NUMERICAL MODELLING AND ANALYSIS OF THE LEFT VENTRICLE

GAUTE A. SLINDE*, VICTORIEN PROT* AND BJØRN SKALLERUD*

*Biomechanics Division, Department of Structural Engineering
Norwegian University of Science and Technology
NTNU, NO-7491 Trondheim, Norway
e-mail: `gaute.a.slinde@ntnu.no`

Key words: Left Ventricle, Finite Element Analysis, Electromechanical coupling

Summary. The development of a well functioning finite element model of the left ventricle is an important step towards a better understanding of the pumping function of the human heart. The current work implements a passive material model with an active contraction. The complex structure of the muscle fibers is included both with and without regional variations of the fiber distribution. Further an electromechanical coupling is implemented governing the active muscle contraction.

1 INTRODUCTION

Heart disease is on of the primary causes of death in the world today. There is a hope that advances made within biomechanics and a deeper knowledge of the structural and mechanical functions of the heart may lead to a better understanding of different diseases and enable us to develop more efficient treatments. In the current work the systolic contraction of the left ventricle is modelled using a hyperelastic, orthotropic constitutive model for the passive myocardium and a simple model for the active stress component. This is extended to include an electromechanical activation model. The influence of the distribution of the muscle fiber field is investigated. The work presented is ongoing with a goal of establishing a robust model for the left ventricle to enable further work in to pasient specific clinical problems.

2 METHODS

The systolic contraction is modelled using an additive decomposition of the Cauchy stress tensor $\boldsymbol{\sigma} = \boldsymbol{\sigma_p} + \boldsymbol{\sigma_a}$, where $\boldsymbol{\sigma_p}$ is the passive part and $\boldsymbol{\sigma_a}$ is the active part.

The passive part representing the inactive muscles response to a load is captured using

the Holzapfel-Ogden constitutive law[1],

$$\Psi(\bar{I}_1, \bar{I}_{4_f}, \bar{I}_{4_s}, \bar{I}_{8_{fs}}) = \frac{a}{2b}(\exp[b(\bar{I}_1 - 3)] - 1) + \sum_{i=f,s} \frac{a_i}{2b_i}(\exp[b_i(\bar{I}_{4i} - 1)^2] - 1)$$
$$+ \frac{a_{fs}}{2b_{fs}}(\exp[b_{fs}\bar{I}_{8_{fs}}^2] - 1) + \kappa(J - 1)^2, \tag{1}$$

where κ is the positive penalty parameter governing the volumetric change enforcing the incompressible nature of the myocardium. We perform a multiplicative decomposition of $\mathbf{F}$ into volume-changing and volume-conserving parts, $\mathbf{F} = (J^{1/3}\mathbf{1})\bar{\mathbf{F}}$ and $\mathbf{C} = (J^{2/3}\mathbf{1})\bar{\mathbf{C}}$. The modified left Cauchy-Green tensor is defined by $\bar{\mathbf{B}} = \bar{\mathbf{F}}\bar{\mathbf{F}}^T$. We also define the following vectors, $\bar{\mathbf{f}} = \bar{\mathbf{F}}\mathbf{f}_0$ and $\bar{\mathbf{s}} = \bar{\mathbf{F}}\mathbf{s}_0$, the fiber and sheet vectors in the deformed state, corresponding to the push-forward of $\mathbf{f}_0$ and $\mathbf{s}_0$ through the volume preserving part of the deformation gradient. The second Piola-Kirchoff stress $\mathbf{S}$ on decoupled form, is derived from Ψ through $\mathbf{S} = 2\frac{\partial\Psi}{\partial\mathbf{C}} = \mathbf{S}_{vol} + \mathbf{S}_{iso}$, in the reference configuration. The Cauchy stress tensor $\boldsymbol{\sigma}$ is then obtained by the push-forward operation of $\mathbf{S}$ to the configuration $\boldsymbol{\sigma} = \frac{1}{J}\mathbf{F}\mathbf{S}\mathbf{F}^T$, giving

$$\boldsymbol{\sigma} = 2\kappa(J - 1)\mathbf{1} + \frac{1}{J}dev\ \bar{\boldsymbol{\sigma}},$$
$$\bar{\boldsymbol{\sigma}} = 2\bar{\psi}_1\bar{\mathbf{B}} + 2\bar{\psi}_{4_f}\bar{\mathbf{f}} \otimes \bar{\mathbf{f}} + 2\bar{\psi}_{4_s}\bar{\mathbf{s}} \otimes \bar{\mathbf{s}} + \bar{\psi}_{8_{fs}}(\bar{\mathbf{f}} \otimes \bar{\mathbf{s}} + \bar{\mathbf{s}} \otimes \bar{\mathbf{f}}), \tag{2}$$

where $dev\ [\bullet] = (I - \frac{1}{3}\mathbf{1} \otimes \mathbf{1}) : (\bullet)$ and $\bar{\psi}_i = \frac{\partial\Psi}{\partial\bar{I}_i}, i = 1, 4_f, 4_s, 8_{fs}$.

The active part of the Cauchy stress tensor $\boldsymbol{\sigma_a}$ has the components in the (ff), (nn) and (sn) directions to obtain a correct deformation pattern during systole.[2] Initially, before expanding to an electromechanical coupling, the active component of the second Piola-Kirchoff stress tensor S_a is given as a linear increasing function with maximum value coinciding with the maximum endocardial pressure. When using the electromechanical coupling the evolution of the active Cauchy stress is dependent on the transmembrane potential through

$$\dot{\sigma}_a = \epsilon\ [k_\sigma(\Phi - \Phi_r) - \sigma_a]. \tag{3}$$

The electrical source is here governed by the phenomenological Aliev-Panfilov model which in a non-dimensional space has the constitutive equations[3]

$$\hat{f}_e^\phi = c\phi(\phi - \alpha)(1 - \phi), \tag{4}$$

$$\hat{f}_m^\phi = \vartheta G_s(\bar{\lambda} - 1)(\phi - \phi_s)\ \text{and} \tag{5}$$

$$\hat{f}^r = [\gamma + \frac{\mu_1 r}{\mu_2 + \phi}][-r - c\phi(\phi - b - 1)]. \tag{6}$$

The muscle fibers are implemented first with a homogenous field with transmural varying fiber angles with a maximum amplitude of 45°, 60°, 70°. Further a fiber field with different distribution for the different regions of the left ventricle is investigated.[4]
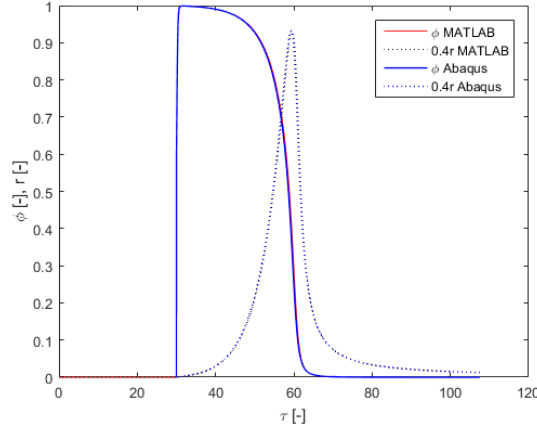
Figure 1: Comparison of the Aliev-Panfilov model implemented in MATLAB and Abaqus, with normalized time and non-dimensional action potential.

3 RESULTS

The different fiber distributions yield a different global deformation during contraction. Regarding the torsion the model gives approximately 27°, 32° and 17° for a max fiber angle of 45°, 60° and 70°, respectively. The ejection fraction is found to be 31 %, 29 % and 29 % for the same fiber angles, respectively. For the inhomogeneous fiber field the model finds 25° torsion. This is somewhat different from the physiological values of approximately 10° torsion and 60-70 % ejection fraction.

Nodes on the basal surface is chosen and given an elevated initial action potential of -10 mV, representing the sinus node and the starting point of the contraction. The potential spreads through the the ventricle as shown in Figure 2 obtain a full contraction at 170ms. Since the model does not include the electrical conduction system, the electrical potential follows only the anisotropic conduction of the myocardium with higher conduction in the fiber direction. The model correctly shows a shorter activation period for muscle cells activated later in systole with the first cells to be activated be the last to be deactivated, as shown for t=225ms.

4 CONCLUSIONS

In general it is found that the model overestimates the torsion and underestimates the ejection fraction compared to what is physiological correct. The different responses for the different implemented fiber orientations speaks to the importance of a correct and realistic fiber field.

The phenomenological electro-mechanical model implemented is shown to give promising results, with a correct evolution of the electrical field. It has benefits being a relatively simple model thus not adding more complexity and uncertainty to the model.
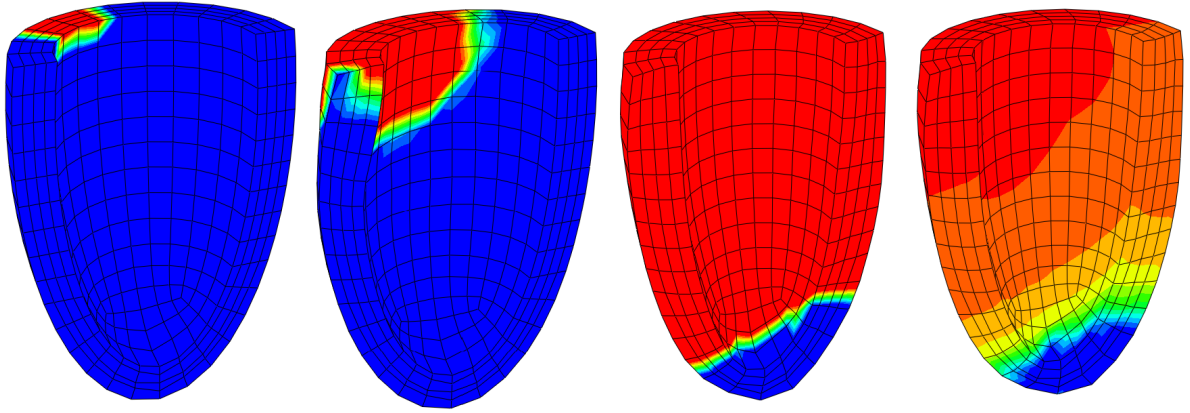
Figure 2: Evolution of the action potential after 0 ms, 50 ms, 150 ms and 225 ms, from left to right, respectively.

REFERENCES

[1] Holzapfel, G. A. & Ogden, R. W. Constitutive modelling of passive myocardium: a structurally based framework for material characterization. *Philosophical Transactions of the Royal Society of London A: Mathematical, Physical and Engineering Sciences* **367**, 3445–3475 (2009).

[2] Dorri, F., Niederer, P. F. & Lunkenheimer, P. P. A finite element model of the human left ventricular systole. *Computer Methods in Biomechanics and Biomedical Engineering* **9**, 319–341 (2006).

[3] Göktepe, S. & Kuhl, E. Electromechanics of the heart: a unified approach to the strongly coupled excitation–contraction problem. *Computational Mechanics* **45**, 227 – 243 (2010). URL `http://search.ebscohost.com/login.aspx?direct=true&db=a9h&AN=45391132&site=ehost-live`.

[4] Rohmer, D., Sitek, A. & Gullberg, G. T. Reconstruction and visualization of fiber and laminar structure in the normal human heart from ex vivo diffusion tensor magnetic resonance imaging (dtmri) data. *Investigative Radiology* **42**, 777–789 (2007).