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Research Article

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Solving Heterogeneities in Defibrillation for a Vascular Remodel of the Human Heart

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Abstract

Introduction: *Current mathematical models impede the amelioration of defibrillation protocol. Heterogeneities such as intracellular clefts, scarring, blood vessels, and fiber orientation are excluded in modeling. Such geometries pose a positive curvature, magnifying resistance when faced with electrical shock. Thus, such geometries have the potential of revolutionizing AED machines-a prospect we have not gained enough data on to consider.*

Objective: *The purpose of my study is to quantify the effect of each non-conformity in relation to the electrical dynamics of the human heart. Post-quantification, I hypothesize that the electrical impedance decreases as the heterogeneity size decreases. Posed with such a window of pertinence, my goal is to remodel the human heart including all heterogeneities-a previous infeasibility.*

Methodology: *Using CHASTE cardiac software library, electrical shock was applied to cardiac tissue engineered in Mesh Lab. Cardiac tissue, the slab geometries, contained blood vessels in the center of varying vessel size-400, 200, 100μm.*

Results: *Overall, the results showed that without heterogeneities biological reality and computational modeling have severe discrepancies; mainly, the experimentally supported therapy of low-energy ant fibrillation shows failure in math modeling. Perpendicular fiber orientation perceived shock at a 1.74x more efficacies. In the most sensitive case scenario, 400 and 200μm affected the defibrillating wave-front, while the 100μm heterogeneity did not.*

Conclusion: *All heterogeneities cannot be extracted by magnetic resonance angiography due to its limiting factor of magnetic susceptibility; however, by filtering can anatomically accurate mathematical remodel capable of representing the necessary cardiac vessels is created.*

Keywords: Leap, Defibrillation, Minimum Radius, Model Filtration, Computational Model, Mathematical Modeling, Chaste Cardiac

Introduction

Cardiovascular disease is the No.1 killer worldwide [1]. The most common type, electrical, ends in a sudden cardiac arrhythmia. Current anti-arrhythmia treatments are ineffective-a 3000V shock scars the surrounding cardiac tissue, which causes successive arrhythmias in the future [2]. Current defibrillation therapy permanently damages the heart, and the cure-pacemakers-are also problematic: installing a separate system to synchronize with the complexity of the human heart, pacemakers fail an impulse is sent too soon or too quick [3].

Mathematical modeling will ameliorate treatments, but current math models are inadequate [4]. In computational research, mediums that can be mathematized holistically are called homogeneities (such as, muscle interaction), while a realm of heterogeneities that cannot be mathematized also exist (such as, intracellular clefts, fiber orientation, blood vessels, and scarring) [5]. Heterogeneities are excluded because the human heart has a fractal configuration of such geometries, and its inclusion rests on the ability of magnetic resonance angiography. Smaller heterogeneities cannot be extracted due to their low magnetic susceptibility [6].

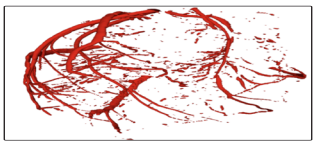

Figure 1: MRA extraction of cardiac geometries.

Methodology

Muscle tissue was constructed in Mesh Lab. The voltage shock formula was used as a pre-requisite to calibrate the new code. All files were created, edited, and recompiled on a personal Linux system. Simulations were sent to Stony Brook's LiRED machines where 96 processors were used for 10 hours. Code was written mainly in CHASTE cardiac altered by adding New Restart functionality and using python scripts to fill in the missing notations in CHASTE cardiac such as tanθ. The bidomain method explicates the trans

membrane potential of the heart. The ionic source term, I'() ϕ, y, was resolved by the ionic current method. The operating splitting method solved the bidomain equations.

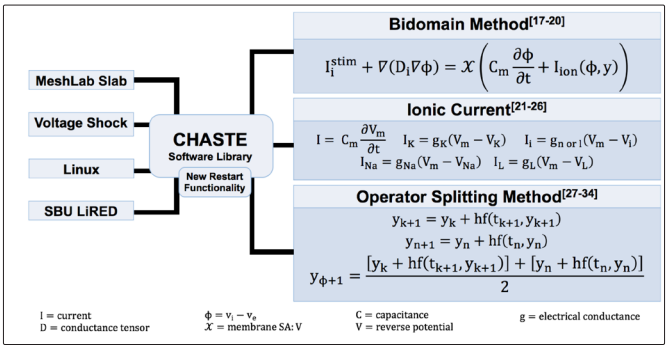

Figure 2: Flow chart of methods.

Results and Discussion

The widely-used Mahajan 2008 cardiac model, which does not include heterogeneities, was tested against a .33V shock. Virtual electrodes (VE) were not formed by the 1st shock, nor by the 2nd, 3rd, 4th, or 5th. Depolarization, VEs, was supposed to occur.

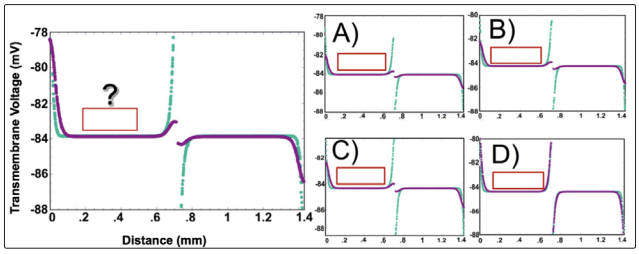

Figure 3: MRA extraction of cardiac geometries

To solve for the discrepancy, the anatomic model (Figure 1) needed to be filtered using a minimum radius. To find a minimum radius, the most optimized case scenario defined by when shock is perceived most efficaciously needed to be defined.

Parallel fiber orientation didn't produce enough electrical resistance against the incoming shock for the blood vessel to locally react.

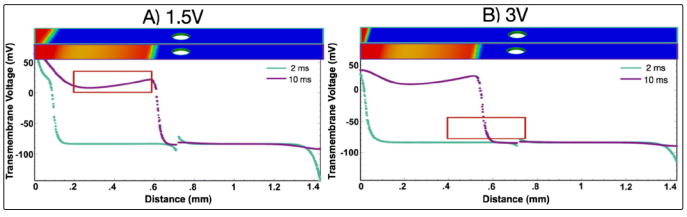

Figure 4: Change of voltage when shock is applied parallel

However, the perpendicular fiber orientation had localized virtual electrode formation.

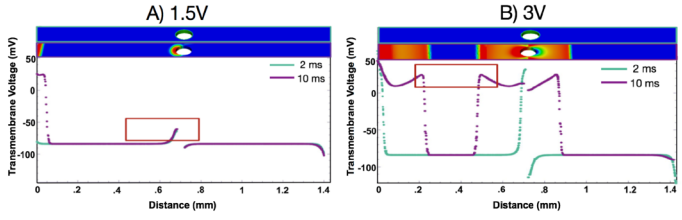

Figure 5: Depolarization as shock is applied bidirectional to fibers

As shock strength increased, the disparity between the perpendicular and parallel difference exasperated.

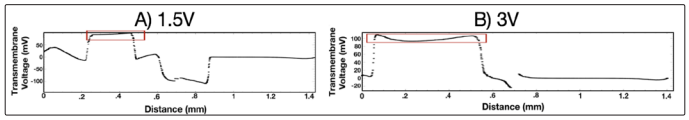

Figure 6: Excitation disparity between parallel and perpendicular.

Creating a ratio between the Trans membrane potential of perpendicular and parallel shock, the relationship is defined as: By solving the infinite geometric series, perpendicular fiber orientation will perceive shock at a 1.74 times more efficacy than parallel shock. Since its more sensitive, heterogeneities that don't effect perpendicular defibrillation, wouldn't affect parallel anti-fibrillation- this is the most optimized case scenario.

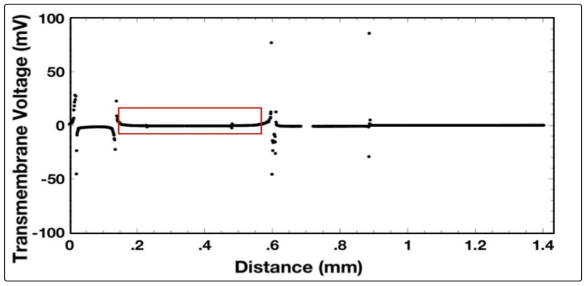

Figure 7: 1.74 ratios

$$\frac{\chi_1\left(C_m \frac{\partial\phi_1}{\partial t_1} + I_{ion}(\phi_1, y_1)\right) + \cdots + \chi_n\left(C_m \frac{\partial\phi_n}{\partial t_n} + I_{ion}(\phi_n, y_n)\right)}{\chi_1\left(C_m \frac{\partial\phi_1}{\partial t_1} + I_{ion}(\phi_1, y_1)\right) + \cdots + \chi_n\left(C_m \frac{\partial\phi_n}{\partial t_n} + I_{ion}(\phi_n, y_n)\right)} \quad (1)$$

$$\frac{C_{m_{x_1}^e} + C_{m_{x_2}^e} + \cdots + C_{m_{x_n}^e}}{C_{m_{x_1}^a} + C_{m_{x_1}^a} + \cdots + C_{m_{x_1}^a}} = \frac{\frac{C_{m_{x_1}^e}}{1-r}}{\frac{C_{m_{x_1}^a}}{1-r}} = \frac{C_{m_{x_1}^e}}{C_{m_{x_1}^a}} = 1.74 \quad (2)$$

When a 6V shock was applied to a perpendicularly fiber slab geometry against a 400µm heterogeneity, both VE formation and velocity of moving wave-front were altered. Before the shock even reached the heterogeneity, there was a local activation and responding wave.

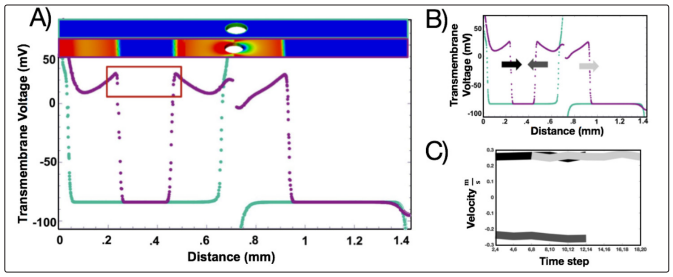

Figure 8: Effect of 400µm heterogeneity

Moving down to 200µm radius size heterogeneities, the tissue locally activates and a returning wave is sent back to the site of shock application at a negligible velocity. The effect is minimizing, but still present.

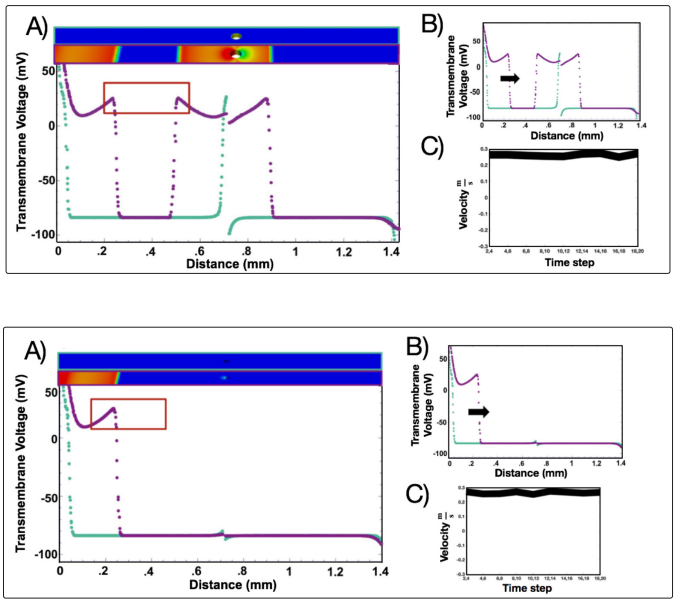

Figure 8: Effect of 100µm heterogeneity

Since ≤ 100µm plays no role in cardiac defibrillation, all ≥ 100µm radii heterogeneities were filtered to create the first anatomically accurate mathematical remodel of the human heart.

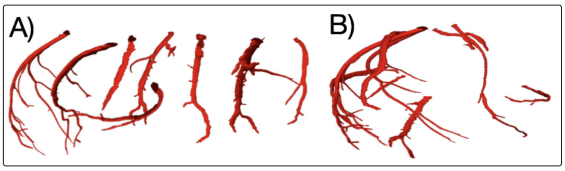

Figure 9: New vascular remodel of the human heart

Conclusions

Previously, including the vascular configuration of the human heart or any cardiac models was not feasible [7]. My novel approach of quantifying the effect of non-continuous mediums revolutionizes the capabilities of mathematical modeling as it can now go beyond continuous biological realities. Since heterogeneities disrupt the depolarization of charge, an inclusion for studying cardiac defibrillation, would allow us to lower the voltage of treatment. Given the low energy anti-fibrillation method, AED machines could be reduced to .33V, a 1000-fold reduction [8]. Such a reduction would make a costly solution to heart attack cost that of an allergic reaction, saving the United States trillions of dollars in health care [9]. However, world-wide, AED machines are inconvenient: too much of a liability for the workplace, too powerful for the home setting, and too expensive ($2k) for developing nations [10]. The secondary alternative is to call for help, delaying CPR protocol and each minute loss decreases chance of survival by 70% [11]. Futuristically, the relationship between blood vessels and SPH intensity can be delineated for the elucidation of a minimum radius in head-impact injury research [12]. Here, mathematical models don't include the complexity of vascular configuration [13] while it's known a high-density of anatomical structure will cushion the brain during impact [14, 15].

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