

Effect of the material properties on modeling of the excitation propagation of the human heart

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World Journal of Biology Pharmacy and Health Sciences, 2025, 22(03), 088–094

Publication history: Received on 24 April 2025; revised on 30 May 2025; accepted on 02 June 2025

Article DOI: <https://doi.org/10.30574/wjbphs.2025.22.3.0564>

Abstract

Accurate modeling of cardiac excitation propagation requires detailed representation of the heart's anisotropic properties and tissue heterogeneity. Traditional imaging modalities such as CT and MRI fail to capture the fiber orientation essential for modeling the myocardium. This study employs the Monodomain reaction-diffusion equation and integrates Diffusion Tensor Imaging (DTI) data to enhance modeling fidelity by accounting for both anisotropic properties and non-uniform conductivity distributions. The proposed method modifies the conductivity tensor using diffusion volume as a proxy for ionic conductivity. Simulation results show significant differences in activation isochrones between isotropic and anisotropic models and between uniform and non-uniform conductivity distributions. These findings emphasize the importance of tissue-specific modeling for realistic cardiac electrophysiology simulations.

Keywords: Heart Modeling; Excitation Propagation; Anisotropy; Non-Uniform Conductivity; Cardiac Electrophysiology

1. Introduction

When modeling the excitation propagation in the heart, two primary structures must be considered: the myocardium and the conduction system. The anatomical structure of the heart is typically derived from medical imaging techniques such as CT scans and MRI. However, these conventional imaging modalities lack the capability to capture tissue anisotropy, which is crucial for accurate modeling. The myocardium exhibits pronounced anisotropic characteristics that significantly influence both electrical and mechanical functions of the heart. While some models treat the myocardium as an isotropic material [1 – 5], the majority recognize its anisotropic nature.

In anisotropic heart models, determining the myocardial fiber architecture is essential and achieved through various approaches. A widely used method, exemplified by the work of Streeter et al. [6], relies on dissection-based data to assign fiber orientations to each voxel in the anatomical heart model [7 – 13]. Alternatively, models like those of Nielson et al. [14] use mathematical formulations to define fiber directions [14 – 21]. More recent developments utilize Diffusion Tensor Imaging (DTI) to map fiber structures. Some models employ DTI primarily for geometric reconstruction [22, 23], while others incorporate DTI data into the computational frameworks for solving the Forward and Inverse Problems [24 – 27]. This study investigates the impact of assuming material isotropy and uniformity on the resulting excitation propagation isochrones.

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2. Methods

2.1. Modeling of the Excitation Propagation of the Heart

The excitation propagation of the heart is modeled based on the Monodomain reaction-diffusion equation in its normalized form namely [28]:

$$\frac{\partial u}{\partial t} = \nabla \cdot (D \nabla u) + f + g \quad \dots\dots\dots (1)$$

where u and t represent the normalized transmembrane potential and the normalized time respectively, D is the normalized effective conductivity tensor of the heart material, f is the function that represent the reaction term due to ions exchange, and finally g represents the external applied input. The normalized conductivity tensor can be written as:

$$D = rI + (1-r)ee^T = e \begin{bmatrix} 1 & 0 & 0 \\ 0 & r & 0 \\ 0 & 0 & r \end{bmatrix} e^T \quad \dots\dots\dots (2)$$

such that

$$r = \frac{\sigma_{it}}{\sigma_{il}} \quad \dots\dots\dots (3)$$

where e represents the first eigenvector of the diffusion tensor, and σ_{il} and σ_{it} represent the intercellular conductivities in longitudinal and traverse directions, respectively and I is the Identity Matrix.

2.2. Modifying the Normalized Monodomain Reaction-Diffusion Equation

As the effective conductivity tensor of the Monodomain reaction-diffusion equation (2) is in its normalized form, where the heart is considered to have uniform conductivity in all of its tissues. This approximates to the real situation, where there are mainly two types of structures, the Myocardium and the conduction system, and as stated earlier they do not have the same conductivities, as the average conductivity of the Purkinje fibers in the longitudinal direction is almost three times the conductivity in Myocardium fibers. Even for the same structure, the conductivity varies slightly from zone to another due to cells dimensions and tissue parameters. Identifying the conductivity of each part of the heart is a difficult task, but with the proposed use of the DTI, it might be possible to identify such parameters.

According to literature, this is the first approach that is capable of identifying the non-uniform conductivity distribution in the biological tissues using the DTI dataset. It was reported that the parameters of the interlaced disks form the “bottle neck” of the conductivity in fibers direction [29]. However, the availability of ions in the intercellular medium is another factor that affects the conductivity. If it is assumed that there is a linear relation between number of ions and the conductivity of the cell, then the conductivity σ can be seen directly related to the number of ions N_{ions}

$$\sigma_l \propto N_{ions} \quad \dots\dots\dots (4)$$

the number of ions is directly related to the volume of the cytoplasm of the cell, and since the DV quantity is directly related to the volume of cytoplasm (as mentioned earlier) then the conductivity can be assumed to be directly related to the DV quantity

$$\sigma_l \propto DV \quad \dots\dots\dots (5)$$

and so, from equation 5

$$w = \frac{\sigma_l}{\sigma_{l1}} = \frac{DV}{DV_1} \quad \dots\dots\dots (6)$$

where DV is the diffusion volume of any tissue, DV1 is the average diffusion volume for the Myocardium tissue, σ_l is the longitudinal conductivity of any tissues, σ_{l1} is the average longitudinal conductivity of the Myocardium tissue, and w is the ratio of these. Assuming that the traverse conductivity of all cells is the same, and then the general form of equation (2) would be

$$D' = rI + (w - r)ee^T = e \begin{bmatrix} w & 0 & 0 \\ 0 & r & 0 \\ 0 & 0 & r \end{bmatrix} e^T \quad \dots\dots\dots (7)$$

The diffusion term in the equation (1) can be modified to include the effect of non-uniform conductivities of the heart tissue

$$\frac{\partial u}{\partial t} = \nabla \cdot (D' \nabla u) + f + g \quad \dots\dots\dots (8)$$

By referring to the Bidomain reaction-diffusion equation, there are many parameters that should be modified in this equation, as well as the assumption of equal conductivities ratio of the longitudinal and traverse directions in both the intercellular domain and the extracellular domain for the result to be accurate. However, at this stage the effect of longitudinal conductivities difference will be included in the diffusion term of the Monodomain reaction-diffusion equation and the other parameters effect will be considered in future work.

3. Results

3.1. The Effect of Modeling the Heart as Isotropic Material on Excitation Propagation

The excitation propagation isochrones for both the anisotropic heart uniform materials (Figure 1) as the reference, and the isotropic heart uniform material (Figure 2) are generated using the conduction network [30, 31] which has been extracted from the heart DTI data [32, 33] using Diffusion Volume (DV) [34, 35].

The conductivity of the isotropic heart material is taken to be the average of both longitudinal and traverses conductivities of the anisotropic materials (where $\sigma_l = 34.4$ mS/mm and $\sigma_t = 5.96$ mS/mm then $\sigma_{AVG} = 20$ mS/mm) [36]. Excluding the anisotropy information about the heart material, significantly affects the produced excitation propagation. Figure 3 show that there are significant differences in activation time between the two cases.

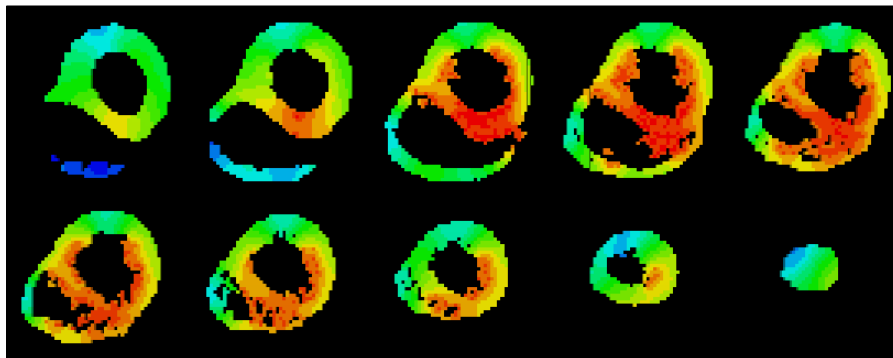


Figure 1 The isochrones for the excitation propagation of the heart when it is considered an Anisotropic material (reference)

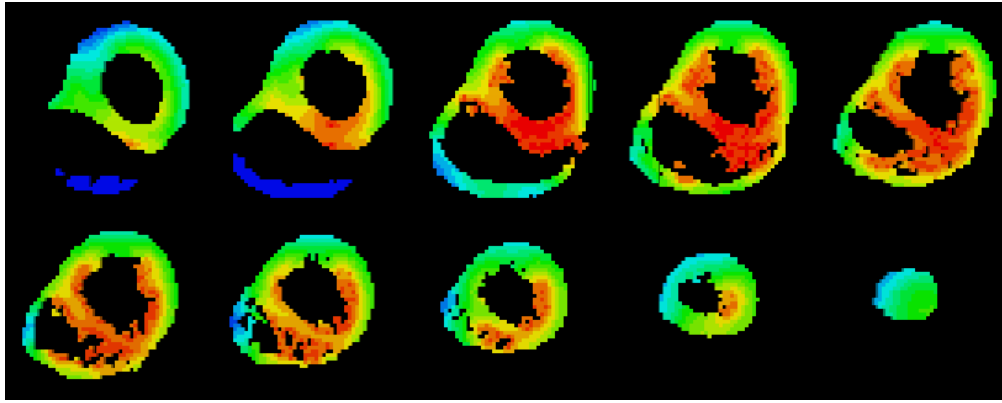


Figure 2 The isochrones for the excitation propagation of the heart when it is considered an isotropic material

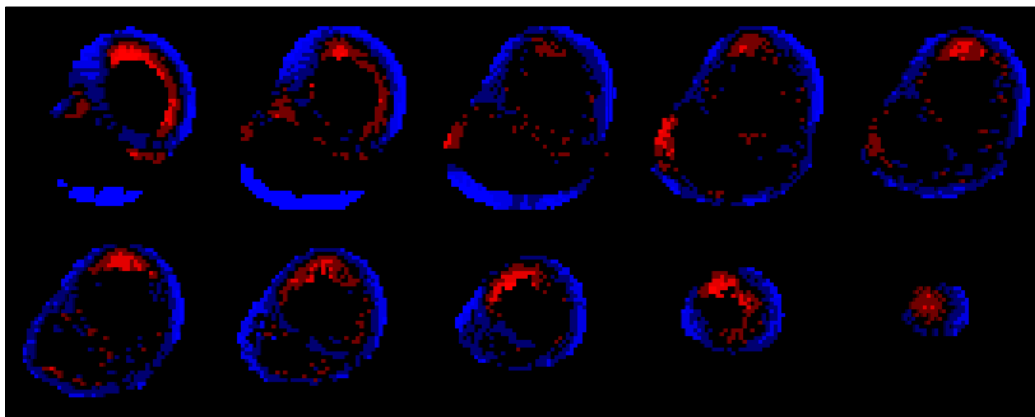


Figure 3 Activation isochrones difference (Red = Lead, Blue = Lag)

3.2. Comparison between Uniform and Non-Uniform Distribution of Ventricles Conductivities

The excitation propagation isochrones for both the anisotropic heart uniform materials (Figure 1) as the reference and the anisotropic heart non-uniform material (Figure 4) are generated. The difference map (Figure 5), shows that the non-uniform conductivity activation leads in the left ventricle free wall and the IV Septum, and lag in the right ventricle free wall. This is much more realistic than the uniform model, since in the activation isochrones of Durrer et. al. [37], the right ventricle free wall is activated about 25 mSec later than the left ventricle.

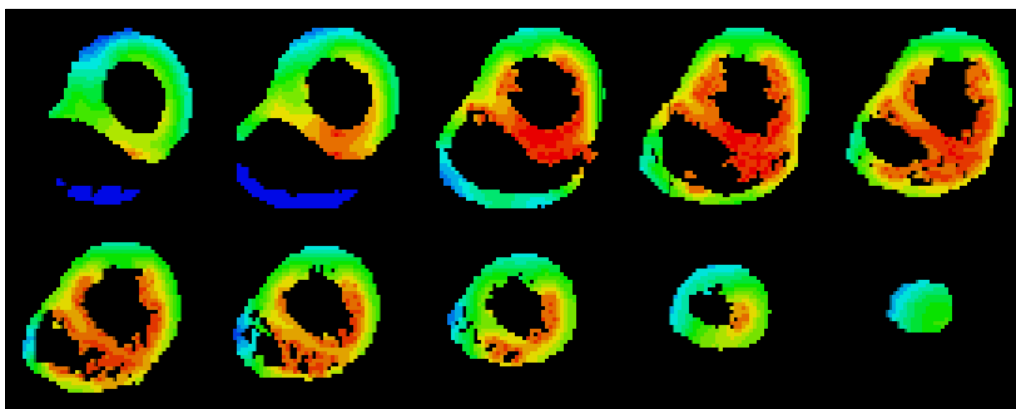


Figure 4 Activation isochrones for non-uniform distribution of conductivities of ventricles

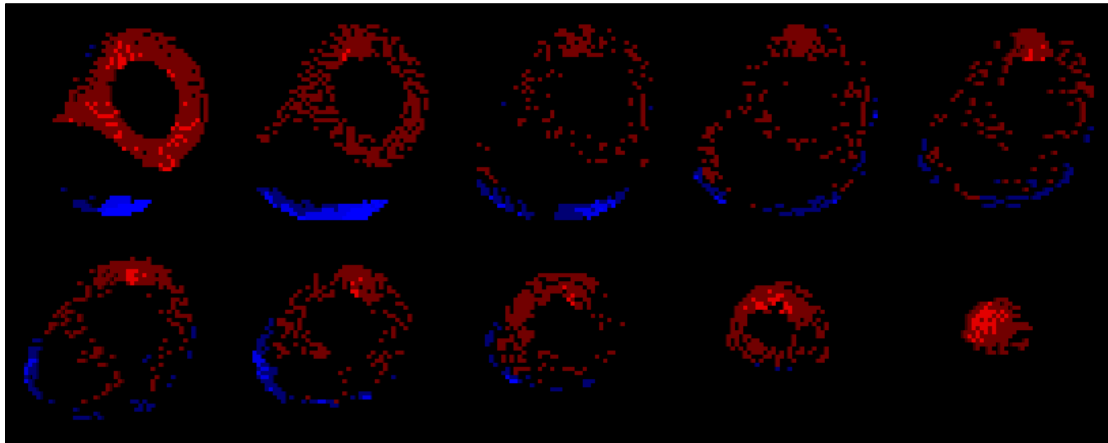


Figure 5 Activation isochrones difference (Red = Lead, Blue = Lag)

4. Conclusion

This study demonstrates the critical impact of both anisotropy and conductivity heterogeneity on the accuracy of excitation propagation modeling in the human heart. Isotropic approximations significantly distort the propagation patterns, while the inclusion of DTI-derived non-uniform conductivity distributions yields results that align more closely with clinical observations. The integration of diffusion volume metrics into the conductivity model provides a novel, biologically grounded approach for representing ionic behavior in myocardial tissue. Future extensions will include a more complete implementation of Bidomain parameters and further validation against empirical activation data.

Compliance with ethical standards

Acknowledgments

Dr. Patrick A. Helm and Dr. Raimond L. Winslow at the Centre for Cardiovascular Bioinformatics and Modelling of John Hopkins University and Dr. Elliot McVeigh at the National Institute of Health for provision of DTI data.

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