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Electromechanical Modeling of the Heart in Healthy and Pulmonary Arterial Hypertension Cases

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Abstract

Cardiovascular diseases are the leading cause of death worldwide. Computer simulations of cardiac function are gradually becoming a powerful tool to better understand cardiac behavior and support clinical decision-making. However, right heart modeling is still in its early stages. Pulmonary hypertension is a pathophysiological disorder that may involve several clinical conditions. This study presents a fully coupled multiscale mathematical and numerical model of cardiac electromechanics with idealized biventricular geometry for normal subjects and pulmonary hypertension patients in COMSOL software. Muscle fibers that control electrical conduction and myocardial contraction, is one of the vital factors for the electromechanical simulation of the heart which was defined using a rule-based method. Windkessel blood circulation model was used to calculate systemic and pulmonary pressures. Blood circulation was modeled with a straightforward approach for the left and right ventricles. Lastly, alterations in the functional behavior of the heart were evaluated between individuals without pulmonary hypertension and healthy subjects. It was observed that right ventricular pressure increases in pulmonary arterial hypertension and ejection fraction decreases.

Keywords: Cardiac Electromechanics, Finite Element Method, Pulmonary Arterial Hypertension, Right Ventricle, Biomechanics.

1. Introduction

Of the 17 million premature deaths due to noncommunicable diseases in 2019, 38% were due to cardiovascular diseases [1]. Pulmonary arterial hypertension (PAH) is a disease characterized by vasoconstriction of the pulmonary arteries, leading to increased pulmonary arterial pressure. Without treatment, PAH can rapidly lead to irreversible right heart failure and death. PAH is a debilitating disease, with a 1-year mortality rate of approximately 15% with modern treatment and a 3-year survival rate of less than 67% [2]. Xi et al. [3] investigated changes in ventricular wall curvature in PAH in a full-beat simulation. The mechanisms by which growth was modeled have not been fully validated, and their dependence on time and pressure alone does not explain the physiological changes that occur during PAH. To provide some mechanistic insight, Kheyfets et al. [4] used a realistic finite element model of the pediatric heart to elucidate the inverse relationship between left ventricular torsion, cardiac apex rotation, and right ventricular remodeling in PAH. They observed that changes in left ventricular mechanics due to right ventricular adaptation in PAH had minimal impact on left ventricular ejection fraction. These observations are in contrast to the study of Xi et al. [3] who stated that PAH can lead to left ventricular remodeling as well as a significant reduction in ejection fraction for both the left and right ventricles.

In this study, an idealized biventricular geometry is utilized in which myocardial microstructure is defined using a rule-based method. Functional changes associated with pulmonary hypertension are modeled using electromechanical physics in COMSOL software. Ventricular circulatory is modeled using a straightforward approach for the left and right ventricles that does not require the use of complex conditional constraints. The model results have been validated against previous studies.

2. Methods

The biventricular geometry was created using COMSOL built-in geometry tools by cutting two concentric ellipses in half, in accordance with Göktepe et al. [5]. Similar to the work of Bekir [6], a small cylindrical region of 1 cm diameter was defined at the apex to allow for isotropic assumption at that region. Furthermore, the distance between epicardial and endocardial walls was calculated and normalized, and the normalized distance was used to define the fiber orientation from -70 degrees at the epicardium

to 70 degrees at the endocardium, as shown in Figure 1.

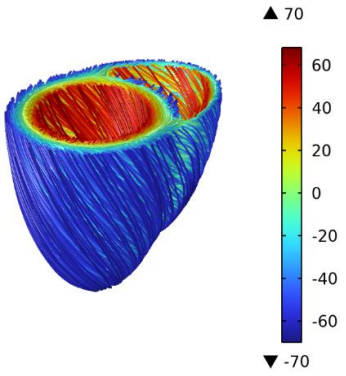


Figure 1. Orientation of myocardial fibers

Myocardial action potential formulation is based on the phenomenological model of Nash and Panfilov [7], modified to integrate units in the original dimensionless form:

$$\chi_m(C_m\frac{\partial \Phi}{\partial \mathrm{t}}+I_{ion})+\nabla\cdot(-\hat{\mathbf{q}})=0 \quad (1)$$

where Φ, $\hat{\mathbf{q}}$, and I_{ion}, are transmembrane potential, potential flux and myocardial cellular electrophysiology, respectively. Other parameters are not directly related to the experimental results but they were included to simulate the action potential behavior. Cardiac Excitation-Contraction modeled by a phenomenological ordinary differential equation as [8]:

$$\frac{\partial S_a}{\partial t}=\epsilon(\Phi)\left(K_{Sa}\left[\frac{\Phi-B}{A}\right]-S_a\right) \quad (2)$$

where S_a and $\epsilon(\Phi)$ are the active stress and the delay function, respectively. K_{Sa}, A and B are constant parameters.

For the passive myocardial response, the transversely isotropic hyperelastic model of Holzapfel and Ogden [9] is used. A nearly incompressible constraint is applied by describing the volumetric strain energy function as [8]:

$$\psi=\psi_{isotropic}+\psi_{fiber}+\psi_{vol} \quad (3)$$

$$\psi_{isotropic}=\frac{a_i}{2b_i}exp(b(I_1-3)) \quad (4)$$

$$\psi_{fiber}=\frac{a_f}{2b_f}[exp(b_f(I_{4f}-1)^2)-1] \quad (5)$$

$$\psi_{vol}=\frac{k(J-1)\,ln(J)}{2} \quad (6)$$

where $\psi_{isotropic}$, ψ_{fiber} and ψ_{vol} are the isotropic, anisotropic, and volumetric strain energy functions of the myocardium, respectively. I_1 and J are the first invariant of the isochoric right Cauchy-Green tensor $\boldsymbol{C}$ and the determinant of the deformation gradient tensor $\boldsymbol{F}$, respectively, while $I_{4f}=\hat{F}\cdot(\boldsymbol{C}\hat{F})$. In order to couple the electrical and mechanical physics, S_a is added to the second Piola-Kirchhoff stress, along the fiber, sheet and normal to the sheet directions, so that the magnitude of effective stress in the sheet and normal-to-sheet directions is 40% of the value along the fiber direction.

In line with the work of Xi et al. [3], an open-loop circulation has been used to simulate ventricular pressure. Both the inlet and outlet flows were defined by specific conditions. The inflow and outflow of the left and right ventricles are defined as follows [10]:

$$Q_{in}=\frac{(P_{LA}-P_{LV})}{R_{mt}},if\ P_{LA}>P_{LV}$$
$$Q_{out}=\frac{P_{LV}-P_{sys}}{R_{Aorta}},if\ P_{LV}>P_{Systemic} \quad (7)$$

$$Q_{in}=\frac{(P_{RA}-P_{RV})}{R_{tri}},if\ P_{RA}>P_{RV}$$
$$Q_{out}=\frac{P_{RV}-P_{Pul}}{R_{PV}},if\ P_{RV}>P_{Pulmonary} \quad (8)$$

where Eqs. (7) and (8) are describing blood flows of left and right ventricle, respectively. Ventricular pressure was determined so that the volume of the chambers was equal to the volume of blood flows entering and leaving the Windkessel models as:

$$P_{LV}:\Omega_{cur,Lv}-\Omega_{div,Lv}=0 \quad (9)$$

$$P_{RV}:\Omega_{cur,Rv}-\Omega_{div,RV}=0 \quad (10)$$

where P_{LV} and P_{RV} are left and right ventricular pressures, respectively. $\Omega_{div,Lv}$ and $\Omega_{div,RV}$ are the left and right ventricular volume obtained from the divergence theorem. $\Omega_{cur,Lv}$ and $\Omega_{cur,Rv}$ are the desired left and right ventricular volume calculated based on the blood flows. In order to simulate pulmonary hypertension caused by high pulmonary blood pressure, the initial pulmonary blood pressure was set to 30 mmHg [6].

3. Validation

To validate the electromechanical model, the results of the active stress of a healthy heart are compared with the results obtained by Bakir [6]. At a point on the epicardial surface of the left ventricular free wall, the active stress was calculated for three cardiac cycles as shown in Figure 2.

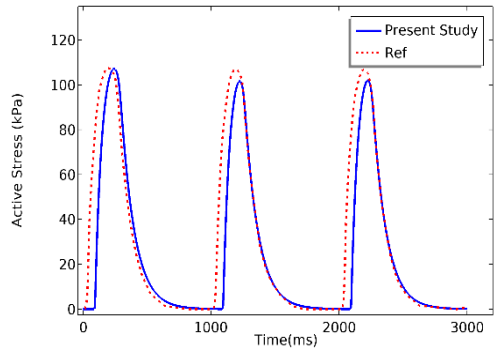


Figure 2. Active stress validation of present study with Bakir [6] for three consecutive cycles

The active stress waveform produced a rapid increase followed by a more gradual decrease. The model produced a time to peak active stress of 168 milliseconds. This duration is consistent with experimental data of Mulieri et al. [11] conducted on the epicardium of the human heart.

4. Results and discussion

Figure 3 compares the right ventricular pressure-volume loop for healthy and PAH cases. The right ventricular ejection fraction in PAH case is reduced compared to normal case (about 5%), indicating hypertension. As the disease progresses and appropriate treatment is not taken, the ejection fraction may decrease by 20% to 30%, meaning the heart pumps a much smaller proportion of the blood volume into the left ventricle and eventually to the body.

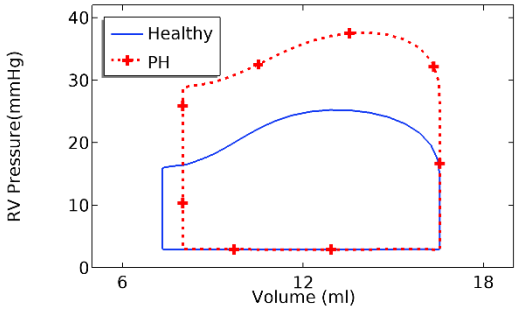
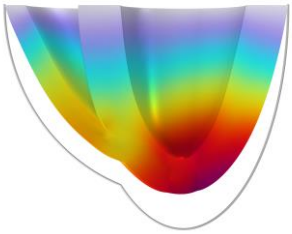


Figure 3. Right ventricular pressure-volume loop in the third cycle of healthy and pulmonary hypertension (PH) cases

Figure 4 shows the displacement contours at 2200 milliseconds of the third cardiac cycle associated with the ejection phase for the healthy and PAH cases. The



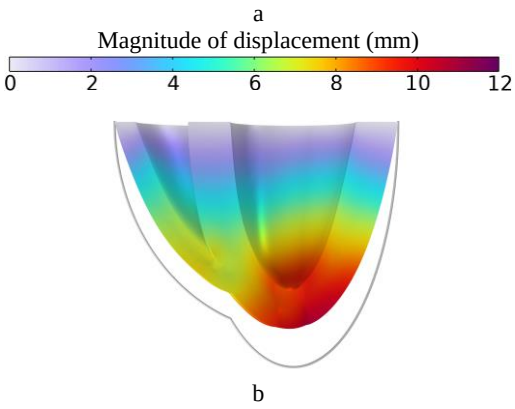


Figure 4. a) Displacement in healthy case, b) Displacement in PAH case

outer edge indicates the epicardial surface of the heart at time 2000 milliseconds, corresponding to the beginning of the third cardiac cycle. The main difference between the two cases is in the mechanical response of the right ventricle. The magnitude of displacement in the PAH case decreased, since the heart was unable to deform as it normally would despite the increased pressure. This led to a reduction in the right ventricular ejection fraction.

5. Conclusion

In this study, an electromechanical model of PAH disease was implemented in COMSOL software and the functional behavior of the heart in a diseased state was compared with a healthy case. A transversely isotropic and nearly incompressible material model was used for describing passive myocardial response. It was observed that with increasing pulmonary pressure, the right ventricular pressure magnitude exceeds the normal range and reaches about 40 mmHg. Furthermore, right ventricular ejection fraction decreased by 5% in PAH case. A decrease in ejection fraction of about 20% to 30% leads to right ventricular failure and can be life-threatening. The method of calculating ventricular pressure using the three-element Windkessel model is straightforward and provide high reproducibility. Moreover, the approach considered in this study was expanded according to Eqs. (8) and (10), in order to be applicable for cardiac biventricular models.

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