

Computational Cardiac Electrophysiology

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Abstract: A Comparative Study About The Electrophysiological Model Of The Heart Is Described Here. In Recent Times, Mathematical Model Of Cardiac Electrical Activity Has Been Recognized As One Of The Significant Approaches Capable Of Revealing Diagnostic Information About The Heart.

Keywords: Cardiac Action Potential, Cardiac Mathematical Models-Cable Model, Hodgkin Huxley, Bidomain, Monodomain Models

I. Introduction

Cardiac Electrophysiology Is The Science Of The Mechanisms, Functions And Performance Of The Electrical Activities Of The Heart[1]. The Most Critical Job Of The Heart Is To Expel Blood To The Whole Body, And Transport Critical Nutrients To And Remove Waste Products From The Tissues. The Heart Is A Muscular Pump With Rhythmic Electrical Activity And Muscle Contraction.

Mechanical Contraction Of Cardiac Tissue Or Muscle Fibers Are Triggered By Electrical Depolarization Of The Cell Membrane, And Coordinated By The Spread Of Depolarization Through Cardiac Tissue From The Sino Atrial Node (SA Node) To Other Regions Of The Heart. The Sequence Of Depolarization And Subsequent Repolarization Can Be Measured With Electrodes And Other Many Techniques Like Optical Imaging [2]-[4]. The Cardiac Muscle Fibers In The Heart Are Arranged In Interconnected Manner. If One Of The Muscle Cell Is Excited, The Action Potential Will Spread To All Of Them, Spreading From Cell To Cell Through Its Interconnection. This Interconnected Nature Of Cardiac Muscle Fibers Is Called Syncytium. Heart Consist Of Three Major Types Of Cardiac Muscles; Atrial Muscle, Ventricular Muscle And Specialized Excitatory And Conductive Muscle Fibers. The Duration Of Contraction Of The Atrial And Ventricular Muscle Fibres Are Too Longer. But The Contraction Of Specialized Excitatory And Conductive Muscle Fibres Are Feebly Because They Contain Few Contractile Fibrils. So They Exhibit Either Automatic Rhythmical Electrical Discharge In The Form Of Action Potentials Or Conduction Of The Action Potential Through The Heart. The Rhythmical Beating Of The Heart Is Controlled By The Excitatory System[5].

The Mathematical Model Of The Heart Is Used To Determine The Diagnostic Information About The Heart. Over The Past 60 Years, Many Mathematical Models Have Already Been Developed For The Same [6] – [10]. The Main Cause Of Sudden Cardiac Death Is Due To Cardiac Electrical Abnormalities, Preventing Blood Circulation To Various Compartments Of The Body. In Diagnosing These Disastrous Cardiac Electrical Abnormalities, Mathematical Modelling Of Cardiac Electrical Activities Plays A Vital Role, Which Reveals The Baseline Diagnostic Information About The Functional Status Of Heart.

This Paper Describes The Review Of Different Mathematical Models Of The Action Potential Propagation In The Heart. This Paper Organized As Follows. The Section 2 Cover The Blood Flow Physiology, In Section 3 The Action Potential, Excitation And The Electrical Conduction Of The Heart Is Analyzed. The Different Cardiac Electrophysiological Models Are Discussed In Section 4 And Finally Comparisons Of The Different Cardiac Models Are Presented In Section 5.

II. Blood Flow Physiology

The Heart Lies In The Center Of The Thoracic Cavity. The Pathway Of Blood Flow Through The Chambers Of Heart Is Shown In Fig.1. The Heart Shown In Figure 1 Is Actually Two Separate Pumps; A Right Heart That Pumps Blood Through The Lungs, And A Left Heart Pumps Blood Through The Peripheral Organs [11]-[12]. The Contaminated Blood (Presence Of Carbon Dioxide) After Circulation Through The Body, Enters The Right Atrium Of The Heart Through Two Veins, Namely Superior Vene Cava (SVC) And Inferior Vene Cava (IVC). The SVC Collects Blood From The Upper Half Of The Body. The IVC Collects Blood From The Lower Half Of The Body. After Contraction Of The Right Atrium, The Blood Pumps To The Right Ventricle Through The Tricuspid Valve. The Ventricle Pumps Blood Through Pulmonary Artery Into Lungs. The Lungs Consist Of, Tiny Blood Vessels Called Capillaries Absorb Carbon Dioxide From The Blood And Replace It With Oxygen. The Oxygenated Blood Then Flows Into Left Atrium Through The Pulmonary Vein.. Oxygenated

Blood Then Pumps Through The Bicuspid Valve And Into The Left Ventricle. The Left Side Of The Heart Contracts And To Send Blood Out The Left Ventricle And Through The Aortic Arch On Its Way To All Parts Of The Body. At This Point, There Are A Few Options For The Blood Flow: Blood Can Be Pumped

- Through The Carotid Artery And Into The Brain.

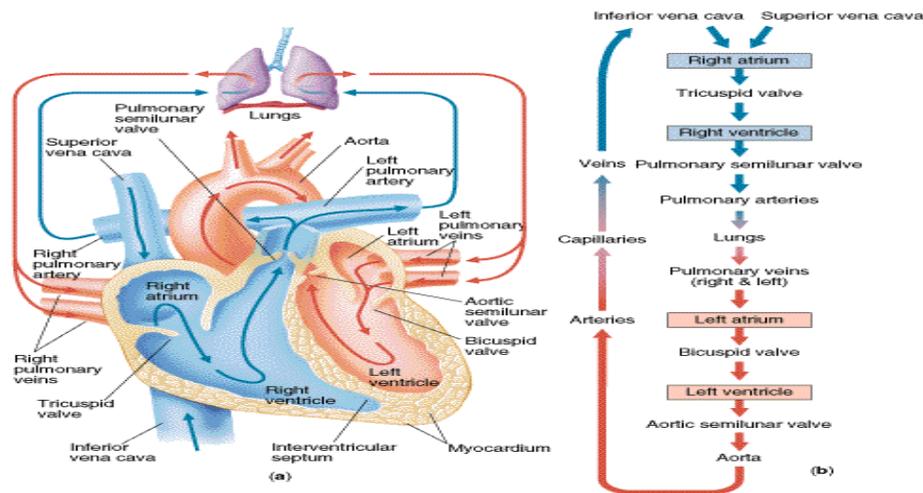


Fig.1 Blood Flow Physiology

- Through The Auxiliary Arteries And Into The Arms.
- Through The Aorta And Into The Torso And Legs.

Blood Will Then Move Through The Arteries, Then Through Capillaries, And Then Return Through The Veins. The Deoxygenated Blood (Blood Without Oxygen) Will Then Return To The Heart And Again The Cycle Repeats.

III. Electrical Excitation & Conduction In The Heart

3.1 Action Potential

An Action Potential Is A Short-Lasting Event In Which The Electrical Membrane Potential Of A Cell Rapidly Rises And Falls, Following A Stereotyped Trajectory [13]. The Fig.2 Below Shows The Action Potential In Cardiac Muscle.

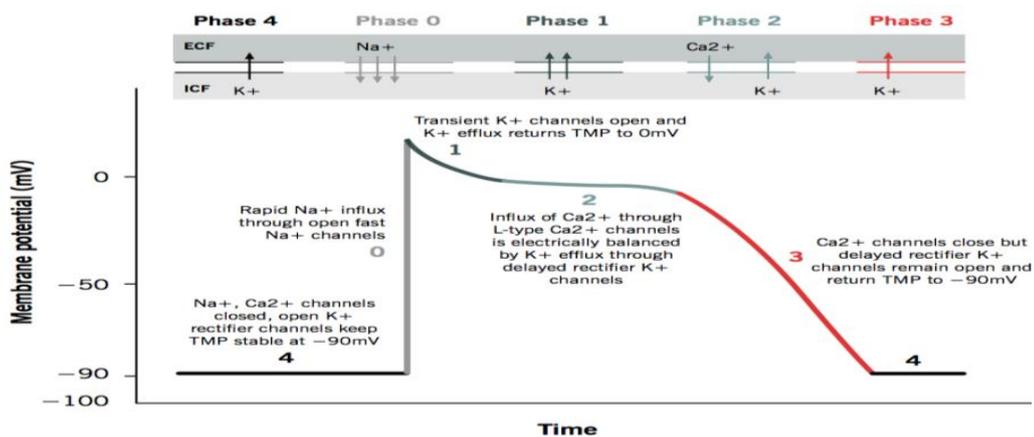


Fig.2. Action Potential In Cardiac Muscle

In The Cardiac Muscle, The Action Potential Is Due To The Opening Of Two Types Of Channels.

- 1) Fast Sodium Channel
- 2) Slow Calcium Channel

The Presence Of The Sodium, Potassium, Calcium And Chlorine Ions In The Inside And Outside Of The Cell Membrane Creates A Potential, Known As Action Potential. These Ion Fluxes, Or Membrane Currents, Can Be Measured And Analyzed At The Level Of Single Channel Or Whole Cell By ECG[14]

Phase 0 (Rapid Depolarization-Upstroke): In This Phase Voltage Is Changes From -90mv To 20mv. This Is Due To Sudden Increase In Membrane Permeability To Sodium Ions And Decrease Of Potassium Permeability.Ie Sodium Pumps In And Potassium Pumps Out And Generate Sharp Spike Of Initial Action Potential.

Phase 1(Early Rapid Repolarization): Potassium Ion's Outward Flow Occurs In This Phase. Its Period Is About 10ms

Phase 2 (Plateau): The Membrane Potential Then Reaches A Steady Point At Around Zero Millivolts. It Is Known As Plateau Of The Action Potential. The Time Period Is 100-150ms. In This Phase Slow Repolarization Is Occurs.Ie Ca^{2+} Inward Flow And K^{+} Outward Flow.

Phase 3 (Late Rapid Repolarization): Rapid Repolarization Occurs.Ie Ca^{2+} Close Up And K^{+} Cannels Are Widen, The Time Required For This Phase Is About 100-150ms.

Phase 4 (Resting Potential): The Voltage Decreases To Its Original Value Where It Will Remain Steady Until The Next Action Potential Is Generated.

3.2 Excitation & Conduction In The Heart:

The Heart Is Endowed With A Special System For (1) Generating Rhythmical Electrical Impulses To Cause Rhythmical Contraction Of The Heart Muscle And (2) Conducting These Impulses Rapidly Through The Heart. The Fig.3 Below Shows The Specialized Excitatory And Conductive System Of The Heart, That Controls The Cardiac Contraction [15].

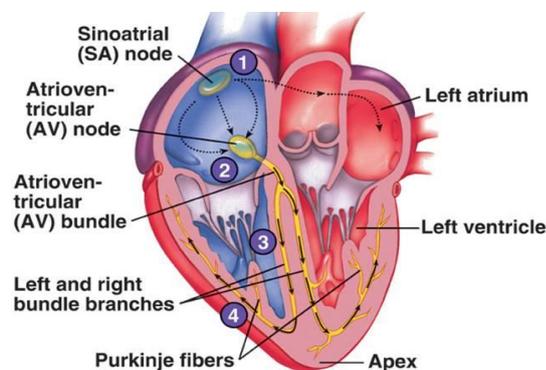


Fig.3 Cardiac Electrical Conduction

The Sinoatrial Node (SA), Atrioventricular Node (AV), AV Bundle, Right And Left Bundle Branches, Purkinje Fibers Are The Main Components Of The Conducting System In The Heart [16]. Normally Spontaneous Action Potentials Are Generated By SA Node. This Electrical Impulse Initiates The Contraction. The Intermodal Pathways In The Right Atrium That Conducts The Impulse From The SA Node To AV Node.Ie The Cardiac Impulse Does Not Travel From The Atria To Ventricle Too Rapidly, It Happens Only Through AV Node. AV Node Is Located In The Posterior Wall Of The Right Atrium Immediately Behind The Tricuspid Valve. From The AV Node, The Impulse Then Travels Through The Bundle Of His And Down The Bundle Branches, Fibers Specialized For Rapid Transmission Of Electrical Impulses, On Either Side Of The Interventricular Septum. Both The Bundle Of Branches Terminated In Purkinje Fibers. Once The Impulse Reaches The Ends Of The Purkinje Fibers, It Is Transmitted Through Ventricular Muscle Mass By The Ventricular Muscle Fibers. The Total Time For Transmission Of The Cardiac Impulse In The Normal Heart From The Initial Bundle Branches To The Last Of The Ventricular Muscle Fibers Is About 0.06second.

IV. Different Cardiac Electrophysiological Model

This Section Describes About The Different Mathematical Model Of Heart. Ie Cable Model, Hodgkin–Huxley Model Of The Action Potential, Bidomain And Monodomain Models Etc. The Mathematical Model Of Cardiac Electrical Activity Has Been Used To Revealing Diagnostic Information About The Heart.

4.1 Cable Model

The One Dimensional Cable Model Of The Cell Is Very Helpful To Understand About How An Action Potential Is Propagated Along The Cell. To Describe The Behavior Of Ionic Currents Across The Cell Membrane, We Need A Model For The Electrical Behaviour Of The Cells In Terms Of Action Potential. For This Purpose Lord Kelvin Developed Cable Equation In 1850s. Here The Cardiac Cell Is Considered As A Cylindrical Membrane Which Separates Internal Conducting Medium From Extracellular Conducting Medium. The Cell Membrane Act As A Relative Insulator With Properties Described In [17] And The Potential Depends Only On The Length Variable, And On Time. Fig.4 Shows The Cable Model Of A Cardiac Cell.

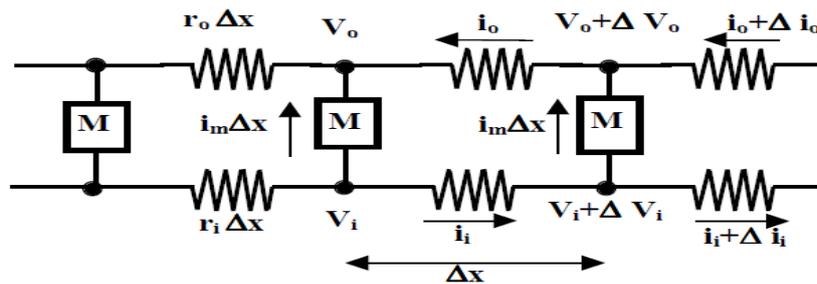


Fig.4 Cable Model Of A Cardiac Cell

In This Model Charge Carriers Are Assumed To Move In Only One Dimension Inside And Outside Of The Cell. The Box M Represents The Lumped Properties Of The Membrane Over A Length ΔX . r_i And r_o Be The Inside And Outside Conductors Resistances Per Unit Length. i_m And v_m Are The Membrane Current And Potential Respectively. v_m Is The Difference Between v_o And v_i . The Voltages And V_i Represents The Extra Cellular And Intracellular Potentials. The Current Flowing Inside And Outside Of The Cell Are i_i And i_o , And At Any Point They Must Be Equal And Opposite.

Apply Ohm's Law At Inner Conductor

$$i_i r_i \Delta X = -\Delta V_i \tag{1}$$

In The Limit ΔX Tends To Zero, This Becomes

$$\frac{\partial V_i}{\partial x} = -i_i r_i \tag{2}$$

In The Same Way Apply Ohms Law At Outer Conductor

$$\frac{\partial V_o}{\partial x} = -i_o r_o = i_i r_o \tag{3}$$

Apply KCL At Any Node, We Get

$$\Delta i_i = -i_m \Delta x \quad \text{Ie, } \frac{\partial i_i}{\partial x} = -i_m \tag{4}$$

According To The Definition Of The v_m , $\frac{\partial v_m}{\partial x} = -i_i (r_i + r_o)$ (5)

Differentiating The Above Equation & Substitute In (4), We Get

$$i_m = \frac{1}{(r_i + r_o) x^2} (\frac{\partial^2}{\partial x^2} v_m) \tag{6}$$

The Membrane Current Per Unit Length Is

$$i_m = C_m \frac{\partial v_m}{\partial t} + \frac{v_m}{r_m} \tag{7}$$

Where C_m Is The Membrane Capacitance Per Unit Length And r_m Is The Membrane Resistance Per Unit Length.

Substitute Equation (7) In Too Equation (6)

$$\frac{\partial^2}{\partial x^2} (v_m(x, t)) = (r_i + r_o) (C_m \frac{\partial v_m}{\partial t} + \frac{v_m}{r_m}) \tag{8}$$

This Is The One Dimensional Cable Equation. One Dimension Cable Theory Is The Useful Tool For The Basic Study Of Active Potential Propagation Through And Point Stimulation Of Cardiac Tissue [18]. This Theory Gives Sufficient Information About Many Documented Cardiac Response To Defibrillation Shocks. But This Theory Cannot Adequately Explain How A Defibrillation Shock Induces Direct Excitation Through The Entire Heart.

4.2 Hodgkin Huxley Model

The Hodgkin–Huxley Model, Or Conductance Based Model, Is A Mathematical Model That Describes How Action Potentials In Heart Is Initiated And Propagated. It Is A Set Of Nonlinear Differential

Equations That Approximates The Electrical Characteristics Of Excitable Cells Such As Neurons And Cardiac Myocytes, And Hence It Is A Continuous Time Model.

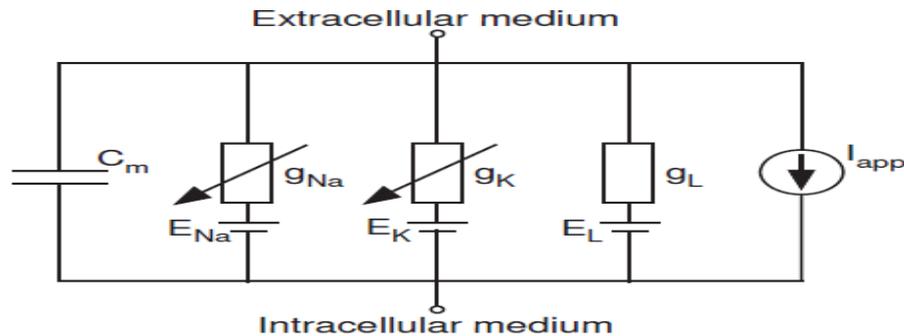


Fig.5. Hodgkin-Huxley Model Of The Action Potential

In This Model Cell Membrane Can Be Modeled As A Capacitor In Parallel With A Number Of Ionic Current. Basic Components Of The Hodgkin-Huxley Model Are Shown In Fig.5 [19-21]. Here Capacitance C_m Represents The Cell Membrane. The Nonlinear Conductance g_{Na} , g_k Are The Voltage Gated Sodium And Potassium Channels. Leak Ion Channels Are Represented By A Linear Conductance g_L . E_{Na}, E_K, E_L Are The Electrochemical Gradients Due To The Flow Of Ions. The Applied Stimulus Current Is Represented By I_{app} . Current Flowing Through The Given Ion Channel Is

$$I_i = g_i(V_m - V_i) \tag{9}$$

Where V_i Is The Potential Of The Ion Channel. Thus For A Cell With A Sodium And Potassium Channel, The Total Current Through The Cell,

$$I_m = C_m \frac{\partial V_m}{\partial t} + I_{Na} + I_k + I_L - I_{app} \tag{10}$$

Ie,
$$I_m = C_m \frac{\partial V_m}{\partial t} + g_{Na}(V_m - V_{Na}) + g_K(V_m - V_K) + g_L(V_m - V_L) - I_{app} \tag{11}$$

This Is The Hodgkin- Huxley Model Of The Action Potential.

4.3 Bidomain Model

The Bidomain Model Is A Mathematical Model For The Electrical Properties Of Cardiac Muscle That Takes Into Account The Anisotropy Of Both The Intracellular And Extracellular Spaces. The Model Is Considered As The Mathematical Equations That Have Been Used For Simulating Cardiac Electrophysiological Waves For Years With The Non Linear Dynamic Nature Of The Cardiac Signal And Giving Realistic Simulation. This Model Is The Generalization Of One Dimensional Cable Theory [17] And Is Also Known As Continuum Model, Ie It Represents The Average Properties Of Many Cells Rather Than Representing Each Cell Individually, Model Gives The Representation Of The Cardiac Tissue At A Macroscopic Scale[22, 23] . Instead Of Accurately Modeling The Geometry Of The Two Domains, They Are Assumed To Be Overlapping, Both Filling The Complete Volume Of The Heart Muscle. Hence, Every Point In The Myocardium Lies In Both The Intracellular And The Extracellular Domain [24, 25]. The Model Accounts For The Different Electrical Conductivities Of The Intracellular And Extracellular Spaces. Both Of These Spaces Are Anisotropic: They Have A Different Electrical Conductivity In The Direction Parallel To The Myocardial Fibers Than In The Direction Perpendicular To Them. Moreover, The Degree Of Anisotropy Is Different In The Two Spaces. In The Intracellular Space The Conductivity Parallel To The Fibers Is About Ten Times Greater Than The Conductivity Perpendicular To The Fibers (10:1), Whereas In The Extracellular Space The Ratio Is Only About 5:2. This Condition Of Unequal Anisotropy Ratios Leads To Many Of The Interesting Predictions Of The Bidomain Model. It Consists Of A System Of Two Nonlinear Partial Differential Equations Coupled To A System Of Ordinary Differential Equations.

The Electrical Circuit Approximation Of The Bidomain Model [26] Is Shown In Fig.6.

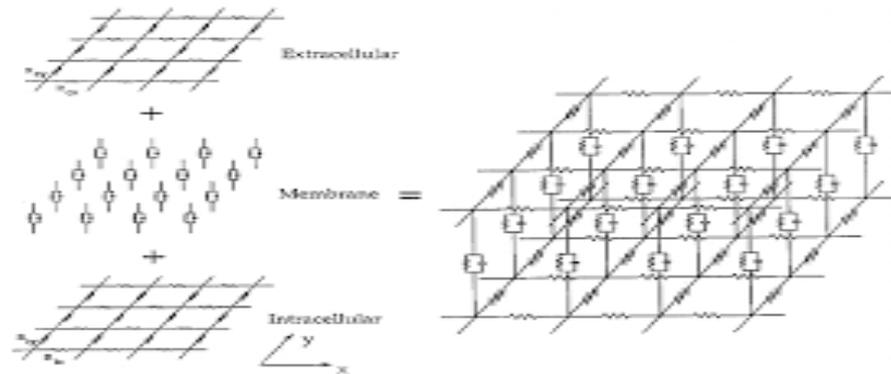


Fig.6 Electric Circuit Approximation Of Bidomain Model

Intra Cellular And Extra Cellular Spaces Are Represented By Using Resistor Grid. The Two Spaces Are Coupled By Means Of Cell Membrane. In Order To Derive The Electrical Activity Of The Heart Using Bidomain Approach The Following Laws Are Used With Divergence Theorem.

- The Electrical Charge Conservation Law
- The Electrical Conduction Law
- The Consequences To The Electromagnetic Induction

The Bidomain Model Describes The Cardiac Tissue As Two Conducting Phase Termed As Intracellular Domain And Extra Cellular Domain, Which Are Characterized By Conductivity Tensors σ_i And σ_e Respectively.

The Fig 7 Below Shows The Schematic Model Of Bidomain Space. Let j_i And j_e Are The Current Density In The Intra And Extra Cellular Domains. I_m Is The Membrane Current

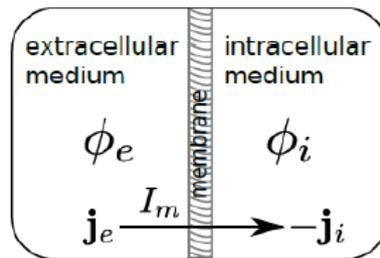


Fig.7 Schematic Model Of Bidomain Space (Ref 27)

$$\nabla \cdot J = 0 \tag{12}$$

$$J = \sigma E \tag{13}$$

$$E = -\nabla \Phi \tag{14}$$

$$J = -\sigma \nabla \Phi \tag{15}$$

Where E Is The Electric Field In V/M, σ Is The Conductivity In Siemen/Meter And Φ Is The Electric Potential In V. The Equations (1) To (4) Are Respectively Called Electrical Charge Conservation Law, Ohms Law And Consequence To Electromagnetic Induction Law. These Equations Are The Basic Equations For Bidomain Approach. Consider The Intracellular And Extra Cellular Domains Can Be Assumed To Be Superimposed On The Whole Heart Volume Ω_H . So The Average Intracellular, Extracellular Electric Potentials, Current Densities And Conductivity Tensors Are Defined In Ω_H . Here We Assume That No Accumulation Of Charge For Body Tissue Outside The Heart. Ie Total Current Entering The Small Volume Ω_H Must Equal To Total Current Leaving The Volume. Hence Application Of Charge Conservation Law Ie Equation (12) On The Heart Volume Leads To

$$\nabla \cdot j_i = -\nabla \cdot j_e = \Psi_m I_m \tag{16}$$

$$\nabla \cdot (j_i + j_e) = 0 \tag{17}$$

Ψ_m Is The Surface To Volume Ratio Of The Cell Membrane Per Meter (m^{-1}).

The Bidomain Model Is The Extension Of The Cable Theory To Three Dimensional Space. Substitute Equ (15) Into Equ (16), We Get

$$\nabla \cdot (\sigma_i \nabla \Phi_i) = -\nabla \cdot (\sigma_e \nabla \Phi_e) \tag{18}$$

The Equation (7) Can Be Expressed In Terms Of Membrane Potential V_m , Which Is The Potential Difference Between The Intracellular And Extracellular Domain.

$$V_m = \Phi_i - \Phi_e \tag{19}$$

Then Equation (18) Become

$$-\nabla \cdot ((\sigma_i + \sigma_e) \nabla \Phi_e) = \nabla \cdot (\sigma_i \nabla V_m) \tag{20}$$

According To Hodgkin And Huxley Model, The Equation Of Electrical Activity In The Heart Is Given By Equ. (10). Substitute Equations (10) And (19) In Equation (16), We Obtain

$$\nabla \cdot (\sigma_i \nabla V_m) + \nabla \cdot (\sigma_e \nabla \Phi_e) = \Psi_m (C_m \frac{\partial V_m}{\partial t} + I_{ion}(V_m, w) - I_{app}) \tag{21}$$

Where W Is The Ionic Variable, Which Satisfies The System Of Ordinary Equation Shown Below

$$\frac{dw}{dt} = G(V_m, w) \text{ In } \Omega_H; \text{ Where G Is The Vector Valued Function.} \tag{22}$$

The Bidomain Model Given By The Equations (20), (21) And (22) Explains About Extracellular Potential Φ_e Coupled With The Differential Equation For The Membrane Potential V_m As Well As An Ordinary Differential Equation Representing Ionic Current W. In Order To Complete The Mathematical Bidomain Model, We Need A Set Of Interface And Boundary Conditions.

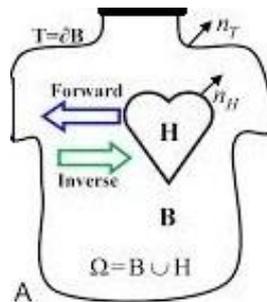


Fig.8 Schematic Representation Of Heart And Torso Domain

Here We Define The Heart Domain As H, Which Consist Of Both Extra Cellular And Intracellular Domain And The Rest Of The Body Represented As T. J_0, σ_0, Φ_0 Be The Current, Conductivity, Potential In The Torso Domain. The Border Between Torso And Heart Is Denoted As ∂H Shown In Fig.8. According To [28], Tung Described That, There Is No Current Going Directly From The Intracellular Domain To Torso Domain.

$$\text{Ie, } J_i \cdot n_H = 0 \text{ On } \partial H \tag{23}$$

Where n_H Is The Unit Length Vector, Directed Outward From The Heart Surface. In Terms Of Conductivity, Equation (23) Becomes,

$$\sigma_i \nabla (\sigma_i \nabla V_m + \sigma_i \nabla \Phi_e) \cdot n_H = 0 \text{ On } \partial H \tag{24}$$

As For The Interaction Between The Extracellular And Torso Domain, These Are Two Connected Volume Conductors And So The Potentials Must Match Up At The Interface, And Any Current Leaving One Medium Must Enter The Other,

$$\text{Ie, } \Phi_e = \Phi_o \text{ On } \partial H \tag{25}$$

$$(\sigma_e \nabla \Phi_e) \cdot n_H = -(\sigma_o \nabla \Phi_o) \cdot n_T \text{ On } \partial H \tag{26}$$

It Is Assumed That No Current Leaves The Body Surface. The Boundary Condition On ∂B Is

$$(\sigma_o \nabla \Phi_o) \cdot n_B = 0 \text{ On } \partial \Omega \tag{27}$$

Now We Have A System Of Partial Differential Equation, That Constitute The Core Of The Bidomain Model.

$$-\nabla \cdot ((\sigma_i + \sigma_e) \nabla \Phi_e) = \nabla \cdot (\sigma_i \nabla V_m) \text{ In } \Omega_H \tag{28}$$

$$\nabla \cdot (\sigma_i \nabla V_m) + \nabla \cdot (\sigma_e \nabla \Phi_e) = \Psi_m (C_m \frac{\partial V_m}{\partial t} + I_{ion}(V_m, w) - I_e) \text{ In } \Omega_H \tag{29}$$

4.4 Monodomain Model

In Order To Understand The Patterns Of Electrical Conduction And Propagation From The Scale Of A Single Tissue To Whole Heart, Some Physical Models Were Constructed In Which The Cell Membrane Is Viewed As An Electrical Network With The Fibers Of Myocardial Cells Constituting A Cable. The Cable Equation In Two Dimensional Form Is Sometimes Called Monodomain Equation, Since It Involves Principally The Intracellular Domain Only. The Monodomain Model Is A Reduction Of The Bidomain Model Of The Electrical Propagation In Myocardial Tissue. The Reduction Comes From Assuming That The Intra And Extracellular Domains Have Equal Anisotropy Ratios. I.e Conductivity In Extra Cellular Space Is Proportional To The Intra Cellular Space. Although Not As Physiologically Accurate As The Bidomain Model, It Is Still Adequate In Some Cases, And Has Reduced Complexity.

$$\sigma_e = \Lambda \sigma_i \tag{30}$$

Where Λ Is The Ratio Between The Conductivity Of The Intra And Extra Cellular Space. Substitute The Above Relation To Bidomain Model, We Get

$$\nabla \cdot \frac{\lambda}{1+\lambda} G_i \nabla V_m = \Psi_m (C_m \frac{\partial V_m}{\partial t} + I_{ion}) \tag{31}$$

Thus It Is Possible To Find The Transmembrane Potential By Solving Equation (31). Here $\frac{\lambda}{1+\lambda} G_i$ Can Be Represented As Effective Conductivity. This Is Called Monodomain Model.

Comparison of Different Electrophysiological Models

The Cable Model Is The One Dimensional Mathematical Model Of The Propagation Of Action Potential In The Heart. The Bidomain Model [29], [30] Is Currently Considered As The Most Accurate And Physiologically Founded Description For The Electrical Cardiac Behaviour And Is Widely Used To Simulate Action Potential Spreading In The Myocardium As Well As Electrocardiograms. Its Mathematical Model Consists Of One Parabolic Diffusion Equation Coupled With One Elliptical Equation. The Bidomain Model Can Be Formulated By Means Of Degenerate System Of Parabolic Reaction Diffusion Equations [31] And In [32] It Can Be Reformulated Into One Parabolic Semi-Linear PDE But Including Non Locality In Space. These Structural Properties Bring Numerical Difficulties. Moreover Cardiac Action Potential Involving Fast Space And Time Potential Variations, Fine Space And Time Grids Must Be Considered. For These Two Reasons Simulating The Cardiac Electrical Activity With The Bidomain Model Has A Very High Cost.

The Major Difficulties With Bidomain Model Are The Computational Grid Size, That Must Be Very Fine To Get A Realistic Simulation Of Cardiac Tissue. Action Potential Is A Wave With Sharp Depolarization And Repolarization Fronts And This Wave Travels Across The Whole Computational Domain Requiring A Very Fine Uniform Mesh. Solving The Bidomain Model Numerically Is A Complex Task, Both In Terms Of CPU Time And Memory, Only Possible With Extensive Parallel Computing Or Massive Supercomputing Facilities

The Monodomain Model Is A Simplification Of The Bidomain Model Reading A Single Parabolic Reaction Diffusion Equation (Still Coupled With The Same ODE System Modelling Cell Membrane). Although This Simplification Has No Mathematical General Justification, And Although The Monodomain Model Lacks Physiological Foundation, It Is Commonly Used In Electrocardiology: Firstly Because It Obviously Lead To Much Lower Computational Efforts Than The Bidomain Model. The Second Reason Motivating The Interest For The Monodomain Model Is That, As An Approximation Of The Bidomain Model, It May Serve To Improve Numerical Scheme Efficiency For The Bidomain Model .

When Monodomain Model Was Extended To Two And Three Dimensions, The Effect Of Anisotropy On Conduction Become Important. We Can't Ignore The Extracellular Conductivity. In Order To Model The Electrical Wave Propagation Inside The Heart, One Must Apply The Cable Equation In Both Extracellular And Intracellular Space. But Monodomain Model Describes The Current Flow Only In The Intracellular Regions Or Treat The Intracellular And Extracellular Conductivities Proportionally, Bidomain Model Considers The Current Flow In Both Spaces. Therefore Bidomain Model Is Considered As More Accurate Description Of The Electrical Wave Activities Than Monodomain Model. The Assumption Of Equal Anisotropy Is Not Supported By Experimental Measurements Of The Two Conductivities [33-34]. This Reduction In Physiological Accuracy Means That Some Physiological Phenomena Can't Investigate By Using Monodomain Model [35]. However This Reduction In Accuracy Leads To Significant Gain In Feasibility; The Computational Cost By Using The Monodomain Model Is About One Half To One Tenth The Cost Of Using The Bidomain Model, Depending On The Complexity Of The Cell Model Used [36]. Understanding The Functional Relationship Between The Discrete Structure And Continuum Behaviour Of Cardiac Tissue At Microscopic And Macroscopic Levels Is A Significant Challenge. Different Models Of Tissue Electrophysiology Involve Different

Assumptions And Simplifications, Yet There Is No Generally Accepted Framework For Choosing An Appropriate Combination Of Cellular Electrophysiology Model, Tissue Model, Geometrical Model, And Numerical Method.

V. Conclusion

In This Work, Dynamical Modelling Of Cardiac Electrical Activity Using Different Approach Was Presented. Apart From The Fact That This Work Has Been Able To Provide Some Insights Into The Electrical Behaviour Of Human Heart, Revealing The Nature Of The Electrical Wave Propagation Pattern In The Normal Cardiac Tissue, Models Of Cardiac Tissue Electrophysiology Have Played An Important Role In Advancing Our Understanding Of Action Potential Propagation In The Heart.

References

- [1]. O.I. Adebisi, I.A. Adejumbi, I.O. Abiala And S.O. Omotainse , Mathematical Modelling Of Cardiac Electrical Activity Using Bidomain Approach, Journal Of Computations & Modelling, Vol.2, No.3, 2012, 109-126, ISSN: 1792-7625 (Print), 1792-8850 (Online), Scienpress Ltd, 2012
- [2]. Durrer, D., Van Dam, R.T., Freud, G.E., Janse, M.J., Meijler, F.L., Arzbaecher, R.C., 1970.Total Excitation Of The Isolated Human Heart. *Circulation* 41, 899e912.
- [3]. Efimov, I.R., Nikolski, V.P., Salama, G., 2004. Optical Imaging Of The Heart. *Circulation Research* 286, H2183eh2194.
- [4]. Gray, R.A., Pertsov, A.M., Jalife, J., 1998. Spatial And Temporal Organization During Cardiac Fibrillation. *Nature* 392, 75e78.
- [5]. Henriquez, C.S., Papazogou, A.A., 1996. Using Computer Models To Understand The Roles Of Tissue Structure And Membrane Dynamics In Arrhythmogenesis. *Proceedings Of The IEEE* 84, 334e354
- [6]. Clayton, R.H., Panfilov, A.V., 2008. A Guide To Modelling Cardiac Electrical Activity In Anatomically Detailed Ventricles. *Progress In Biophysics & Molecular Biology* 96, 1943
- [7]. Nash, M.P., Bradley, C.P., Sutton, P.M., Clayton, R.H., Kallis, P., Hayward, M.P., Paterson, D.J., Taggart, P., 2006. Whole Heart Action Potential Duration Restitution Properties In Cardiac Patients: A Combined Clinical And Modelling Study. *Experimental Physiology* 91, 339e354
- [8]. Niederer, S.A., Smith, N.P., 2007. An Improved Numerical Method For Strong Coupling Of Excitation And Contraction Models In The Heart. *Progress In Biophysics & Molecular Biology* 96, 90e111.
- [9]. Ten Tusscher, K.H.W.J., Mourad, A., Nash, M.P., Clayton, R.H., Bradley, C.P., Paterson, D.J., Hayward, M.P., Panfilov, A.V., Taggart, P., 2009. Organization Of Ventricular Fibrillation In The Human Heart: Experiments And Models. *Experimental Physiology* 94, 553e562. Tentusscher, K.H.W.J., Noble, D., Noble
- [10]. Www.Vhlab.Umn.Edu, Atlds Human Cardiac Anatomy, University Of Minnesota
- [11]. List Of Blood Flow Through Heart Pump
- [12]. Deborah A. Jaye, Yong-Fu Xiao, And Daniel C. Sigg, *Basic Cardiac Electrophysiology: Excitable Membranes (Chapter 2)*
- [13]. O. Kittnar1, M. Mlček1, 1Institute Of Physiology, First Faculty Of Medicine, Charles University In Prague, Prague, Czech Republic *Review On Analysis Of Electric Field, Physiol. Res.* 59 (Suppl. 1): S19-S24, 2010
- [14]. Guyton And Hall Textbook Of Medical Physiology, W.B. Saunders Company, Philadelphia (1996)
- [15]. Ganong's Review Of Mmedical Physiology, M. R. Boyett, A. Clough, J. Dekanski, And A. V. Holden, *Modelling Cardiac Excitation And Excitability*, In A. V. Panfilov And A. V. Holden, Editors, *Computational Biology Of The Heart*, Pp. 1-47, John Wiley & Sons Ltd, Chichester, UK (1997)
- [16]. Prof. Roger G. Mark, Principles Of Cardiac Electrophysiology, Departments Of Electrical Engineering, Mechanical Engineering, And The Harvard-MIT Division Of Health Sciences And Technology, HST.542J: Quantitative Physiology: Organ Transport Systems Instructor: Roger Mark
- [17]. David Rosenbaum, B.H Smaill , P.J Hunter, *Quantitative Cardiac Physiology (Chapter 2)*
- [18]. Nico Kuijpers *Cellular Electrophysiology: Modeling And Simulation. (Chapter 2)*
- [19]. A. L. Hodgkin And A. F. Huxley, The Components Of Membrane Conductance In The Giant Axion Of Loligo, *J Physiol* 116: 473-495 (1952)
- [20]. A. L. Hodgkin And A. F. Huxley, Currents Carried By Sodium And Potassium Ions Through The Membrane Of The Giant Axon Of Loligo, *J Physiol* 116: 449-472 (1952)
- [21]. M.S. Shuaiby, A.H. Mohsen And E. Moumen, Modeling And Simulation Of The Action Potential In Human Cardiac Tissue Using Finite Element Method, *J. Of Commun. & Comput. Eng.*, 2(3), (2012), 21-27.
- [22]. Y. Belhamadia, Recent Numerical Methods In Electrocardiology, In: D.Campolo (Ed.), *New Development In Biomedical Engineering*, Retrieved September 5, 2011 From Intechopen: [Http://Www.Intechopen.Com/Book](http://Www.Intechopen.Com/Book), 2010
- [23]. Keener, J P, And Sneyd, J. *Mathematical Physiology*.Springer-Verlag, New York, 1998
- [24]. Keener, J P., Cytrynbaum, E. The Effect Of Spatial Scale Of Resistive Inhomogeneity On Defibrillation Of Cardiac Tissue. *Journal Of Theoretical Biology* 223(2003) 233-248
- [25]. B.J Roth, How The Anisotropy Of The Intracellular And Extracellular Conductivities Influences Simulation Of Cardiac Muscle, *J.Math. Biol* 30, 633 (1992)
- [26]. B.T Roth And J.P Wikswo, Electrical Stimulation Of Cardiac Tissue: A Bidomain Model With Active Membrane Properties, *IEEE Transactions On Biomedical Engineering* 41, 232 (1994)
- [27]. B.F. Nielsen, T.S Ruud, G.L.Lines And A.Tveito Optimal Monodomain Approximations Of The Bidomain Equations,*Applied Mathematics And Computation.* 184:276-290, 2007
- [28]. L. Tung. A Bidomain Model For Describing Ischemic Myocardial D-D Properties. Ph.D. Thesis, M.I.T., 1978.
- [29]. W. Krassowska And J.C. Neu. Homogenization Of Syncytial Tissues. *CRC Crit. Rev. Biomed. Eng.*, 21(2):137{199, 1993.
- [30]. P. Colli-Franzone And G. Savar_E. Degenerate Evolution Systems Modeling The Cardiac Electric Field At Micro- And Macroscopic Level. *Evolution Equations, Semi- Groups And Functional Analysis*, 2002.
- [31]. Y. Bourgault, Y. Coudi_Ere, And C. Pierre. Existence And Uniqueness Of The Solution For The Bidomain Model Used In Cardiac Electrophysiology. *Nonlinear Analysis: Real World Applications*, 10(1):458{482, 2009.

- [34]. Yves Bourgault, Charles Pierre, Comparing The Bidomain And Monodomain Models In Electro-Cardiology Through Convergence Analysis 2010. <Hal-00545888v2>
- [35]. J. Sundnes, G.T.Lines, X.Cai, B.F.Nielsen, K.A Mardal And A. Tveito. Computing The Electrical Activity In The Heart, Springer-Verlag, Berlin, 2006
- [36]. Sundnes, G.T.Lines, A. Tveito , An Operator Splitting Method For Solving The Bidomain Equations Coupled To A Volume Conductor Model For The Torso. Math. Biosci, 194(2):233-248, 2005
- [37]. Sundnes, G.T.Lines, X.Cai, B.F.Nielsen, K.A Mardal And A. Tveito. On The Computational Complexity Of The Bidomain And The Monodomain Models Of The Electrophysiology, Ann.Biomed.Eng., 34(7):1088-1097, 2006

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