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Modeling of the excitation propagation of the human heart

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Abstract

This study presents a computational model for simulating excitation propagation in the human heart using a Monodomain reaction-diffusion framework coupled with the Aliev-Panfilov model for the ionic reaction term. The objective is to address the Forward Problem in cardiac electrophysiology by modeling how electrical activation initiated at the conduction system propagates through the myocardium. Cellular and tissue-level dynamics are integrated using diffusion tensor imaging (DTI)-derived anisotropy and conduction network structures. Two conduction system models are evaluated, one based on trabecular muscle anatomy and another using diffusion volume (DV) metrics. Numerical simulations demonstrate activation isochrones comparable to experimental data from Durrer et al., highlighting the model's validity in capturing realistic ventricular excitation patterns. Visualization was achieved using OpenGL-based C/C++ simulations.

Keywords: Cardiac Electrophysiology; Excitation Propagation; Monodomain Equation; Activation Isochrones

1. Introduction

There are two problems in modeling the electrophysiology of the heart: the Forward Problem and the Inverse Problem [1]. The heart Forward Problem involves designing a model that is capable of determining the field on the surface of known body (conductor) that is generated by electrical-sources inside the heart. Solving the Forward Problem requires the development of electrical models which are capable of describing the bioelectrical criteria of the heart and the body (Figure 1). The heart Inverse Problem [1] is to design a model that is capable of recovering the electrical-sources locations in the heart from a measured field on the body surface. The forward problem of electrophysiology modeling starts with modeling excitation propagation inside the heart's myocardium and latter, it will be extended to generate the electrical field on the surface of the body.

The conduction system represents the initial excitation points of the heart Myocardium. The scope of much of the work deals with identifying the ventricular conduction system. The most common models that are used to identify the ventricular conduction system are those described by Tawara [2], Massing et. al.[3], and Durrer et. al.[4].

Cell scale models have been used for modeling excitation propagation [5 – 7], while other models tend to employ tissue scale models [8 – 13]. Some qualitative models for cellular excitation have also been developed [14 – 21] to model the cardiac excitation.

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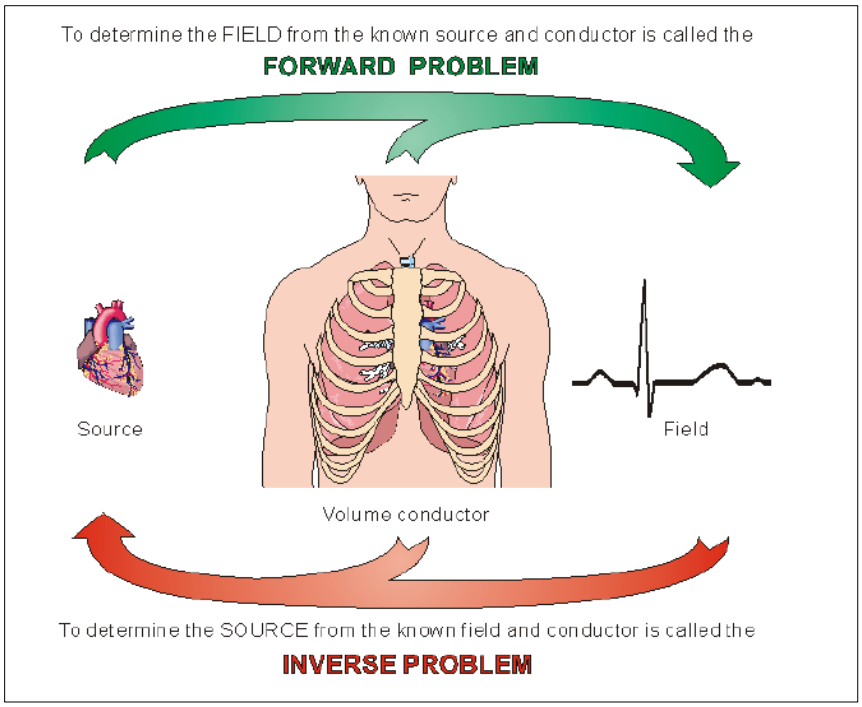

Figure 1 The Forward and the Inverse Problems of the heart electrophysiology [1]

2. Methods

2.1. Cardiac Excitation models

Modeling of the heart electrical excitation is presented on either the cellular scale or for the whole tissue. The cellular scale models describe the cell action potential according to an individual cell. Cellular scale relates the effect of different ions current to the variation in the action potential. The most famous model of this type of excitation model was introduced by Hodgkin and Huxley [22]. It is considered the first quantitative model that describes the excitation of the nerve cell. This model relates the effect of sodium ions, potassium ions, and leakage currents on the action potential that is measured on the surface of the cell membrane with respect to time. Many other quantitative models that represent the action potential in the cell scale have also been presented. These are mainly modified versions of Hodgkin-Huxley (HH) model, which are capable of handling the different types of cell and ions currents. Nobel [23] introduced a model that describes the excitation in Purkinje cells, which includes the sodium current and two potassium currents, the depolarizing and the pace maker that is slowly increasing. Another model, the Beeler-Reuter (BR) model [24] introduces the action potential of ventricular Myocardium cells, where four currents are included, the fast sodium current (voltage and time dependent), a secondary slow current of mainly calcium (voltage and time dependent), outward potassium current exhibiting inward-going rectification (time dependent), and another outward potassium current (voltage and time dependent). The Lou-Rudy (LR) model [25] is a modified version of the BR model, which introduces information about extracellular and intercellular domains.

Multi-cellular models have also been developed which describe the excitation propagation of action potential in the whole tissue. They can be used in a single cell scale as well. The excitation propagation inside the heart can be described by Bidomain equation of reaction diffusion or Monodomain equation of reaction diffusion. Modeling of Monodomain equation for excitation propagation has been introduced in different research studies. However, using Bidomain models requires more parameters and more computation time where Monodomain models require less parameters and less computation time and the differences between the Monodomain and the Bidomain results were extremely small [26].

2.2. Aliev-Panfilove (AP) Model

The most used model was introduced by FitzHugh and Nagumo (FHN) [27], and represents the excitation by two variables representing the depolarization and the repolarization of the cell membrane. This model is simplified and presented in different forms [28, 29] but was updated by Aliev and Panfilove [30], where they modified the normalized form of the FHN model to include the effect of Action Potential Duration (APD).

$$f = -ku(u-a)(u-1) - uv \quad \text{..... (1)}$$

such that

$$\frac{\partial v}{\partial t} = \varepsilon(u,v)(-v - ku(u-a-1)) \quad \text{..... (2)}$$

and

$$\varepsilon(u,v) = \varepsilon_0 + \frac{\mu_1 v}{u + \mu_2} \quad \text{..... (3)}$$

where for the heart tissues, k=8, a=0.15, ε_0 =0.002, μ_1 =0.2 and μ_2 =0.3.

2.3. 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 [13]:

$$\frac{\partial u}{\partial t} = \nabla.(D\nabla u) + f + g \quad \text{.....(4)}$$

where u and t represent the normalized transmembrance 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. For cardiac cells, u and t can be calculated as:

$$u = \frac{V_m + 80}{100} \quad \text{.....(5)}$$

$$t(unit \cdot time) = \frac{t(actual \cdot time)}{12.9} \quad \text{.....(6)}$$

and the normalized conductivity tensor can be written as:

$$D = rI + (1-r)ee^T = e\begin{bmatrix} 1 & 0 & 0 \\ 0 & r & 0 \\ 0 & 0 & r \end{bmatrix}e^T \quad \text{.....(7)}$$

such that

$$r = \frac{\sigma_{it}}{\sigma_{il}} \quad \text{.....(8)}$$

where e represents the first eigenvector of the diffusion tensor, and σil and σit represent the intercellular conductivities in longitudinal and traverse directions, respectively and I is the Identity Matrix. The longitudinal and the traverse conductivities take the values σil =34.4 mS/mm, and σit =5.96 mS/mm, which make r = 0.17. Modeling of the reaction part of the equation is accomplished using the Aliev-Panfilove [30] model.

The solution to equations (2) and (4) can be written in discrete form using Taylor expansion series as follows:

$$u^{t+1} = u^t + dt[\nabla.(D\nabla u^t)] + dt[-ku^t(u^t - a)(u^t - 1) - u^t v^t] + dt[g(I_{app})] \quad \text{.....(9)}$$

$$v^{t+1} = v^t + dt[\varepsilon(u^t, v^t)(-v^t - ku^t(u^t - a - 1))] \quad \text{..... (10)}$$

where dt is the time step and Xt+1 and Xt represents the next and the current values of the variable respectively. Where (0≤ u ≤ 1)

The diffusion term can be written as:

$$\nabla.(D\nabla u) = \begin{bmatrix} d_{11}\frac{\partial^2 u}{\partial x^2} + d_{22}\frac{\partial^2 u}{\partial y^2} + d_{33}\frac{\partial^2 u}{\partial z^2} + \\ 2d_{12}\frac{\partial^2 u}{\partial x \partial y} + 2d_{23}\frac{\partial^2 u}{\partial y \partial z} + 2d_{13}\frac{\partial^2 u}{\partial x \partial z} + \\ \frac{\partial u}{\partial x}\left[\frac{\partial d_{11}}{\partial x} + \frac{\partial d_{21}}{\partial y} + \frac{\partial d_{31}}{\partial z}\right] + \\ \frac{\partial u}{\partial y}\left[\frac{\partial d_{12}}{\partial x} + \frac{\partial d_{22}}{\partial y} + \frac{\partial d_{32}}{\partial z}\right] + \\ \frac{\partial u}{\partial z}\left[\frac{\partial d_{13}}{\partial x} + \frac{\partial d_{23}}{\partial y} + \frac{\partial d_{33}}{\partial z}\right] \end{bmatrix} \quad \text{..... (11)}$$

By applying Taylor's expansion series such that

$$\frac{\partial u}{\partial x} = \frac{u_{x+1,y,z} - u_{x-1,y,z}}{2h_x} = \frac{(u_{x+1,y,z} - u_{x,y,z}) + (u_{x,y,z} - u_{x-1,y,z})}{2h_x} \quad \text{.....(12)}$$

$$\frac{\partial^2 u}{\partial x^2} = \frac{u_{x+1,y,z} - 2u_{x,y,z} + u_{x-1,y,z}}{h_x^2} = \frac{(u_{x+1,y,z} - u_{x,y,z}) - (u_{x,y,z} - u_{x-1,y,z})}{h_x^2} \quad \text{.....(13)}$$

$$\frac{\partial^2 u}{\partial x \partial y} = \frac{u_{x+1,y+1,z} - u_{x-1,y+1,z} - u_{x+1,y-1,z} + u_{x-1,y-1,z}}{4h_x h_y}$$
$$= \frac{(u_{x+1,y+1,z} - u_{x,y,z}) + (u_{x,y,z} - u_{x-1,y+1}) + (u_{x,y,z} - u_{x+1,y-1,z}) + (u_{x-1,y-1} - u_{x,y,z})}{4h_x h_y} \quad \text{.....(14)}$$

where h's are the displacements between nodes in each direction. These are given from the DTI dataset settings (hx = 0. 4297 mm, hy= 0. 4297 mm, and hz=1.0 mm [31]). Deriving the first derivative as a central difference will provide less solution error than the forward or backward difference [32]. The term dt[g(Iapp)] is set to 1 when the desired location is considered to be externally activated.

2.4. Heart Model for Generating Activation Isochrones

As heart activation is the base point to generate the Body Surface Potential Map (BSPM) on the developed human torso [32], activation isochrones is generated based on the heart model which has been developed in an earlier stage [33] using DTI scans [34]. Activation inside that model will be initiated using two models of conduction network [35]. The first model of conduction network is built manually based on the Trabecular muscles locations and the other was extracted using Diffusion Volume quantity (DV) [35 – 37].

3. Results

The implementation of the Forward Problem solution was accomplished using the C/C++ programming language. The OpenGL library was used in all of the implemented programs to visualize structural and Forward Model results for both the body and the heart models. The excitation propagation (QRS complex only) of normal activation was implemented for both proposed models of the conduction system. The excitation isochrones of both models are shown in Figure 2 and Figure 3.

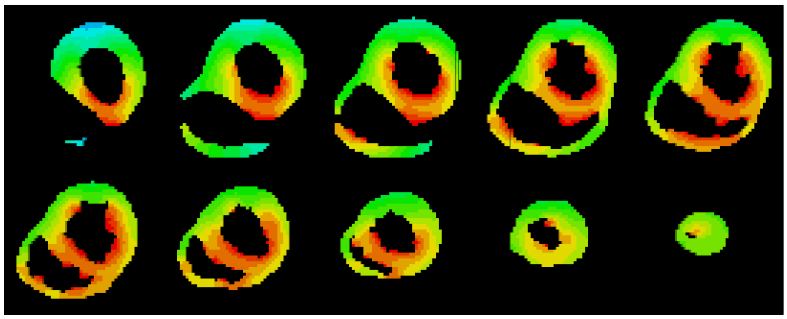

Figure 2 Sample of Excitation Isochrones of Normal Activation using model 1 of conduction system

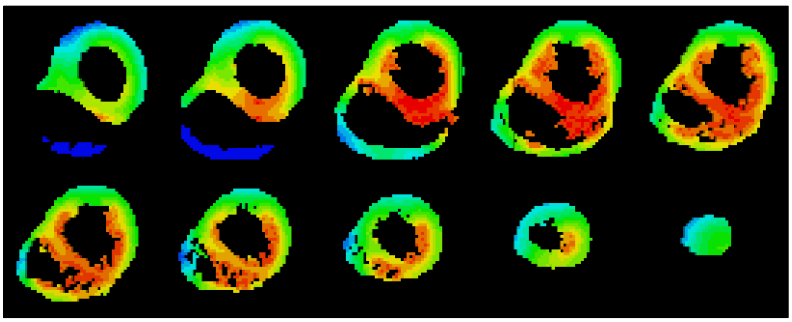

Figure 3 Sample of Excitation Isochrones of Normal Activation using model 2 of conduction system

Employing the proposed conduction system models in human heart myocardium produce ventricular excitation propagation isochrones similar to the measurements of Durrer et. al. [4] as shown in Figure 4.

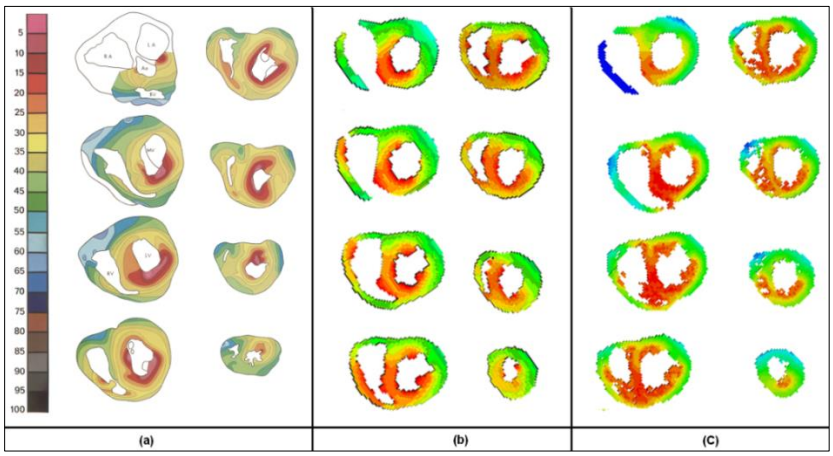

Figure 4 Activation isochrones of the normal activation of ventricles (a) from Durrer et. al measurements [4] (b) using the manual conduction system (Model 1) (c) using the proposed conduction system (Model 2)

The remarkable nature in this propagation is that the activation starts in the Endocardium wall and propagates towards the Epicardium wall through the Myocardium. The anterior parts of the free walls are activated later than the posterior

ones. The RV completes its depolarization later than the LV. However, these isochrones may not be identical, as the conduction systems of different human hearts do not have exactly the same structure [3].

4. Conclusion

This work successfully models the excitation propagation of the human heart by solving the Monodomain equation with an Aliev-Panfilov reaction model, incorporating anatomically-based anisotropy and conduction system structure. The simulation results closely align with experimental isochrones reported in literature, confirming the accuracy of both proposed conduction models. These findings validate the effectiveness of DTI-derived models in replicating physiological excitation behavior and form a solid basis for future efforts in body surface potential mapping and inverse problem solving in cardiac electrophysiology.

Compliance with ethical standards

Acknowledgments

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References

[1] J. Malmivuo and R. Plonsey "Bioelectromagnetism: Principles and Applications of Bioelectric and Biomagnetic Fields" Oxford Univ. Press, 1st Ed., (1995); ISBN: 0195058232.

[2] S.Tawara "The Conduction System of the Mammalian Heart" English Ed., World Scientific Pub. co., (1998), ISBN: 981023502X.

[3] G.K. Massing, and T.N. James "Anatomical Configuration of the His Bundle and Bundle Branches in the Human Heart" Circ. (1976); 53(4):609-621.

[4] D. Durrer, R.TH. Van Dam, G.E. Freud, M.J. Janse, F.L. Meijler and R.C. Arzbaecher "Total Excitation of the Isolated Human Heart" Circulation, (1970); 41(6):899-912.

[5] B.H. Smaill, I.J. LeGrice, D.A. Hooks, A.J. Pullan, B.J. Caldwell and P.J. Hunter "Cardiac structure and electrical activation: Models and measurement" Proc. of the Australian Physiological and Pharmacological Society, (2004); 34: 141-149

[6] D.S. Farina, O. Skipa, C. Kaltwasser, O. Dossel and W.R. Bauer "Personalized Model of Cardiac Electrophysiology of a Patient" IJBEM (2005);7(1): 303-306

[7] S.B. Knisley, N. Trayanova, and F. Aguel "Roles of Electric Field and Fibre Structure in Cardiac Electric Stimulation" Biophysical Journal, (1999); 77:1404–1417

[8] D.F. Scollan "Reconstructing The Heart: Development and Application of Biophysically Based Electrical Models of Propagation in Ventricular Myocardium Reconstructed from DTMRI", Ph.D. Thesis, Johns Hopkins University (2002)

[9] [54] L.W. Wang, H.Y. Zhang, P.C. Shi "Simultaneous Recovery of Three-dimensional Myocardial Conductivity and Electrophysiological Dynamics: A Nonlinear System Approach" Computers in Cardiology, (2006);33:45-48.

[10] O. Berenfeld and J. Jalife "Purkinje-Muscle Reentry as a Mechanism of Polymorphic Ventricular, Arrhythmias in a 3-Dimensional Model of the Ventricles" Circ. Res., (1998);82;1063-1077

[11] M. Sermesant, Y. Coudiere, H. Delingette, N. Ayache, J. Sainte-Marie,D. Chapelle, F. Clement and M. Sorine "Progress towards model-based estimation of the cardiac electromechanical activity from ECG signals and 4D images" ESAIM, 2002; 12: 153-162.

[12] M. Sermesant , H. Delingette, and N. Ayache "An Electromechanical Model of the Heart for Image Analysis and Simulation" IEEE Trans. Med. Imag. (2006); 25(5): 612-625.

[13] I. ELAFF (2018) "Modeling of realistic heart electrical excitation based on DTI scans and modified reaction diffusion equation," Turkish Journal of Electrical Engineering and Computer Sciences: Vol. 26: No. 3, Article 2. https://doi.org/10.3906/elk-1710-118J

[14] M. Seger "Modeling the Electrical Function of the Human Heart", Ph.D. Thesis, Institute of Biomedical Engineering, University for Health Sciences, Medical Informatics and Technology, Austria (2006).

[15] C. Hintermuller "Development of a Multi-Lead ECG Array for Noninvasive Imaging of the Cardiac Electrophysiology", Ph.D. Thesis, Institute of Biomedical Engineering, University for Health Sciences, Medical Informatics and Technology,Austria, (2006)

[16] M. Seger, R. Modre, B. Pfeifer, C. Hintermuller and B. Tilg "Non-invasive Imaging of Atrial Flutter" Computers in Cardiology (2006);33:601-604.

[17] T. Berger, G. Fischer, B. Pfeifer, R. Modre, F. Hanser,T. Trieb, F. X. Roithinger, M. Stuehlinger, O. Pachinger,B. Tilg, and F. Hintringer "Single-Beat Noninvasive Imaging of Cardiac Electrophysiology of Ventricular Pre-Excitation" J. Am. Coll. Cardiol. (2006);48:2045-2052.

[18] L. Cheng "Non-Invasive Electrical Imaging of the Heart", Ph.D. Thesis, The University of Auckland,New Zealand (2001).

[19] B. He, G.Li, and X. Zhang "Noninvasive Imaging of Cardiac Transmembrane Potentials Within Three-Dimensional Myocardium by Means of a Realistic Geometry Anisotropic Heart Model" IEEE Trans. Biomed. Eng. (2003); 50(10): 1190-1202.

[20] G. Li, X. Zhang, J. Lian, and B. He "Noninvasive Localization of the Site of Origin of Paced Cardiac Activation in Human by Means of a 3-D Heart Model" IEEE Trans. Biomed. Eng. (2003); 50(9): 1117-1120.

[21] Z. Liu, C. Liu, and B. He "Noninvasive Reconstruction of Three-Dimensional Ventricular Activation Sequence From the Inverse Solution of Distributed Equivalent Current Density" IEEE Trans. Med. Imag. (2006); 25(10): 1307-1318.

[22] A.L. Hodgkin and A.F. Huxley "A quantitative description of membrane currentand its application to conduction and excitation in nerve" the Journal of Physiology (1952);117:500-544.

[23] D. Noble "A modification of the Hodgkin—Huxley equations applicable to Purkinje fibre action and pacemaker potentials" the Journal of Physiology (1962);160:317-352.

[24] G.W. Beeler and H. Reuter "Reconstruction of the action potential of ventricular myocardial fibres" the Journal of Physiology (1977);268:177-210.

[25] C.H. Luo and Y. Rudy "A model of the ventricular cardiac action potential. Depolarization,repolarization, and their interaction" American Heart Association. (1991);68:1501-1526.

[26] M. Potse, B. Dube, A. Vinet and R. Cardinal "A comparison of monodomain and bidomain propagation models for the human heart" IEEE EMBS'06, (2006); Conf. Proc.: 3895-3898.

[27] R. Fitzhugh "Impulses and Physiological States in Theoretical Models of Nerve Membrane" Biophysical J., 1961; 1: 445-466.

[28] A. V. Panfilov, P. Hogeweg "Spiral breakup in a modified FitzHugh—Nagumo model" Phys. Let. A, 1993; 176: 295—299.

[29] A.T. Winfree "Varieties of spiral wave behavior: An experimentalist's approach to the theory of excitable media" CHAOS ,(1991) ;1(3): 303-334.

[30] R.R. Aliev, A.V. Panfilov "A simple two-variable model of cardiac excitation." Chaos, Solitons and Fractals, 1996; 7(3): 293-301.

[31] The Center for Cardiovascular Bioinformatics and Modeling, John Hopkins University Site "http://www.ccbm.jhu.edu/research/DTMRIDS.php", July 2011.

[32] Stanley I.Grossman “Calculus” 5ht Ed., Saunders Collage Publishing, (1992), ISBN: 0-03-096420-2.

[33] I. ELAFF “Modeling of 3D Inhomogeneous Human Body from Medical Images”, World Journal of Advanced Engineering Technology and Sciences. 2025, 15(02): 2010-2017. https://doi.org/10.30574/wjaets.2025.15.2.0772

[34] I. I. ELAFF “Modeling of the Human Heart in 3D Using DTI Images” World Journal of Advanced Engineering Technology and Sciences, 2025, 12(02), 2450-2459. https://doi.org/10.30574/wjaets.2025.15.2.0812

[35] I. ELAFF “Modeling Human Heart Conduction Network in 3D using DTI Images” World Journal of Advanced Engineering Technology and Sciences. 2025, 15(02), 2565–2575, https://doi.org/10.30574/wjaets.2025.15.2.0838

[36] I. ELAFF, I. A. EL-KEMANY, and M. KHOLIF "Universal and stable medical image generation for tissue segmentation (The unistable method)," Turkish Journal of Electrical Engineering and Computer Sciences: Vol. 25: No. 2, Article 32, 2017. https://doi.org/10.3906/elk-1509-100

[37] I. Elaff, “Medical Image Enhancement Based on Volumetric Tissue Segmentation Fusion (Uni-Stable 3D Method)”, Journal of Science, Technology and Engineering Research, vol. 4, no. 2, pp. 78–89, 2023. https://doi.org/10.53525/jster.1250050.