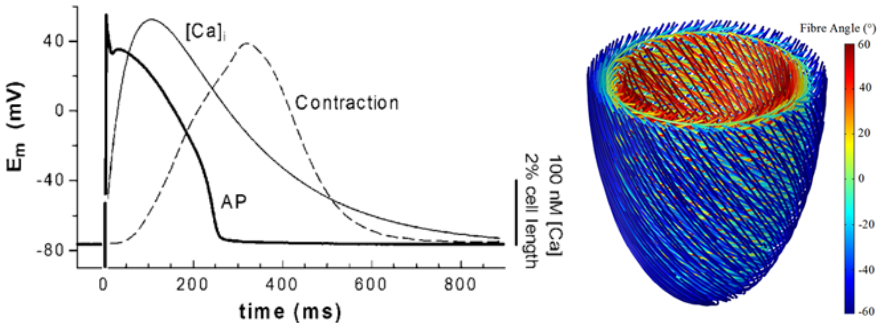


Figure 1: (Left) Plot of action potential (AP), calcium transient ([Cai])and active contraction profiles of rabbit cardiomyocyte (Rice et al., 2008). (Right) Simulated cardiac muscle fibre (Bakir et al. 2018).

**MATERIAL AND METHODOLOGY**

A 3-dimensional 10x10x1 cm rectangular myocardial block structure was used to test the electromechanics framework. The block in Figure 2 was embedded with fibre direction aligned-45 degrees relative to the y-axis as shown in Figure 2. The block was held fixed by a spring constraint at its rectangular edges with spring constant of 1e-3 N/mm. The model face that aligns with the yz-plane at y = 0 was set to be fixed along its x-axis but allowed to deform within yz-plane. An electrical stimulus was applied at the centre domain of the block.

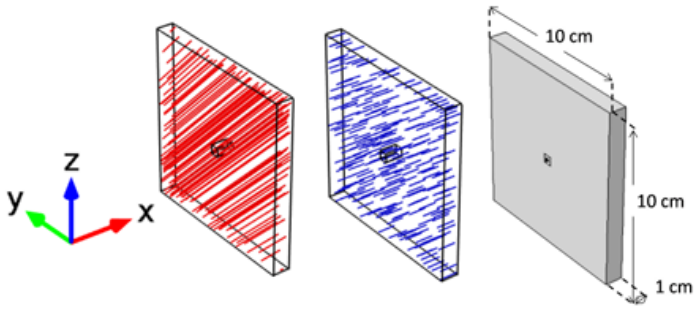


Figure 2: The rectangular myocardial block setup for electromechanical testing showing the fibre (left) and the sheet (middle) orientations. The block dimensions are given in the figure panel at right, with the centre domain being the stimulus site.

Myocardial active electrical properties were based on the Nash and Panfilov (2004) for mulations, modified to incorporate appropriate units and dimensions into the originally dimensionless formulations

$$\beta\left(C_m\frac{\partial V_m}{\partial t}+k_1k_2(V_m-B)\left(\left[\frac{V_m-B}{A}\right]-a\right)(V_m-1)+k_2R(V_m-B)\right)=\nabla\cdot(\sigma\nabla V_m) \quad (1)$$

$$\frac{\partial R}{\partial t}=\left(\varepsilon_0+\frac{\mu_1R}{\left[\frac{V_m-B}{A}\right]+\mu_2}\right)\left(-R-k_1\frac{V_m-B}{A}\left(\frac{V_m-B}{A}-a-1\right)\right) \quad (2)$$

where $V_m$ represents myocardial membrane potential and $R$ is an auxiliary recovery variable. Eq. (1) governs the membrane potential dynamics whilst Eq. (2) controls the relaxation dynamics within the myocardium. The details on the parameters are available in the original work (Bakir et al. 2018). The electrical conductivity tensor, σ, was set to exhibit an anisotropy ratio of 4:2:1 along the fibre, $\hat{F}$, sheet, $\vec{S}$, and sheet normal, $\vec{N}$ directions respectively, in accordance with Hooks et al. (2007). This tensor, **σ**, was determined from scalar conductivities defined in the local fibre ($\sigma_f$), sheet ($\sigma_s$) and normal-to-sheet ($\sigma_n$) according to Eq. (3):

$$\boldsymbol{\sigma} = \sigma_f\left(\hat{F} \otimes \hat{F}\right) + \sigma_S\left(\hat{S} \otimes \hat{S}\right) + \sigma_N\left(\hat{N} \otimes \hat{N}\right) \tag{3}$$

The simplified formulation of excitation-contraction, proposed by Nash and Panfilov (2004) modified by G¨oktepe and Kuhl (2010), was chosen to generate active stress triggered by the action potential. Excitation-contraction was represented by Eqs. (4) and (5).

$$\frac{\partial T_a}{\partial t} = \epsilon(V_m)(k_{Ta}(V_m - B/A) - T_a) \tag{4}$$

$$\epsilon(V_m) = \epsilon_0 + (\epsilon_\infty - \epsilon_0)\exp\left(-\exp\left(-\xi(\mathrm{V_m} - V_{threshold})\right)\right) \tag{5}$$

where Ta is the active stress and the fixed parameters are listed in (Bakir et al. 2018). The myocardium was represented by the transverse isotropic form of the hyperelastic formulation of Holzapfel and Ogden (2009), as detailed in Eq. 6.

$$\psi = \frac{a_i}{2b_i}\exp(b_i(I_1 - 3) + \frac{a_f}{2b_f}\exp\left(b_f\left(I_{4f} - 1\right)^2 - 1\right) + \frac{\kappa(J-1)\ln J}{2} \tag{6}$$

where $I_i$ denotes the first invariant of the isochoric elastic right Cauchy Green tensor, **C**, whilst $I_{4f} = \hat{F} \cdot (\mathbf{C}\,\hat{F})$. J represents the determinant of the deformation gradient tensor, **F**. To ensure material stability, $I_{4f}$ was set to zero when $I_{4f} < 0$, to account for the assumption that myocardial fibres do not contribute significantly to passive mechanics during compression (Holzapfel and Ogden, 2009).

**RESULT AND DISCUSSION**

The electromechanical framework with Holzapfel and Ogden (2009) material, was tested in a simple rectangular block to observe whether active contraction can be achieved. The snapshots of the deformation are shown in Figure 3.

With the stimulus initiated at the centre of the block, the activation spread in an elliptical shape along the fibre direction since it was the most conductive direction. As the action potential spread out, the block can be seen contracting. The contraction occurred first along the fibre direction, as shown in the snapshots in Figure 3. As the fibre direction shortened, the transverse direction elongated. The block relaxed, and returned to its original shape as the electrical activation ceased. Since the boundary was fixed along the x-axis at the frontal face of the yz-planein-plane motion was only observed at the back face of the yz-plane. This simulation indicates the success of the electromechanical coupling between the electrophysiology model and the myocardial mechanics model. The propagation also followed the anisotropic properties defined earlier in the conductivity tensor. The model can also be used to test various case conditions, emulating, strands of muscle. Some examples include simulating arrhythmic wave (Bakir et al. 2015) as well as a testbed to test new formulation where integration with myocardial perfusion model can be performed (Delestri et al. 2023).

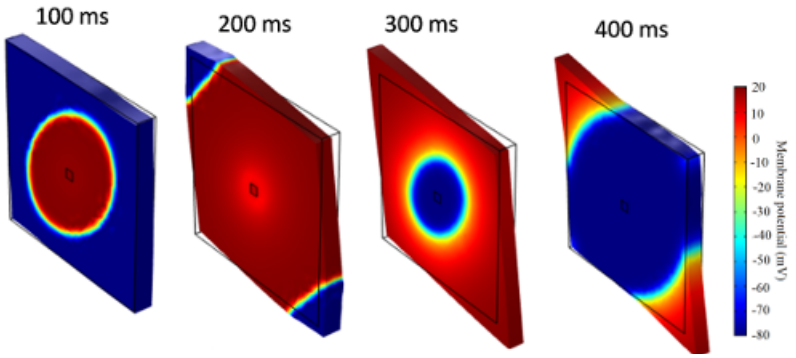

Figure 3: Rectangular myocardial block deformation following applied electrical stimulus at the centre of the domain. The black outlines indicate the original undeformed geometry.

## CONCLUSION

The work presented here describes the basic approach in modelling cardiac muscle electromechanical phenomena. As shown in the results, it can be seen that the model is capable to simulate cardiac muscle contraction. Using similar formulations, this can be easily translated to complex realistic geometry to simulate more realistic heart mechanics.

## ACKNOWLEDGEMENT

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