Radio-Frequency Catheter Ablation for Treatment of Atrial Fibrillation: The Influence of Probe Contact on Impedance and Lesion Formation

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Radio-Frequency Catheter Ablation for Treatment of Atrial Fibrillation: The Influence of Probe Contact on Impedance and Lesion Formation

by

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled “Radio-Frequency Catheter Ablation for Treatment of Atrial Fibrillation: The Influence of Probe Contact on Impedance and Lesion Formation” submitted by Neal P. Gallagher in partial fulfillment of the requirements for the degree of MASTER OF SCIENCE.

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Abstract

Radio-frequency (RF) catheter ablation is a promising, minimally invasive treatment modality in the battle against the most prevalent cardiac arrhythmia, Atrial Fibrillation (AF). While it is known that catheter contact affects the size and shape of lesions produced during an ablation procedure, little has been done to quantify this effect. The purpose of this research was to establish a framework to determine the relationship between contact and lesion formation, and how contact can be assessed using electrical impedance.

First, an electroquasistatic computer model was created to approximate the Left Atrium (LA) and the complex impedance was calculated. It was found that impedance varies in proportion to catheter contact area; not just to penetration depth as popularly thought.

Finally, a thermal model was implemented. It was found that angle can significantly affect lesion sizes. Interestingly, lesion formation rates were highly dependent on catheter contact area while the transient response of the maximum temperature detected in the tissue was more dependent on catheter penetration depth.

Clinically, these discoveries are a step towards understanding the electrical and thermal behaviours that are inherent in cardiac ablation.
Keywords: Catheter ablation, atrial fibrillation, electrothermal coupling, biophysical modelling
Acknowledgements

The work presented in this thesis is the result of a monolithic effort put forth by many people. It is because of these people that graduate studies has been one of the most fulfilling and rewarding experiences of my life (with the exception being the preparation of this document)! I owe a huge debt of gratitude to so many people for their love, support and patience. Because of this, and the fact that I am quite sure that I can expound on the matter without the fear of ‘wrap it up’ music drowning me out, I would like to use the rest of this section to thank but a small fraction of them.

First, I would like to thank my supervisors Drs. Fear and Vigmond. It is because of your guidance, direction and firm prodding that, against all odds, I have been able to get through this with a body of work that I am quite proud of. Thank you so much Dr. Fear for taking a chance on me when even I was not sure that I deserved one. Your even hand, support, technical mastery and patience were invaluable to me as I battled through this experience. Dr. Vigmond, I really appreciate the time that you spent mentoring me and the fact that you certainly didn’t ‘spare the rod’! You are the best at what you do and provided me with constant motivation to excel. Working with two of the best that the world has to offer drove me to be better than I ever thought that I could be, constantly learning and improving. Thank you both
I would also like to thank Dr. Nygren for the consultations and outside perspective that he provided of my work on several occasions in the lab and at the lab meetings. Our clinical collaborator, Dr. Byrd was so helpful in providing information on technical issues and realities with catheter ablation as well as providing much of the experimental support.

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Finally, as any graduate student knows well, often the most difficult battle we wage is against the total loss of sanity itself. My (debatable) triumph in this battle
is due in no small part to the legion of really good friends that stood by me and it would be remiss not to thank you guys. Over the roughly three years that it took to complete this study, I have subjected you to an agenda that consisted heavily of two things: complaining and whining about academic life! Without the continuous support and provision of distractions by truly awesome people such as Matt Scrivens, Marshall Ross, Julia Sabey, Cheryl Graas, Danielle Vogt, Kate Sloan, Kate Johnson, Aunt Bibi, Uncle Tom, Miles and so many others, I am sure that I would be lost. Now I’m done.
Dedication

This thesis, and all of the hard work that went into it, is dedicated to my family:

Mom, you’re always there for me and you have always pushed me to work hard and excel at academics. Thank you so much for your support, I love you and will always be there for you, no matter what.

Dad, thank you for allowing me to live the grad student dream: to live with one’s parents while grinding it out! Seriously though, I will always appreciate how gracious you were during one of the most difficult periods of my life.

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<table>
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<th>Description</th>
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<tbody>
<tr>
<td>AF</td>
<td>Atrial Fibrillation</td>
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<tr>
<td>AV</td>
<td>Atrio-Ventricular</td>
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<tr>
<td>CFL</td>
<td>Courant-Friedrich-Levy</td>
</tr>
<tr>
<td>CT</td>
<td>Computed Tomography</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>FDTD</td>
<td>Finite Difference Time Domain</td>
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<tr>
<td>FEM</td>
<td>Finite Element Method</td>
</tr>
<tr>
<td>LA</td>
<td>Left Atrium</td>
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<tr>
<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
</tr>
<tr>
<td>PV</td>
<td>Pulmonary Vein</td>
</tr>
<tr>
<td>PVAI</td>
<td>Pulmonary Vein Antrum Isolation</td>
</tr>
<tr>
<td>RF</td>
<td>Radio-frequency</td>
</tr>
<tr>
<td>SA</td>
<td>Sino-Atrial</td>
</tr>
<tr>
<td>$\epsilon_0$</td>
<td>Permittivity of Free space</td>
</tr>
<tr>
<td>$\epsilon$</td>
<td>Permittivity</td>
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<tr>
<td>$\sigma$</td>
<td>Electrical Conductivity</td>
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<tr>
<td>$\rho$</td>
<td>Density</td>
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<tr>
<td>$k$</td>
<td>Thermal Conductivity</td>
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<tr>
<td>$c$</td>
<td>Specific Heat Capacity</td>
</tr>
<tr>
<td>$Gr$</td>
<td>Grashof Number</td>
</tr>
<tr>
<td>$Re$</td>
<td>Reynolds Number</td>
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<tr>
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<td>Rayleigh Number</td>
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<tr>
<td>$Pr$</td>
<td>Prandtl Number</td>
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<td>$Nu$</td>
<td>Nusselt Number</td>
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Chapter 1

Introduction

Atrial Fibrillation (AF) is often characterized by a rapid, chaotic fluttering of the upper chambers of the heart (the atria). Clinically it is the most common arrhythmia.\(^1\) It accounts for approximately one in three hospital admissions for cardiac disturbances\(^1\) and these rates are rising.\(^2\) One study estimated that approximately 6% of the population over the age of 65 has AF and the number approaches 10% in people over the age of 85\(^3\) while another more recent study conducted in Europe concluded that overall prevalence of AF was 5.5% in individuals aged 55-59 and 17.8% in those aged 85 years and above.\(^4\) Effectively, this sets the risk of a person currently aged 55 years at approximately 22% of developing AF within his/her lifetime! In terms of absolute numbers, this puts the numbers of individuals afflicted with AF at approximately 2.2 million in the United States\(^5\) (with similar rates in Canada) and 4.5 million in the European Union.\(^6\)

While AF itself is not typically a directly life-threatening arrhythmia, it is associated with increased risk of stroke (accounting for up to 24% of all ischemic strokes\(^7\)), heart failure and death. Patients often report weakness, anxiety (associated with rapid, irregular heartbeats), shortness of breath, faintness and low energy levels; not an ideal way for anyone to spend their golden years. Given that AF is such a prevalent and deleterious arrhythmia, more work must be done to attempt to curtail its effect
on the population; particularly with an aging population.

The most common forms of AF are thought to originate in the Pulmonary Vein (PV)s although, this is not necessarily the case for all. Current treatment options generally centre around pharmacological rate restoration and these have mixed results at best. Surgical options, such as the Maze procedure (and its implementation using catheter ablation), are considered secondary due to the high consequence, procedural difficulties and lower than desired success rates (53%-90% depending on the success metric used) although surgery provides the patient with the only real opportunity for a cure. Given that AF is such a serious and prevalent health problem there is a need to better understand the limiting factors of the less traumatic surgical option, catheter ablation, such that a greater number of AF cases can be safety cured.

Essentially, catheter ablation is a minimally invasive treatment modality wherein a catheter is advanced to a potential trouble spot in the heart (usually through a venous access point in the patient’s leg) then a Radio-frequency (RF) current is passed through the tissue. The RF current heats the tissue, in essence, "cooking" it so that it is electrically dead and offers isolation from the rest of the heart. This procedure is much less invasive, features quicker recovery times and is safer then the open surgery options. However, clinical studies in this field are full of well documented shortcomings mostly as a direct result from the highly sensitive area in which the procedure takes place, relegating any human clinical trials to binary pass/fail metrics without providing any precise reasoning for why any particular trial failed (or succeeded).\textsuperscript{8,9} Experimental studies abound but are fraught with technical difficulties ranging from repeatability to transferability.\textsuperscript{10-12} Biophysical modelling, on the other hand, provides the potential for cheap, non-invasive studies which can collect data in
ways that would be impossible in an experimental or clinical setting and have already established quite a foothold in the field of catheter ablation. Modelling however, is not without its own shortcomings and limitations: a model is only as reliable as the material properties and modelling decisions made (i.e.- modelling physics, geometry, discretization considerations). Deploying a set of simulation tools, a model of the atria can be constructed and precise experiments can be conducted by adjusting variables that govern that catheter’s interacting with the atria (which would be mostly unknown to a practising physician) and new insights can be gained. Given the precise control and repeatability that computer modelling offers when it comes to slight changes in catheter penetration depth and incident angle with the tissue surface, a unique opportunity is presented to finally investigate how catheter contact affects lesion formation during catheter ablation and whether or not contact can be inferred from measured electrical impedance.

1.1 Outline

This work provides the account of how the ablation catheter’s contact geometry affects lesion formation in a typical ablation procedure and proposes a method to evaluate this contact using simple measurements that are already being taken in a clinical setting. Chapter 2 provides an overview of the physiological systems at work responsible for the arrhythmia; both its genesis and sustainment. A more detailed problem description as it relates to the challenges of atrial fibrillation is presented as well as a summary of the state of the art of ablation research. After this in-depth analysis of the state of research in the field, remaining questions are posed and a strategy to address them is developed. Pursuant to this plan, chapter 3 presents the construction
process of the electrical model. The comparative merits of different modelling techniques are discussed and parameter selections are made. The results of the electrical simulations are presented and a simple contact assessment correlation is drawn. This process is repeated in chapter 4 for the solution of the thermal problem however given the extreme range of fundamental thermal parameters a range of possible solutions is explored. Chapter 5 involves collecting simple experimental data and relates results to chapter 3’s modelled solution of the electrical data, providing the beginnings of a validation framework for the model. Finally, chapter 6 presents the salient points and contributions to the field of this work and develops ideas for future research projects based on it.
Chapter 2

Background and Motivation for Work

2.1 Cardiac Physiology: A Brief Overview

The human heart is a wonderfully complex machine and its perpetual operation is a fundamental requirement for life. It adapts equally to increases in activity rate and prolonged periods of inactivity, all the while fulfilling its role of supplying oxygenated blood to the body's multitude of tissues and organs. Simply, the heart is a muscle-bound pump that simultaneously operates two sub-systems.

The heart is a muscle, however is has complex structure, depicted in Figure 2.1. We can see the complex construction and the three main layers that are found in a representative slice of cardiac tissue.\(^\text{13}\)

1. **Endocardium**: the innermost layer of tissue that lines the chambers of the heart.
   
   It is a soft tissue composed of endothelial cells similar to those that line blood vessels.

2. **Myocardium**: the fibrous muscular tissue responsible for the heart’s forceful contractions.

3. **Epicardium**: the outermost layer of tissue on the heart. This tissue acts as a
Figure 2.1: Tissue layers in the atrium. Reprinted with permission from Dr. Allan F. Wiechmann, Department of Cell Biology, University of Oklahoma Health Sciences Center.

A protective layer and lubricates the heart during the pump cycle.

While seemingly complex, the systems interact in perfect harmony in order to provide a nourished, lubricated environment for the powerful muscle to operate.

The two sub-systems, as seen in Figure 2.2, are the oxygenated (red) and non-oxygenated pathways (blue). Non-oxygenated blood from the body enters the right atrium through the superior and inferior vena cava. From there, the right atrium contracts, forcing blood into the larger right ventricle; this process is known as atrial systole and is shown in Figure 2.3(a). The next step is ventricular systole, shown in Figure 2.3(b), where the right ventricle contracts and propels the blood to the lungs via the pulmonary arteries.

Once inside the lungs, blood flows into the alveolar sacs where carbon dioxide is taken from the blood and oxygen is infused. The freshly oxygenated blood returns
The Pulmonary Vein (PV)s are connected at opposite ends of the Left Atrium (LA). The second of previously described sub-systems is the bigger, stronger and more significant left (oxygenated) system. Simultaneously, during the aforementioned diastole period, the LA contracts and forces blood into the immensely powerful left ventricle. Systole then follows and blood is expelled from the heart via the aorta, providing oxygenated blood for every tissue, structure and organ throughout the body. Every single human being is somewhere along this cycle every single moment of his or her life, rhythmically repeating itself over and over again.
2.1.1 The Cardiac Action Potential

The behaviour described in the previous section is the macroscopic result of billions of cardiac cells working in lockstep, each responding to an electrical stimulus at a prescribed moment in order to contract and expand the tissue as needed to pump blood throughout the body. This macroscopic action is the summation of complex behaviours on a cellular level. Cardiac cells respond to a gradient in membrane voltage in a highly non-linear way: for each cell there exists a threshold below which, the cell will simply return to resting levels. Above this threshold however, an action potential occurs and the cell performs its function (i.e.- contracting myocyte).

Normally, and as shown in Figure 2.4, there are five distinct phases of each action potential:\textsuperscript{14}

1. Phase IV is the cell at rest. The voltage at which the cell rests, typically 90 mV in normal myocardial cells, is called the resting membrane potential. The
cell will stay at this rate until it receives an electrical stimulus either internal (automaticity) or external.

2. Phase 0 is known as depolarization. Cells undergo depolarization only if the membrane voltage is raised above the threshold. It is because of this phase (and its steep slope) that rapid propagation of electrical activity occurs throughout the tissue.

3. Phase I is first stage of repolarization (a rapid repolarization). This phase is responsible for the notch in the action potential curve which is caused by the transient imbalance of potassium an chlorine ions.

4. Phase II constitutes the pleateau in Figure 2.4. It is the longest of all phases.

5. Phase III is last phase before the cell is restored to its resting state. It is the rapid repolarization of the cell.

While an in depth ion-level explanation of the action potential cycle is beyond the
scope of work for this project, it is important to understand a few properties inherent to cells. Namely, that each subsequent cellular stimulation greater than the threshold doesn’t necessarily elicit the same response. This is particularly true when the stimuli arrive in short succession of one another; this introduces the idea that there is some recovery period required to generate another action potential. This period, defined as the interval within which it is impossible to elicit another action potential no matter how large the applied stimulus may be, is known as the absolute refractory period.

It is necessary to grasp these concepts at some level to better understand on a macroscopic level how electrical activity is propagated throughout the heart. Also, the idea that some tissue will be in its refractory period while adjacent tissue has fully recovered and is ready to be stimulated lays somewhat of a basis for how otherwise healthy tissue can host (and sustain) nefarious arrhythmia.

2.1.2 Sinus Rhythm

The normal rhythmic nature described, also known as sinus rhythm, is made possible by very precise timing provided by the heart’s built-in conduction system. Shown in Figure 2.5, the heart’s conduction system comprises of a few main elements:

- Sino-Atrial (SA) node: The SA node is the heart’s normal intrinsic pacemaker.
- Atrio-Ventricular (AV) node: The AV node acts as gatekeeper for electrical activity propagating to the ventricles.
- Bundle of His, right and left bundle branches and Purkinje fibres.

Located in the upper part of the right atrium (specifically the sulcus terminalis) the SA node acts as a pacemaker and provides the initial impulse that begins the cardiac cycle. This impulse spreads through the interatrial tract concurrently depo-
Figure 2.5: Illustration of conduction system showing primary pathways of electrical activity in the heart. Reproduced with permission from CEUfast. Taken from http://www.ceufast.com/courses/viewcourse.asp?id=239, 2012.

Depolarizing the myocardium in the left and right atria in only 80-100ms. The depolarizing wavefront is responsible for the physical contraction of the myocardium that follows in its wake. This wavefront propagates to the AV node where there is a slight delay before the impulse is transmitted through to some specialized conductive fibres beginning with the bundles of His. These bundles branch out and bifurcate into the left and right ventricles of the heart. The right and left bundle branches conduct the impulse until the distal end where Purkinje fibres penetrate all along the myocardium and serve as the activation sites for the beginning of the ventricular depolarization wavefront. The ventricles then depolarize in perfect synchrony as the wavefront propagates from apex to the top of the ventricles causing a contraction that is similar to wringing a towel.
Figure 2.6 depicts the familiar image of a simple Electrocardiogram (ECG) depicting sinus rhythm, where each label (P,Q,R,S,T) corresponds to a different part of the cardiac electric cycle. The low amplitude, leading P-wave corresponds to the depolarization of the left and right atria. This is followed by a brief time lapse known as the PR interval which reflects the amount of time required for atrial depolarization and transmission through the AV node. Not surprisingly, the large amplitude QRS-complex can be measured as a result of the rapid depolarization of the ventricles. This is followed by another time lapse, the QT-interval, characterized as the amount time for complete ventricular depolarization and repolarization. The final part, the T-wave corresponds to the rapid repolarization of the ventricles.

Figure 2.6: Schematic diagram of an ECG depicting sinus rhythm. Taken from http://en.wikipedia.org/wiki/Electrocardiography, 2012.
The conduction system itself is wonderfully complex: it is regulated simultaneously by the body’s sympathetic (autonomic) and para-sympathetic nervous systems in perfect lockstep in order to answer any demand that we put on our bodies. Like any machine however, there can be complications that cause decreased efficiency or output but by and large, the heart is an incredibly resilient organ with a multitude of systems in place to protect it when things begin to go awry; this is when things begin to get complicated!

2.1.3 Arrhythmia: Atrial Fibrillation

Arrhythmia simply refers to any activity in the heart that deviates from basic sinus rhythm shown in Figure 2.6. Arrhythmia range from totally harmless to catastrophic; In fact, it is completely normal for every person to experience the occasional skipped beat or extra forceful beat. Atrial Fibrillation (AF) is an extremely common arrhythmia and Figure 2.7 shows a simple schematic of its presentation.

It is clear from the sample ECG in Figure 2.7(b) that there is chaotic electrical activity between QRS complexes; it is this disorganized electrical activity that characterizes AF. In general, there are four main mechanisms for clinical cardiac arrhythmias:\textsuperscript{15}

1. Reentry
2. Automaticity
3. Abnormal anatomic structures
4. Triggered activity

Reentry can take place due to a number of factors but it is dependent on the existence of a unidirectional block or alteration of a conducting pathway (such as infarction or
(a) Schematic diagram of atrial arrhythmia.

(b) ECG of a heart undergoing Atrial Fibrillation. Note the arrows which depict a P-wave in the normal ECG (bottom) and is obscured in the ECG with Atrial Fibrillation (top).

Figure 2.7: Schematic diagram of atrial arrhythmia. Figure 2.7(a) reprinted from Nature Reviews Drug Discovery, Vol 4, Page, Richard L.; Roden, Dan M., Drug therapy for atrial fibrillation: where do we go from here?, 899, Copyright (2005). ©Nature Publishing Group. Figure 2.7(a) taken from http://en.wikipedia.org/wiki/Atrial_fibrillation, 2012.
peri-infarct region), as well as precise timing of the redirected wavefront toward the recovering tissue which is to some extent a function of the length of the refractory period of the tissue. Essentially, for reentry to occur there needs to be an irregular source and a substrate.

Inferable from the electro-physiology background previously presented, the precise timing of the prevailing depolarization wavefront is crucial for coordinated contraction of the heart. Automaticity refers to modifications by disease, disorder or pharmacological that cause depolarization induced automaticity without any external influence. It is typically observed under conditions of reduced resting membrane potential such as ischemia or infarction and is another way of referring to premature ectopic beats originating outside of the SA node that are capable of initiating self sustaining electrical activity. The gradient in refractoriness creates a ‘window of vulnerability’ for the tissue to be re-excited, opening the door for self-sustaining electrical activity. While it is somewhat difficult to distinguish from other mechanisms, Haissaguerre et al. showed the AF can be triggered by rapid automaticity arising in the pulmonary veins.

Abnormal anatomic structures that result in accessory electrophysiological connections are most commonly associated with ventricular arrhythmia although the crista terminalis has been associated with atrial flutter. Either way, these abnormal structures vary on a case to case basis and have little bearing on this study.

The final dominant source of arrhythmia is triggered activity. Triggered activity refers to any beat that is generated by an oscillation that follows a cardiac action potential. These oscillations depend on transmembrane activity and are referred to as afterdepolarizations. If an afterdepolarization occurs with sufficient amplitude to
bring a membrane to its threshold then a spontaneous action potential is triggered.\textsuperscript{19} If this suprious electrical activity occurs with the atria as a substrate then either an ectopic beat (skipping a beat or irregularly or asynchronously contracting) or, more seriously, self-sustaining reentrant activity occur that renders the tissue non respondent to sinus beats since it is refractory at that moment.

AF, as defined by a World Health Organization task force on the matter, is characterized by “an irregular, disorganized, electrical activity of the atria”.\textsuperscript{20} In Figure 2.4 we can see that a coordinated depolarization of the atria results only in a small amplitude ‘bump’ known as the P wave; this is obviously absent during AF. Instead it is replaced by a chaotic and irregular waveform (as seen in the ECG shown in Figure 2.7(b)). Physically, the choatic activation causes irregular contraction of the atrial myocytes causing the atria to quiver. Prolonged exposure to AF causes electrical and structural remodelling which, due to AF may predispose a patient to more AF.\textsuperscript{21} Atrial flutter is very similar to the above mentioned symptoms except that it has a very regular rate, while AF is totally chaotic; however, atrial flutter often degenerates into AF. The main arrhythmogenic mechanisms that are responsible for AF are automaticity and macro (or micro) reentry (although triggered activity is also suspected).\textsuperscript{15}

There is considerable overlap in the symptoms and factors that are inherent to AF. In 2007 an expert consensus document\textsuperscript{8} was compiled by the foremost practitioners in the field which proposed that there are three main types of AF and that the following definitions of these types be used in studies of AF ablation (such as this one):

1. **Paroxysmal AF**: Defined as recurrent AF ($\geq$2 episodes) that terminate spontaneously within 7 days.
2. Persistent AF: Defined as AF that is sustained beyond 7 days, or less than 7 days but requires pharmacologic or electrical cardioversion (restoration of normal rhythm or rate using drugs or electricity). Included in this category are the cases called “longstanding” persistent AF which is defined as continuous AF of greater than 1 year duration.

3. Permanent AF: Defined as AF in which cardioversion has failed or not been attempted. These cases are not relevant to this study because it generally refers to patients in which the decision was made not to attempt to restore sinus rhythm for whatever reason by any means.

The ostia of the pulmonary veins (opening of the veins to the atrium) have been identified as a common source of premature triggering foci for both paroxysmal AF and atrial flutter. Although any of the previously mentioned mechanisms can induce arrhythmia, the premature ectopic beats originating in the pulmonary veins are thought to be the most common cause of paroxysmal atrial fibrillation. The mechanisms of persistent AF are thought to be more complex, potentially originating in the autonomic ganglionated plexi (autonomic nervous sites) but similar to treatment of paroxysmal AF, the procedure usually includes isolation of the pulmonary veins.

2.1.4 Anatomy of the Left Atrium: Implications for Atrial Fibrillation

Despite being the focus of procedures hoping to correct atrial arrhythmia, the LA is generally considered simpler than the right atrium and correspondingly, precious little is known about it. After a clinical trial in the late 90’s showing that 94% of ectopic foci originated in the pulmonary veins, much more attention was paid to the LA and its anatomic idiosyncrasies. In this same study, it was then found that the
earliest recorded activation sites were 2 to 4 cms distal to the venoatrial junction; it was this finding that lead to the concept that muscular sleeves actually surround the veins, a feature that had previously been overlooked. These findings lead to further study of the LA but from the viewpoint of an electrophysiologist; with the foci located somewhere in the PVs, Pulmonary Vein Antrum Isolation (PVAI) is clearly a good place to start in a curative therapy.

The left atrium can be described as having four distinct regions:

1. Septum: The region located between the left and right atria. It contains the oval foramen, the structure through which the catheter passes during the transseptal puncture from the right to left atria. Characterized by fairly smooth tissue.

2. Appendage: A pouch extending from the side of the atrium that contains an array of pectinate muscles; it acts as a reservoir for the LA. In AF cases it is common for blood clots to form in the left atrial appendage in more than 90% of cases. Obviously this is a serious problem as these clots can dislodge and be pumped throughout the body causing stroke and other embolism.

3. Vestibule: The vestibular component is a smooth, thin circumferential area that continually tapers until it terminates at the mitral valve.25

4. Venous: The largest part of the atrium, where the the pulmonary veins are received. This wall is generally smooth and is of varied thickness: from under 2mm to over 6.5mm, with the superior wall being the most substantial. There is a sleeve of myocardium that surrounds the proximal portion of the veins but its thickness varies from heart to heart and vein to vein.25

While Figure 2.1 would imply that the heart is comprised of thick walls, structures
Figure 2.8: A, View of the posterior wall of the LA. Note the light shining through the thinner parts of the structure. The epicardium has been removed and the orientation of the myocardial fibers can be seen in the walls. B, View of the posterior and inferior walls show the rapid transition in orientation of the myocardial fibers (arrows). An interatrial muscle bundle is present in this heart (double-headed arrow). C, Left side shows the myocardial attachment of the left superior (LS) and left inferior (LI) pulmonary veins. D, This view shows the identical myocardial attachment (muscle sleeves) between LS and LI veins. Left Atrial Appendage (LAA) can be seen in A&C. Reprinted with permission from Wolters Kluwer Health: S. Y. Ho, J. A. Cabrera, D. Sanchez-Quintana, *Left Atrial Anatomy Revisited*, Circulation: Arrhythmia and Electrophysiology *5* (2012), no. 1, 220-228.
near the vestibule (and throughout the LA) are nearly paper thin, therefore extra care
of accidental incision needs to be taken when operating anywhere near the area as
“closure may be virtually impossible\textsuperscript{26}”. These ultra thin structures exist in other
places in the LA as well: in Figure 2.8, light can be clearly seen shining right through
some of them! Adding to the complexity of the LA is the fact that only in a few areas
(such as the dome), do the fibres have the same orientation throughout the thickness
of the tissue. It is far more common for a sample thickness to have up to three distinct
'layers'. Another confounding factor is that there are bridges of muscular continuity
on the subepicardial surface which can provide the electrical continuity required for
sustained arrhythmic activity, despite linear isolation.

In the end, despite being considered simpler than those in the right atrium, the
structures of the LA present some unique challenges including (but not limited to):

- Walls of varied thickness ranging from paper thin (less than 2mm) to much
  thicker (greater than 6mm).
- Walls comprised of layers of myocardial fibres that are potentially oriented in
  much different directions.
- Abrupt spatial changes in fibre direction and wall thickness.
- Sections of trabeculated tissue.
- Subepicardial muscle bundles that can potentially bridge regions that would be
  targeted for linearly isolation.

In light of all of these factors we, as a scientific community, have our work cut out for
us in order to create a strategy that will safely and effectively cure AF despite the
unique challenges presented by this fascinating organ.
2.2 Radio Frequency Catheter Ablation and the Treatment of Atrial Fibrillation

Radio-frequency (RF) Catheter ablation is a remarkably powerful technique with applications ranging from curative cancer treatment in the liver to procedures of the cornea. Despite the wide range of applications,\textsuperscript{27} RF ablation is commonly used clinically in the heart, specifically for treating atrial fibrillation and flutter. Currently, catheter ablation is not considered as a first line therapy for AF.\textsuperscript{8} After the case has been deemed nonrespondent to at least one class I or class III anti-arrhythmic drug, catheter ablation is considered. There are some exceptions to this rule but as the efficacy of the procedure rises and the safety concerns are assuaged it will likely assume a more prominent role in treatment. This is of course due to an important distinction between the methodologies, as catheter ablation is potentially a cure while pharmacological intervention is perpetual and comes with its own serious side effects. Pharmacological intervention often allows AF to persist, seeking only to restore normal rhythm. In fact, one recent study showed that in patients with AF, RF ablation reduced the risk of AF recurrence by 65\% compared with anti-arrhythmic drugs.\textsuperscript{9}

Ablation in the atria and the unique challenges presented by its complex thin and trabeculated structures as well as the associated safety concerns will be the focus of this research.

2.2.1 Radio Frequency Catheter Ablation: The Procedure

During the standard procedure, an electrophysiologist advances the catheter through a venous access point into the patient’s right atrium through the vena cava. A trans-
septal puncture is made in order to access the LA. Figure 2.9 depicts a typical ablation procedure (although it shows a ventricular ablation rather than a PVAI. It is interesting to note both the access to the heart and surface electrode that serves as a return path for RF current. From there, the catheter tip (electrode) is positioned at the target endocardial site using an imaging modality such as fluoroscopy. Figure 2.10 shows an image from a fluoroscopy procedure. As is immediately evident from the image, there is extremely little soft tissue contrast available for the operator to landmark, locate and assess the contact that the catheter is making with the ablation site. Coupled with the amount of other hardware that must also be in the area (mapping catheters, sheaths, other imaging or ultrasonic devices), the complex anatomy of the atria, and the fact that the entire organ is continuously moving, it is clear that the procedure is incredibly complex. Once in place, an RF current is applied between the catheter electrode and a dispersive electrode which is placed on the surface of the patients body. This current heats the myocardial tissue and it
is thought that at temperatures in excess of 50°C, cellular necrosis occurs (caused by protein denaturation of the cellular membrane).\textsuperscript{28} That thermal induced injury occurs and causes irreversible loss of electrical conductivity is not disputed; however, the fact that there are transient recoveries from damage suggest that the amount of time spent above 50°C is also an important consideration.\textsuperscript{29} The goal of course is the permanent loss of electrical excitability, effectively eliminating or confining the focus of arrhythmic activity.

RF catheter ablation has rapidly gained popularity as a method of treating paroxysmal AF. In the case of atrial ablations, it is generally accepted that that the ectopic focus of paroxysmal AF is located in the PVs. Accordingly, to treat this arrhythmia, the physician performs pulmonary vein antrum isolation (PVAI) illustrated in Fig-
Figure 2.11: Illustration of Pulmonary Vein Antrum Isolation procedure. Note the presence of the ablation and lasso mapping catheters. Used with permission from the Cleveland Clinic. © Cleveland Clinic Journal of Medicine, 2009. 76(9):545. Taken from http://my.clevelandclinic.org/heart/atrial_fibrillation/, 2012.

 completely encircles the antrum of the vein. The operator must move the ablation catheter along the ostium of each vein, creating a contiguous lesion that encircles it. The goal is to provide full electrical isolation of the atria from the ectopic focus similar to the long, thin lesions created in the Maze procedure. Once the gold standard, the Maze (and Micro Maze) procedures are highly invasive surgical options to create the same isolation that is sought in ablation.
2.2.2 Conditions for Successful Procedural Outcomes

The success of this procedure is highly dependent on the precise positioning of the electrode as well as effective lesion formation. Lesion dimension is governed by the following factors:

- Catheter geometry\textsuperscript{11}
- Blood flow around the ablation site (cooling)
- Applied power
- Duration
- Preset temperature at the catheter tip (in temperature-controlled ablation)
- \textit{Catheter-Endocardial contact}
  - Penetration depth. Lesion depth is proportional to the electrode penetration depth.\textsuperscript{30} Lesion width changes significantly as penetration depth changes.
  - The angle between the catheter and endocardial surface also affects lesion dimension.\textsuperscript{31}

The success of this procedure is entirely dependent on full transmural lesion formation\textsuperscript{32} for electrical isolation. If too much energy is applied, there are the serious risks of perforating the atrium or creating an embolism.\textsuperscript{33} Of all the factors that influence lesion formation, local catheter-endocardial contact geometry (penetration depth and incident angle) is the one that is least well controlled due to a lack of soft tissue contrast in the fluoroscopy images. Despite the fact that unknown endocardial contact geometry is a well known limitation of the procedure, little work has been put into determining how lesion formation is affected by the incidence angle of the catheter. This omission seems even more glaring considering previous studies have
shown that there are significant differences in initial measured electrical impedance depending on catheter/tissue angle even at the same penetration depth.\textsuperscript{34}

Current techniques using fluoroscopy for positioning don’t provide sufficient soft tissue contrast to determine the penetration depth or contact angle. Instead, the electrophysiologist just pushes and rotates the catheter hard into the Endocardium in order to ensure adequate contact. Since this is currently such an inexact procedure, it is also very difficult to know how much energy is being deposited into the tissue and where the heat is accumulating. There are significant safety concerns associated with this uncertainty: should insufficient penetration occur and too much electrode contact with the surrounding blood is made, thrombi and steam can be produced, both which are potentially lethal. Catheters with built in temperature sensors have been attempted to curb this danger yet the convective cooling of the tissue-catheter interface often leads to readings that are falsely low, allowing for the unsuspected formation of serious hot-spots deeper in the myocardium.

Despite being significant, the contact that the catheter makes with the endocardial surface is one of the least studied and least well understood factors in catheterized ablation therapy. Specifically, the angle that the catheter makes with the endocardial surface affects the electrical coupling of the system and, in turn, lesion formation. This problem is particularly challenging in-vivo considering the incredibly complex three dimensional geometry and the fact that the chest cavity and atrial chambers themselves are constantly moving throughout the procedure. Given the complexity of the problem, there has been little progress in quantifying what makes for a successful ablation given realistic conditions. Clinical in-vivo studies tend to focus on endgame outcomes (procedures that need to be repeated vs. ones that are successful in termi-
nating AF on the first try) as opposed to technical realities (catheter angle and depth vs. electrical impedance and lesion formation) that have the potential to develop insights leading to positive clinical outcomes. In-vitro studies are not much more conclusive as it is often difficult to reproduce results as the complex anatomy and difficulty in taking measurements without destroying the sample make these experiments notoriously frustrating! As a result, an in-silico analysis of a simple ablation will be conducted in order to get a detailed understanding of how electrical impedance is related to catheter/endocardial surface contact.

Currently, electrical impedance is monitored but it is only used as an indicator that the procedure may have gone on too long and damaged some tissue. Impedance is a potentially powerful indicator of the electrical coupling between the probe and the endocardial surface. Unfortunately, direct measurement of electric fields is challenging from an in-situ standpoint, but the dynamics of the system can be carefully studied using an advanced computer model. RF ablation has long been an application that has seen extensive use of computer models in order to predict electrical and thermal performance.\textsuperscript{27} If the complex impedance of the system can be accurately calculated in a model then it should be possible to non-invasively correlate the complex impedance measured in an ablation procedure with known geometric parameters (catheter depth and angle).
2.3 State of Contact Assessment Research of Radio-Frequency Catheter Ablation

Considering the overwhelming amount of data illustrating the important effect that catheter/endocardial contact has on electrical coupling, naturally occurring cooling, heat accumulation, maximum temperature, the transient response of each and the resulting lesion formation (and so many others), precious little is available to the ablation practitioner in terms of contact assessment feedback. Instead, the procedure is undertaken by only the most experienced physicians with strict, conservative guidelines in place to attempt to maximize patient safety; in spite of all of this, first time procedural success rates are around 57% while major complication rates are around 5%. It is clear that there is room for improvement in this area, not only from a procedural success standpoint but also from a patient safety standpoint. One of the areas with the least amount of understanding is the contact evaluation which, in turn, makes this an interesting target for improvement.

In one of the more valiant efforts made to directly assess catheter/endocardial contact during RF ablation, Hong Cao et al. considered the difference in the resistivity of the blood in the atrium and the myocardium and used this difference to attempt to predict the resulting insertion depth of the catheter. To obtain a dataset the group performed in-vitro cow heart experiments and attempted to fit calibration curves using interpolation (or extrapolation) and considered both impedance ratio and impedance differencing methods. Their fit curves were essentially linear and extrapolation was then performed for points outside of the experiments conducted. The calibration curves are based on the data from a single tissue set at a particular
frequency therefore it was not surprising that some inconsistencies arose in the prediction accuracy in other catheter locations and tissue sets. In a real ablation procedure, compiling these results would not only be time consuming but also would vary from location to location. Another potential issue that was cited by the authors was having the tissue samples immersed in a saline solution as opposed to using actual blood. As a result, the frequency profile of the resistivity of the saline solution that was used as a blood substitute did not match that of blood for all frequencies tested. Given these shortcomings however, the study was quite good at predicting the catheter’s linear penetration depth. A main issue is also that the incident angle is only considered to be at 90° with the tissue, which given the imprecise nature of situating the catheter during the procedure, is a limitation given that the surface geometry of the atria is so complicated.

Another approach to identifying the catheter’s proximity to the tissue and perhaps assess to what degree contact is being made is to use electrical signals measured at the catheter tip. Conventional wisdom would dictate that the magnitude of an electrogram recorded from an ablation catheter indicates proximity to the heart tissue. However, it was found that, while electrogram amplitude was undoubtedly an indicator of macroscopic distances within the myocardium, electrograms are not at all reliable indicators of tip-tissue contact or even proximity to the tissue within a few millimeters. The hypothesis driving this study was that the phase angle shift is an effective predictor of tissue contact as the catheter begins to engage the tissue. Phase angle was declared ‘a much better indicator of tip-tissue proximity’ yet the correlation is still extremely poor. Overall, recovering the catheter’s depth from an electrogram amplitude and phase shift measurement was not found to be reliable. This result
is far from surprising as electrograms are much more susceptible to native electrical activity in the heart, pacing, noise and other measurement signals than impedance measurements.

In an interesting alternative to contact assessment using impedance measured at the catheter tip, another group focused on developing a novel method of impedance-based catheter tip-to-tissue contact assessment for use in left atrium ablations. This features a three terminal model designed to return the impedance of the catheter tip to the tissue.\(^{37}\) Unfortunately, the proposed method really only offered a binary metric to determine whether or not contact was made. It is well known that the degree of contact that the catheter has is in large part directly responsible for natural cooling achieved and, ultimately, the size and shape of the resulting lesion. Therefore, a method that returns a binary contact or no-contact result would be of limited significance given the importance of knowing the degree of contact especially given the multitude of other techniques available that would be able to provide a similar verdict. Also, the use of subjective terms such as no contact, light contact, moderate contact and firm contact are purely qualitative measures based on operator feedback. The in-vivo nature of this study makes it impossible to know if these contact settings actually refer to identical levels of catheter tip/endocardial surface contacts or levels that are even similar. Given that pairwise comparisons between these groups did not offer statistical significance, there are serious doubts with the methods. These kinds of concerns are most effectively addressed using in-vitro or modelling studies where contact can be precisely controlled.

From the previously cited studies, it is clear that catheter contact has a very significant (albeit difficult to assess and quantify) effect on measured impedance. However,
the impact that this difference in impedance has on end lesion dimensions has yet to be discussed. As this is such an important question, investigators have attempted many different solutions using alternative methods, such as catheter force measurement, to address it.\textsuperscript{50,51} If the catheter could measure the contact force when depressed in the tissue, then the operator would know whether adequate contact has been made. In one particularly interesting paper, the investigator attempted to correlate the end lesion dimensions during linear ablation with pre-ablation impedance measurements while controlling the catheter/tissue contact (as qualified by force) using in-vivo pig thigh preparations.\textsuperscript{36} Different contact conditions were created by varying the downward force on the catheter (no force, 30g for light contact, and 90g for firm contact). One of the findings of this study was that the measured impedance varied between animals, even at the same contact force. This is likely a manifestation of a common problem with force based contact assessment in ablation studies: while force sensing may provide additional feedback for catheter manipulation, force does not measure how well electrical energy is coupled between the catheter tip and the tissue. Subsequent experimentation at duplicate sites is potentially tainted due to pre-compression of the tissue; at very least, there exists the potential for significantly different catheter tip/tissue contact conditions. Despite the lower than expected correlation between contact force and lesion dimensions, the major finding of this study was that there is a significant correlation between the pre-ablation impedance and the dimensions of the linear lesion. This finding supports pre-ablation impedance assessment. In an actual ablation however, the applicability of pre-procedure impedance based contact assessment is perhaps limited given the complexity of this problem. The complex geometry and constantly moving anatomy make the real-world applicability of this correlation
tenuous at best. Nonetheless, it provides significant incentive to study the effect that catheter contact (assessed by measured impedance) has on lesion formation.

To further the study of catheter contact assessment, more must be know about how the catheter tip interacts with the myocardial surface. Given the inexactitude with which contact is made, a range of angles and depths are possible. In one study, the investigator examines the effect that electrode orientation has on lesion sizes produced by open and closed irrigated RF catheters. They compared lesions created by open irrigated, closed irrigated and non-irrigated catheters in porcine left ventricle preparations using a constant power ablation algorithm. The main findings of this paper were that:

1. That the lesion sizes that are produced by irrigated catheters are larger in a vertical orientation (catheter perpendicular to the tissue) than at horizontal orientation (catheter parallel to the tissue).
2. Increased irrigant flow reduces lesion volume in the irrigated catheters.
3. A larger area of tissue is actively cooled when the catheters are in the horizontal position compared to the vertical position.

While points 2 and 3 deal strictly with effects due to irrigation and cooling, the dominant factor in the size of lesions created by ablation catheters is the heating which is directly proportional to the current density distribution. This creates a major problem with this study as the catheters have greater length than width. Effectively, at the two configurations the catheters experience different contact areas which results in different impedances. The two scenarios aren’t electrically identical so the relevance of the findings is disputable, but one interesting point that this paper certainly raises is irregardless of the ablation method used, the most significant factor is the contact
(or orientation) with respect to the tissue.

Catheter contact, a known determinant of resulting lesion size, has been previously investigated but an appropriate solution to assess it still eludes researchers. Despite being an integral part of the contact puzzle, the role that catheter angle plays has also not been adequately considered; this is a perfect application for modelling. Some research groups have approached the solution in a totally different way: if they can use different techniques altogether (usually Magnetic Resonance Imaging (MRI)) to situate and assess final lesion dimension then electrical based methods aren’t required.

Given the extreme difficulty of situating the catheter given the lack of soft tissue contrast provided by fluoroscopy, more advanced techniques have been developed to precisely position the catheter. Systems like CARTO take a pre-procedure Computed Tomography (CT) scan to determine the precise structure of the atria and then using the feedback of stationary magnetic fields, the operator can determine the exact position of the catheter in heart. This system increases repeatability and has lead to the development of advanced imaging techniques to attempt either catheter contact assessment or post-hoc lesion definition. While this and similar techniques are potentially very accurate (< 2mm) they are limited by the physicians ability to exactly locate a corresponding anatomical location in the patient. This is further confounded by the continuous movement in the cardiac and respiratory cycles. Using a special technique known as delayed-enhancement cardiovascular magnetic resonance imaging (DE-CMRI) a modality was proposed to define the extent of left atrial wall injury after an ablation to treat AF. The study surmises that since RF lesions on the epicardium of dogs have been characterized using gadolinium enhanced MRI and right ventricular enhancement, and that these findings correlated well with histopathology
showing coagulation necrosis, further testing in humans should be done to ascertain the technique’s viability as a non-invasive manner of performing post procedural lesion assessment. This technique would be extremely valuable as it would provide the clinician with an immediate means of predicting whether or not the procedure will be a successful and whether or not each individual lesion is continuous and transmural. In addition to the fact that ablations are typically quite long in duration (which would then additionally require the use of an MR system) the main flaws with this theory are:

1. The assessment technique is purely visual; albeit visual according to the resulting spin physics of the altered tissue, there is little to say that the conductivity is necessarily zero in the pathway.

2. The voxel sizes were 1.25 x 1.25 x 2.5 mm and reconstructed to 0.625 x 0.625 x 1.25mm. This is a major problem considering that at its thickest, the wall of the left atrium is only 4mm. It is challenging to properly assess the size and transmurality of a lesion when there are potentially only 1-3 voxels for the entire depth of the structure that you are meant to be analyzing!

In addition to the ones described in detail, there are many other thermal electric modelling papers that explore how lesions form based on many different variables: catheter tip materials,\textsuperscript{48} adding a layer of epicardial fat,\textsuperscript{45} both endocardial end epicardial (see\textsuperscript{27} for a more complete list of papers) ablation but few of them evaluate the effect that different catheter depths has on the resulting lesion and none of them investigate how catheter angle may change the lesion size. While it is clear that there is quite a range of physiologically acceptable values that can be used in thermal simulations, these values are often non-linear with respect to temperature and
large changes in their values have been shown to cause huge variations in resulting lesion size from nominal values. It is unlikely that such huge variations in material properties would exist at the same site. Moreover, many of these changes would likely manifest themselves in changes in the electrical coupling between the catheter and tissue. At any rate, some value will have to be chosen for simulation and it is likely that in a clinical setting that material differences would simply be some kind of difference or ratio from the chosen values.

Simply put, given the known significance of catheter/myocardium contact there is a glaring hole in the field radiofrequency cardiac catheter ablation when it comes to being able to precisely quantify its effect. In essence, given a comprehensive background review, a few conclusions can be drawn as to the most logical way forward:

- Exact values as far as individual parameters go matter less than trends; exact values can vary from patient to patient and could largely be normalized using ratio or differencing techniques to assess electric coupling with tissue. In the end, what is most important is not being able to precisely predict from the measured impedance what the contact is but instead to have some insight into how heat will propagate through the tissue and how the lesion will form. The body of research surveyed has shown this idea to be more than reasonable therefore the most logical choice is to first gain a further understanding of how impedance responds to changes in catheter contact.

- It is extremely important to precisely control contact so that a true correlation between impedance and contact can be established. It is clear that by using contact pressure many different contact scenarios, all with potentially different electrical realities may exist. The inexactness of the methods used to date make...
this a particularly difficult correlation to obtain particularly since experiments can be notoriously difficult to control all of the required variables. Fortunately, this is a shortcoming that mathematical modelling does not have. Precisely and repeatably replicating trials is something that in-silico experimentation is particularly adept at and an advanced modelling study of the problem is undoubtedly a good place to start.

- While irrigation does lead to bigger lesions, it often over complicates the problem that is not fully understood right now: given that the way the catheter can contact the tissue is so unknown and uncontrollable, why is angle not being studied? Is lesion formation only affected by depth or also by contact angle? Does the complex part of the impedance measurement provide any supplementary information?

Given a clear path forward, a detailed computer modelling study of radiofrequency catheter ablation as it relates to the field of atrial fibrillation treatment will be carried out. The goals of this study are to assess how catheter impedance changes due solely to changes in the catheter’s depth in the tissue and the angle made with the tissue. From there, a continuation of the work will explore how the same differences in contact affect heat propagation and endgame lesion formation in modelled myocardial tissue.
Chapter 3

Computer Modelling of Electrical Problem

In an ablation procedure, the operator passes a current through the tissue with the goal of rendering the tissue permanently electrically dead. The success of this procedure has been found to be heavily dependant on the way that the catheter contacts the tissue; both the degree of penetration but also the angle that the catheter makes with the tissue. Given that this is essentially a problem dependant on the electrical coupling of the catheter with the tissue it should be possible to evaluate the the contact that the catheter is making with the tissue using a measurement already being taken by the practising physician: electrical impedance. The following chapter explores the relationship of measured electrical impedance with catheter/tissue contact and attempts to make a correlation between the two. The end goal, of course, is to be able to use measured impedance as a tool to tell the electrophysiologist when appropriate contact has been made.

3.1 Model

In order to effectively and accurately compute internal electric field values and current density distributions that occur during an ablation and are not easily measured in-
vivo, a computer model had to be constructed. The key considerations in the model are:

- Model structure (sizes, shapes and locations of the components of the model)
- Electrical properties of the individual model components
- Numerical considerations (Solver physics used, boundary conditions, gridding and voxelling)

To accomplish this task, SEMCAD’s (SPEAG, Switzerland) low-frequency solver was chosen. When constructing the physical model, the main goal was to implement structures that were geometrically and computationally simple while providing enough 'life-likeness' to be able to draw meaningful conclusions for practical situations. To accomplish this, many decisions and informed concessions had to be made in each important aspect of the model.

3.1.1 Model Geometry

With simplicity and authenticity in mind, the model is comprised of the following elements:

- Catheter Tip
- Catheter Body
- Blood Volume
- Tissue
- Dispersive Patch Electrode

The dimensions of the catheter itself were chosen based on St. Jude’s Therapy Cool Path™ 1300 Series Ablation Electrophysiology Catheter, shown in Figure 3.1. It is a standard unipolar ablation electrode and has dimensions of 7 French (2.33mm
diameter) and 4mm overall electrode length.

Figure 3.1: Image of St. Jude’s Therapy Cool Path\textsuperscript{TM} 1300 Series Ablation Electrophysiology Catheter, the ablation catheter being modelled. The small holes shown are for irrigant (coolant) flow. ©St. Jude Medical Inc., used with permission. Taken from http://www.sjmprofessional.com/, 2012.

The catheter body was modelled simply by extruding the catheter tip footprint to the edge of the simulation region.

The blood volume in which the entire model is immersed defines the simulation region. Despite the fact that the absolute impedance of the tissues and fluids on the epicardial side of the atrium is much different than that of blood, modelling this area as blood ends up being a decent representation since the resulting current densities decay exponentially as distance from the catheter tip increases. Two different blood volumes, and therefore solution spaces, were attempted: a first attempt at 30 cm x 30 cm x 15 cm and a second (halving each dimension) at 15 cm x 15 cm x 7.5 cm. The first volume was chosen to accurately represent a standard chest volume but also to ensure that the imposition of boundary conditions (i.e. zero flux) would be easily satisfied and not create numerical artifacts. The second, reduced volume was used
because it made the simulations run much more quickly and the results generated were consistent with those of the larger volume (refer to Sec. 3.1.4 on model space optimization).

The dispersive patch electrode is located near the boundary of the model in the Z-direction. It occupies the entire bottom (X-Y plane) of the solution space and is implemented as a surface, not a volume. Depending on the final size of the solution volume, the dispersive electrode may be larger than an actual electrode used in an ablation scenario but given the current distribution in the model, the electrode could be made much smaller before any significant impact is seen.

Finally, the tissue slab size, 20 cm x 20 cm x 4 mm in the first attempt and 10 cm x 10 cm x 4 mm in the second, was chosen so that it filled a good part of the available X-Y plane area. A smaller piece of tissue could be chosen in the future but the current size is desirable because it vaguely represents a large, flattened atrium. The final physical model is shown in Figure 3.2.

3.1.2 Electrical Material Properties

Once a good physical model was built, the time came to choose the key electrical properties that would, in large part, dictate the findings of the simulation. Knowing the dielectric properties of biological materials is a somewhat difficult task due to variability of properties with respect to patient, frequency, temperature, water content and several other factors. Fortunately, throughout the 1900’s scientists have been gathering dielectric property databases at broad frequency ranges for various applications ranging from discerning the resistivity of tissue to modelling electromagnetic dosimetry. Finally, in 1996 Gabriel et al. performed a literature review of available,
pertinent data, performed the required experiments to fill out datasets for important missing tissues at various missing frequencies and then fit a parametric model to the spectrum of dielectric tissue properties in what would prove to be one of the seminal works in the field. To facilitate the incorporation of discrete material property data points into models, a continuous formulation would be ideal to interpolate (or extrapolate) the data for the exact frequency required by the user. To accomplish this, they fitted multiple dispersion models such as the multiple Cole-Cole and the well-known Debye model to the experimental data. The model used to described the dielectric properties of both the blood and heart tissue was taken from this work and is described in Equation 3.1.
\[
\hat{\epsilon}(\omega) = \epsilon_\infty + \sum_n \frac{\Delta\epsilon_n}{1 + (j\omega\tau_n)^{(1-\alpha_n)}} + \frac{\sigma_i}{j\omega\epsilon_0}
\] (3.1)

This equation follows a well known representation of the dielectric spectra of biological tissues\textsuperscript{57} characterized by three main relaxation regions at low (\(\alpha\)), medium (\(\beta\)) and high (\(\gamma\)) frequencies. These three relaxation regions represent the polarization mechanism which is condensed into a single time constant, \(\tau\). The complexity of both the structure and composition of biological materials however, is such that each dispersion region may have several contributions to it. This is accounted for with the introduction of a distribution parameter \(\alpha\). \(\epsilon_\infty\) refers to the permittivity at field frequencies where \(\omega\tau \gg 1\) and \(\epsilon_s\) is the permittivity at which \(\omega\tau \ll 1\). Permittivity of Free space \((\epsilon_0)\), \(\sigma_i\) is the static ionic conductivity and \(\omega\) is the angular frequency. Finally, the magnitude of dispersion is \(\Delta\epsilon_n\). The model parameters for blood and heart tissue as reported in the Gabriel paper\textsuperscript{55} and used in this study are shown in Table 3.1.

The integration of these Cole-Cole model parameters show how significantly the tissue properties are changing in the frequency range considered (shown in Figure 3.3). As can be seen in Figure 3.3 and will be discussed at greater length later in this document, the values for both permittivity and conductivity are most different between blood and heart tissue at lower frequencies. Therefore, at 20kHz the range of impedance difference due to change in catheter penetration depth should be the greatest.

At the chosen frequencies, Permittivity \((\epsilon)\) and Electrical Conductivity \((\sigma)\) values are calculated. These values are used in the simulations and their values are listed in Table 3.2.
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Blood</th>
<th>Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\epsilon_\infty$</td>
<td>4.0</td>
<td>4.0</td>
</tr>
<tr>
<td>$\Delta \epsilon_1$</td>
<td>56.0</td>
<td>50.0</td>
</tr>
<tr>
<td>$\tau_1$ (ps)</td>
<td>8.38</td>
<td>7.96</td>
</tr>
<tr>
<td>$\alpha_1$</td>
<td>0.10</td>
<td>0.10</td>
</tr>
<tr>
<td>$\Delta \epsilon_2$</td>
<td>5200</td>
<td>1200</td>
</tr>
<tr>
<td>$\tau_2$ (ns)</td>
<td>132.63</td>
<td>159.15</td>
</tr>
<tr>
<td>$\alpha_2$</td>
<td>0.10</td>
<td>0.05</td>
</tr>
<tr>
<td>$\Delta \epsilon_3$</td>
<td>0.0</td>
<td>$4.5 \times 10^5$</td>
</tr>
<tr>
<td>$\tau_3$ ($\mu$s)</td>
<td>-</td>
<td>72.34</td>
</tr>
<tr>
<td>$\alpha_3$</td>
<td>-</td>
<td>0.22</td>
</tr>
<tr>
<td>$\Delta \epsilon_4$</td>
<td>0.0</td>
<td>$2.5 \times 10^7$</td>
</tr>
<tr>
<td>$\tau_4$ (ms)</td>
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<td>4.547</td>
</tr>
<tr>
<td>$\alpha_4$</td>
<td>-</td>
<td>0.00</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>0.700</td>
<td>0.050</td>
</tr>
</tbody>
</table>

Table 3.1: Parameters of equation 3.1 used to predict the dielectric properties of Blood and Heart tissue.

<table>
<thead>
<tr>
<th>Frequencies</th>
<th>20 kHz</th>
<th>485 kHz</th>
<th>2 MHz</th>
</tr>
</thead>
<tbody>
<tr>
<td>Property</td>
<td>$\epsilon_r$</td>
<td>$\sigma_r$</td>
<td>$\epsilon_r$</td>
</tr>
<tr>
<td>Tissue</td>
<td>37433</td>
<td>3332.3</td>
<td>1087.4</td>
</tr>
<tr>
<td>Saline (Blood)</td>
<td>5236.5</td>
<td>4227.1</td>
<td>1680.6</td>
</tr>
</tbody>
</table>

Table 3.2: Electrical Parameters used for simulations

3.1.3 Electrical Problem

Now that an acceptable physical model has been chosen, appropriate decisions must be made in order to make the electromagnetic solution space representative of the atria bioelectrically during an ablation.

Electro-quasi static Assumption

As in any electromagnetics problem, the first step is to consult Maxwell’s equations! Conducting an analysis of the solution space using the full blown time varying electromagnetic equations would be possible yet prohibitively expensive computation-
Figure 3.3: Multiple dispersion plot of Cole-Cole model of permittivity and conductivity for heart tissue and blood.

ally with an approach such as a fully coupled electromagnetic Finite Difference Time Domain (FDTD) solver. The problem with using FDTD analysis is that it is remarkably ill-suited for an ablation application. In a typical ablation, the frequencies used are all quite low with a typical ablation current having a frequency of approximately 485 kHz. In this study, frequencies of 20 kHz, 485 kHz and 2MHz were considered in order to see the effect that different frequencies in the lower RF range have on impedance-based contact assessment. When using such low frequencies, the ablation currents are associated with wavelengths that are quite large (∼15 km, 620m, 150m). This presents a problem for an FDTD solver because, in comparison, the dimensions of the ablation problem are typically in the sub-millimetre range. The reason that an
FDTD solver is *such* a poor fit for the intended application is this temporal/spatial incompatibility of the problem. For even a reasonable spatial resolution required to properly discretize the ablation electrode tip (covered in greater length in Sec. 3.1.4) the temporal step size used for updating in an explicit finite-difference scheme must be made correspondingly small according to the Courant-Friedrich-Levy Courant-Friedrich-Levy (CFL) criterion for continued numerical stability. For the FDTD formulation of Maxwell’s equations on a staggered grid, the CFL criterion is described by equation 3.2.

\[
\Delta t \leq \frac{1}{c \sqrt{\frac{1}{(\Delta x)^2} + \frac{1}{(\Delta y)^2} + \frac{1}{(\Delta z)^2}}} \tag{3.2}
\]

After appropriately setting the temporal and spatial step sizes, an FDTD solution requires a few complete wavelengths to be simulated before stability is reached and at an extremely small step size this would required an infeasible amount of computing time. Herein lies the problem, how to solve the solution space without sacrificing spatial resolution?

Since the dimensions of the problem are so small relative to the wavelengths used in typical ablation procedures, the electroquasistatic assumption was tested for use. The idea is that the electroquasistatic approximation is valid if an electromagnetic wave can propagate the entire characteristic length of the system in a time that is much smaller than a period of the wave, something that seems reasonable for the given application. Equation 3.3 describes the conditions for the Electroquasistatic assumption to hold.
\[ |\gamma \ell| \ll 1 \quad \text{where: } \gamma = [(i\mu_0 \omega) (\sigma + i\epsilon \omega)]^{1/2} \] (3.3)

Where \( \gamma \) is the medium propagation constant, \( \ell \) is the characteristic length of the physical problem, \( \mu_0 \) is the magnetic permeability, \( \omega \) is the angular frequency, \( \sigma \) is the conductivity and \( \epsilon \) is the permittivity. Testing this assumption with the frequency range used in RF ablation we see that the maximum gap between the catheter and dispersive electrode for the electroquasistatic assumption to hold, at 2 MHz (our maximum tested frequency), is approximately 23.87 m which is a much greater distance than anything we would encounter in an ablation scenario!

The advantage of using this assumption is that it allows the decoupling of the Electric and Magnetic fields by setting the time varying magnetic field to zero. This assumption drastically simplifies the computation required and allows us to go from a FDTD solution to a low frequency, frequency domain Finite Element Method (FEM) solution. Another advantage is that it makes no assumptions regarding the relative magnitudes of the conduction current \( \sigma E \) and displacement current \( i\epsilon \omega E \).^58

**Electrical Model Space Solution**

Now that the analysis method has be chosen, it is prudent to cover what exactly will be solved! After decoupling the time varying component of the magnetic field, Faraday’s law simplifies to:

\[ \nabla \times E = -\frac{\partial B}{\partial t} \quad \rightarrow \quad \nabla \times E = 0 \] (3.4)
This simplification allows us to neglect the circulating electric field created by our time varying magnetic field. The advantage of this simplification is that by simply manipulating Gauss’s law (eqn. 3.5), a solution can be had for the entire problem space without worrying about the magnetic field’s contribution to the electric field (and later, current).

\[ \nabla \cdot \mathbf{D} = \rho_v \quad (3.5) \]

There is no deposited electric charge in the model as we hope to model the effect that the RF current has in the tissue space so the \( \rho_v \) term is neglected. Rearranging equation 3.5 we arrive at the final form shown in equation 3.6.

\[ \nabla \cdot \tilde{\varepsilon} (-\nabla \phi) = 0 \quad \text{where:} \quad \tilde{\varepsilon}_r = \varepsilon_r \varepsilon_o - j \left( \frac{\sigma}{\omega} \right) \quad (3.6) \]

Initial conditions in the form of affixed potentials are applied to the dispersive patch electrode (ground) and the catheter tip (supply voltage). What follows is the solution of a a complex valued Laplacian problem.

**Complex Impedance Calculation**

Once an appropriate model was constructed, the next step was to accurately calculate the current. There are several potential options to compute the impedance but the chosen method included calculating the displacement current as shown in Equation 3.7

\[ I_{enc} = j\omega \int_S \tilde{\varepsilon}_r \mathbf{E} \cdot dS \quad (3.7) \]

This was the best solution, given the options, because there was no averaging of field values and the electric field values are easily obtained. Once current has been
solved for, the complex impedance can easily be found. Equation 3.8 outlines how the complex impedance is calculated from the previously computed current.

\[
Z = \frac{V_{\text{catheter}} - V_{\text{plate.electrode}}}{I_{\text{enc}}} = \frac{V_{\text{catheter}} - V_{\text{plate.electrode}}}{\int_S \sigma E \cdot dS + j\omega \int_S \varepsilon \varepsilon_0 E \cdot dS}
\] (3.8)

The strength of this approach is visible in equation 3.8 wherein the denominator we can see current contributions from both the conduction current and displacement current. Both are important to consider in a model, since it is essentially a parallel plate capacitor that is constantly charging and discharging.

A surface is required to solve for the current components and the surface is chosen to be just a few voxels above the dispersive patch electrode (in the +z direction) and extend to the full extents of the solution space in x and y (as shown in Figure 3.4).

![Figure 3.4: Model showing location of Gaussian integration surface.](image)

**3.1.4 Numerical Considerations**

The final aspects to consider for accurate modelling are the numerical ones. The decisions on boundary conditions and grid density are perhaps among the most important
as they are responsible for proper representation of the problem.

**Boundary Conditions**

Generally, there are two main boundary conditions that are implemented for partial differential problems such as this one:

- Neumann "no flux" boundary conditions
- Dirichlet fixed potential boundary condition

The Neumann zero flux boundary condition is described by equation 3.9.

\[
\frac{\partial \Phi(r)}{\partial n} = 0 \quad r \text{ on } S \tag{3.9}
\]

Essentially, it describes any system where the normal derivative of \( \Phi \) vanishes at the boundary \( S \) of the solution region \( R \). This fixes a constant flux at the edge of the problem.

A Dirichlet boundary condition affixes a constant value for \( \Phi \) along any particular boundary. For a ground plate, the potential is fixed to 0V. This boundary condition is slightly more simple than a Neumann boundary condition but it requires that that potentials naturally be at (or near) the fixed value otherwise the problem will suffer badly from numerical artifacts.

Given our solver type (Low frequency FEM), relatively large saline volume, and low frequency ablation currents, it would probably be acceptable to implement either boundary condition. However, the Neumann zero flux boundary condition is a better choice for the boundaries because it would be extremely unlikely for there to be a non-zero flux value so far away from the catheter tip. Although unlikely, a potential other than zero could exist near the boundary and it is imperative to choice an
appropriately sized solution region to ensure that this does not occur.

**Gridding and Model Size Optimization**

The last numerical issue, gridding and voxelling, proved to require the most refining. The catheter tip can be modelled as a cylinder with a hemisphere on the end. The hemisphere is the shape that is most difficult to voxelize (especially if the catheter is held at an angle) because the curvature of the sphere is extremely complicated to represent using cubic elements in a regular grid. In order to most accurately voxelize the catheter tip so as to minimize resulting numerical artifacts in the simulation, it was found that the best approach was to combine three methods: refine the mesh at the tip of the catheter, allow for non-uniform grids and use a local gridding solution wherein the grid used to voxelize the catheter tip need not necessarily correspond to the prevailing grid used in the rest of the model (where a much more sparse grid would suffice). This being said, the challenge still remains to minimize the problem appropriately so that the problem space is not excessively large (leading to long solution times) or sparse (leading to numerical artifacts and erroneous solutions).

The first simulation set was run with a large solution space (30 cm x 30 cm x 15 cm) and used an extremely fine discretization strategy. This point serves as a kind of reference/baseline for any subsequent attempts at a solution. From there, the model was made successively smaller (in x, y and z dimensions) in order to reduce the overall model size as much as possible while keeping a fine discretization strategy. This strategy obviously has a profound impact on reducing the number of voxels but is limited by the potential for source/boundary interactions if the model space is made too small. Once the model was reduced to half of the original size (15cm x 15cm x 7.5cm) a few different discretization strategies were investigated, each using fewer
voxels than the original half size version. In order to evaluate the deviation of the solution based solely on discretization, two different cases were studied for all of the voxelization strategies discussed:

1. Catheter penetration depth of 1.6mm and incident angle of 90°. This serves as a kind of simple test, the catheter is normal to the grid therefore the curvature of the hemisphere will be most easily rendered. The results of this study are shown in Figure A.1 and Table A.1.

2. Catheter penetration depth of 3.2mm and incident angle of 15°. This case is much more difficult to render due to the misalignment of the catheter and the prevailing grid. It serves as a kind of worst case scenario for any voxelization scheme. The results of this study are shown in Figure A.2 and Table A.2.

As can be observed in both Figures A.1 and A.2 the calculated impedance doesn’t change very much as the model is made smaller and discretization is kept very fine. Also, as more intelligent means of reducing model discretization are employed (BR1,2,3) but any reduction in how the catheter tip was resolved contributed to large changes in calculated impedance. The worst schemes, RD6 (where the tip discretization was reduced by 60%) and NL (where no local gridding scheme was employed) were pretty horrific; these scenarios yielded the highest observed discrepancy in impedance from the finely voxelized versions. Nowhere are the differences more startling than when these changes are inspected visually, as in Figure 3.5.

In the end, after reducing the model to half of its original physical dimensions it was decided to keep the model as finely discretized as the original full size version for several reasons:

- Simulation time was drastically increased. From approximately one hour with
Figure 3.5: Comparison between voxelling schemes of the ablation catheter with catheter at 3.2mm penetration depth and 15° incident angle.

- The calculated impedance hasn’t significantly diverged from the full size model (or any subsequent steps) and it was deemed important to keep the tip resolution as fine as possible because it is the most sensitive parameter in the analysis.

- It was important to keep the spatial resolution inside of the tissue higher than it absolutely needed to be in order to have sufficient modelling of the tissue for the thermal simulations that will follow. Of particular import was to have sufficient points in the Z direction (tissue depth) to accurately define how deep the resulting lesion of a particular configuration may be. If the tissue was discretized as sparsely as possible for the electrical simulation, it is possible for there to have been only a few nodes between the top of the tissue surface and the bottom; not nearly enough resolution for effective characterization of any lesion.

Accordingly, the grid labelled 'Half Size' and catheter tip depicted in Figure 3.5(a) are to be used for the rest of the work.
3.2 Simulation Set

Given the complex physical geometry ever present in a living biological structure such as the left atrium, it is obvious that an ablation catheter could be incident upon its surface in any number of ways. Therefore, to investigate ablation in the atria sufficiently thoroughly, an appropriately large number of geometric realizations must be chosen and these simulations run.

The impedance is said to jump by several ohms when the catheter first makes contact with tissue. In fact, exploitation of this phenomena has been proposed as a method to determine when the catheter is at a desired location,\textsuperscript{3710} and is followed by further insertion of the tip into the tissue. As such, the first set of points required for study is when the catheter is in the blood pool and some distance away from the atrial tissue slab altogether. This first point serves as a sort of reference as it is the only time in practise that the catheter will contact only a single medium. The next data points are collected as the catheter begins to contact the tissue. As this is an imprecise concept, ‘just touching the tissue’ is taken to mean when the catheter is at 90° and the catheter tip contact is 1%. This configuration therefore has a catheter penetration depth of 0.04mm, defined as the point on the catheter most deeply penetrating the tissue. From here, the depth is fixed and the catheter/tissue angle is then varied in 15° increments from 90° (catheter normal to the tissue) to 15° (nearly laying down on the tissue) as shown in Figure 3.6. To complete the simulation set, catheter tip/tissue contact area percentages of 20%, 40%, 60%, 80% and 100% were chosen and all corresponding depth values were calculated when the catheter is at 90° with the tissue.

The complete simulation list is in increments of contact area. Percentages are
Figure 3.6: Catheters at different angles. Penetration depth shown = 1.6mm

listed in terms of the area of the catheter tip touching the tissue and are:

- 4 mm above the tissue
- 1% contact (calculated as 0.04mm depth at 90°)
- 20% contact (calculated as 0.8mm depth at 90°)
- 40% contact (calculated as 1.6mm depth at 90°)
- 60% contact (calculated as 2.4mm depth at 90°)
- 80% contact (calculated as 3.2mm depth at 90°)
- 100% contact (calculated as 4mm depth at 90°)

At each depth, independent simulations were run at 15° increments between the tip perpendicular to the tissue (90°) and parallel to the tissue (0°). At each depth and angle different frequencies were also considered. Table 3.3 shows the variables with combinations from which the complete simulation set is formed.

<table>
<thead>
<tr>
<th>Frequencies</th>
<th>Depths</th>
<th>Angles</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 kHz</td>
<td>-4 mm</td>
<td>15°</td>
</tr>
<tr>
<td>485 kHz</td>
<td>0.04 mm</td>
<td>30°</td>
</tr>
<tr>
<td>2 MHz</td>
<td>0.8 mm</td>
<td>45°</td>
</tr>
<tr>
<td></td>
<td>1.6 mm</td>
<td>60°</td>
</tr>
<tr>
<td></td>
<td>2.4 mm</td>
<td>75°</td>
</tr>
<tr>
<td></td>
<td>3.2 mm</td>
<td>90°</td>
</tr>
<tr>
<td></td>
<td>4 mm</td>
<td></td>
</tr>
</tbody>
</table>

Table 3.3: List of Required Simulations
Such an exhaustive set of simulations is required in order to give the best chance of identifying trends in impedance as depth, angle and frequency are changed. Unfortunately, each simulation requires approximately 20-30 minutes to setup, between 15 minutes to an hour to run (depending on discretization) and another hour to extract the important data and analyze it, bringing the total time per simulation to approximately one to two and a half hours. While this may be acceptable for a few simulations, there are 126 simulations required! In order to streamline this process and minimize the machine’s downtime and the time that it actually needed human supervision, SEMCADs python interface was taken advantage of. After writing a set of scripts, much of the above-mentioned work could be done automatically without manual intervention. A brief description of the scripts follows:

- The first script sets up the model based on three simple input parameters: catheter depth, angle and frequency. List of the input files are generated so that the transfer of these files to the high performance computing cluster could be optimized.

- The second script takes the list of input files corresponding to the simulations setup and runs the solver for each simulation listed. The results are returned and catalogued.

- The third script opens the results files, once they have been returned by the cluster, and extracts the required data fields and returns them in easily usable Matlab data arrays.

- The fourth script accesses each array and then executes two Matlab based utilities that I wrote to compute the impedance and the contact area. All of the data is analyzed and returned in a table that contains all of the important
parameters.

Once all scripts have finished running, all that is left to do is assemble the massive amounts of data generated and compile it in a sensible way!

## 3.3 Results

### 3.3.1 Impedance vs. Catheter Penetration Depth and Angle

Although figures 3.7, 3.8 and 3.9 are quite busy, there is a significant amount of important data to analyze. To begin with, there is a clear trend of increasing impedance magnitude, increasing resistance being the principal driver, with increasing catheter depth. While this is a finding that has been known for sometime, the more interesting point is that for a particular penetration depth and frequency, we can see that there is a significant difference in resistance and reactance that is due solely to the catheter contact angle. Also, the effect that the catheter angle has on impedance seems to be proportional to the increase in penetration depth. This is perhaps a puzzling finding but becomes clear when you consider that at deeper penetration depths, a simple rotation of the catheter causes quite a difference in the amount of the catheter’s surface that contacts the tissue (this finding is further discussed in Section 3.3.3). Given that the tissue is of lower conductance than the blood the greater the contact with the tissue, the more current forced through the tissue and the greater the resulting resistance (and impedance magnitude). From these results, we can infer that the problem essentially boils down to a simple case of two resistors in parallel (shown in Figure 3.10) where based on the proportion of the catheter that is engaged with either the tissue or the blood, the current pathways change to reflect the difference
(a) Resistance and Reactance as a function of catheter penetration depth at 20 kHz.

(b) Impedance Magnitude and Phase as a function of catheter penetration depth at 20 kHz.

Figure 3.7: Plots of complex impedance quantities vs. catheter penetration depth at 20 kHz.
(a) Resistance and Reactance as a function of catheter penetration depth at 485 kHz.

(b) Impedance Magnitude and Phase as a function of catheter penetration depth at 485 kHz.

Figure 3.8: Plots of complex impedance quantities vs. catheter penetration depth at 485 kHz.
(a) Resistance and Reactance as a function of catheter penetration depth at 2 MHz.

(b) Impedance Magnitude and Phase as a function of catheter penetration depth at 2 MHz.

Figure 3.9: Plots of complex impedance quantities vs. catheter penetration depth at 2 MHz.
It is also important to observe that there is an exponentially increasing trend of impedance with depth that holds everywhere except at 4mm penetration depth. That is likely explainable by the fact that that tissue slab modelled was only 4mm thick. At maximal penetration depth, the increase in impedance associated by being fully engaged with the tissue is likely balanced out by the closer proximity of the catheter to the dispersive electrode and to the blood pool. This theory is further tested in section 3.3.5.

Given that the resistance is the principal driver of the measured impedance in the system and the reactance (or phase shift) is a component that is often overlooked, it would be prudent to study its effect given the opportunity to do so. In Figures 3.7(a), 3.8(a) and 3.9(a) it is observed that the reactance decreases almost as a mirror image to the increase in resistance (albeit at a much smaller magnitude). This change can be explained by the small increase in capacitance due to the catheter being in greater contact with the higher permittivity issue. Unfortunately, this change, while measureable, is of much smaller range than that observed in the resistance and offers virtually no additional information. Practically speaking then it
makes sense to only consider the impedance magnitude as it is easier to measure and has greater ”dynamic range” making changes easier to detect.

3.3.2 Impedance vs. Frequency

Another interesting, yet predictable finding from figures 3.7, 3.8 and 3.9 is that not only is the calculated impedance magnitude and phase smaller at higher frequencies but the angle related difference in impedance magnitude and phase is also smaller at higher frequencies. This can no doubt be explained by Figure 3.3 wherein we see that the conductivity increases in both blood and tissue at higher frequencies which would cause the principal component of the impedance (resistance) to decrease at the higher frequencies. More complex however is the relationship between the two materials at increasing frequencies. At higher frequencies, the conductivity and permittivity of blood and tissue begin to approach each other. This is reflected in Figure 3.9 in the smaller depth and angle related change in impedance at 2MHz. While it is known that passing the kinds of large currents required to ablate the tissue at low frequencies (such as 20 kHz) interferes with the heart’s natural rhythm, directly causing arhythmia, it is beneficial to using tiny ”sensing” currents at low frequencies to attempt to evaluate the contact as there is the electrical properties of the blood and atrial tissue differ the most at low frequency. It is also obvious from Figures 3.3 and 3.7, 3.8 and 3.9 that this case lends itself well to an impedance spectroscopy study and that would certainly be a priority in any future work.
3.3.3 Impedance vs. Catheter Contact Area

In order to fully understand the drastic change in impedance at deeper penetrations depths that can only be attributed to the change in catheter incidence angle observed in section 3.3.1, the area of the catheter in contact with the tissue was computed and plotted against impedance as shown in Figures 3.11, 3.12 and 3.13. The plot clusters are linked by penetration depth. Here we see a clear exponential connection between impedance (all of its constituents) and the catheter contact area. Once again, the only data grouping that bucks the trend is the subset at 4mm penetration depth. It is important to observe that, despite being at different penetration depths and angles, the points with similar contact area percentages had virtually identical impedances. This theory is further tested in section 3.3.4 where several permutations of depth and angle are tested in order to get roughly the same contact area %.

3.3.4 Impedance vs. Contact Area Percentage when Contact is held Approximately Constant

Figure 3.17 shows that for the set of catheter angles and penetration depths shown in Table 3.4, specifically chosen to maintain an approximate catheter contact area percentage while changing angle or depth, the impedance stays quite constant. Table 3.4 shows the impedance values calculated when the catheter’s angle and penetration depth are varied but the contact area percentage is held relatively fixed at 20 kHz. It is plain to see from the table and the figure that there is very little difference in the impedance (at most 7Ω). This implies that for a given catheter/endocardial surface area contact, the impedance that would be measured by the physician would be relatively similar. This is potentially clinically relevant because it would allow the
(a) Resistance and Reactance as a function of catheter/tissue contact area percentage at 20 kHz.

(b) Impedance Magnitude and Phase as a function of catheter/tissue contact area percentage at 20 kHz.

Figure 3.11: Plots of complex impedance quantities vs. catheter/tissue contact area percentage at 20 kHz.
(a) Resistance and Reactance as a function of catheter/tissue contact area percentage at 485 kHz.

(b) Impedance Magnitude and Phase as a function of catheter/tissue contact area percentage at 485 kHz.

Figure 3.12: Plots of complex impedance quantities vs. catheter/tissue contact area percentage at 485 kHz.
(a) Resistance and Reactance as a function of catheter/tissue contact area percentage at 2 MHz.

(b) Impedance Magnitude and Phase as a function of catheter/tissue contact area percentage at 2 MHz.

Figure 3.13: Plots of complex impedance quantities vs. catheter/tissue contact area percentage at 2 MHz.
(a) Resistance and Reactance as a function of catheter/tissue contact area percentage with a thick tissue slab at 20 kHz.

(b) Impedance Magnitude and Phase as a function of catheter/tissue contact area percentage with a thick tissue slab at 20 kHz.

Figure 3.14: Plots of complex impedance quantities vs. catheter/tissue contact area percentage with a thick tissue slab at 20 kHz.
(a) Resistance and Reactance as a function of catheter/tissue contact area percentage with a thick tissue slab at 485 kHz.

(b) Impedance Magnitude and Phase as a function of catheter/tissue contact area percentage with a thick tissue slab at 485 kHz.

Figure 3.15: Plots of complex impedance quantities vs. catheter/tissue contact area percentage with a thick tissue slab at 485 kHz.
(a) Resistance and Reactance as a function of catheter/tissue contact area percentage with a thick tissue slab at 2 MHz.

(b) Impedance Magnitude and Phase as a function of catheter/tissue contact area percentage with a thick tissue slab at 2 MHz.

Figure 3.16: Plots of complex impedance quantites vs. catheter/tissue contact area percentage with a thick tissue slab at 2 MHz.
(a) Resistance and Reactance as a function of catheter/tissue contact area percentage.

(b) Impedance Magnitude and Phase as a function of catheter/tissue contact area percentage.

Figure 3.17: Investigating how impedance parameters are affected when depth and angle are changed but the catheter/tissue contact area is held roughly constant.
operator to have an immediate assessment of the contact that the catheter is making in the atrial chamber.

3.3.5 Impedance vs. Catheter Penetration Depth and Angle for a Thick Tissue Slab

Figures 3.18, 3.19 and 3.20 and 3.14, 3.15 and 3.16 show the effect that changing catheter penetration depth and angle has on impedance when the tissue slab considered is thicker (10 mm as opposed to 4 mm). While there is no physiological reason for studying such a thick tissue slab, as the thickness of human atrial tissue is no thicker than 4mm, this study is interesting because it sheds some light on the deviations from the smooth, increasing exponential trend seen in all of the datasets at all depths other than 4mm penetration depth. In this figure, we see that there is nothing special about 4mm penetration depth (no effect due to the fact that all of the metal surface of the catheter is now engaged with the tissue) other than penetration effects that manifest themselves as a plateau in the impedance readings. Physiologically, this would be an extremely relevant finding as a physician could observe the impedance increasing and subsequent plateau with increased contact pressure as a sign that the catheter has nearly penetrated the atrial wall.

3.4 Summary of Electrical Modelling Results

Indisputably, characterization of the contact made between an ablation catheter’s tip and the endocardium during an ablation procedure is an extremely important remaining challenge. Despite a few attempted in-vitro experimental attempts it remains a challenge that is largely unaddressed due to technical complexity. In this chapter, we
(a) Resistance and Reactance as a function of catheter penetration depth with a thick tissue slab at 20 kHz.

(b) Impedance Magnitude and Phase as a function of catheter penetration depth with a thick tissue slab at 20 kHz.

Figure 3.18: Plots of complex impedance quantities vs. catheter penetration depth for a thick tissue slab at 20 kHz.
(a) Resistance and Reactance as a function of catheter penetration depth with a thick tissue slab at 485 kHz.

(b) Impedance Magnitude and Phase as a function of catheter penetration depth with a thick tissue slab at 485 kHz.

Figure 3.19: Plots of complex impedance quantities vs. catheter penetration depth for a thick tissue slab at 485 kHz.
(a) Resistance and Reactance as a function of catheter penetration depth with a thick tissue slab at 2 MHz.

(b) Impedance Magnitude and Phase as a function of catheter penetration depth with a thick tissue slab at 2 MHz.

Figure 3.20: Plots of complex impedance quantities vs. catheter penetration depth for a thick tissue slab at 2 MHz.
have presented a method using a computer based model that precisely controls the incident catheter’s penetration depth and angle. Exploiting the repeatability of this computer model we were then able to evaluate the relationship between impedance parameters, that even now are being measured by ablation practitioners, and parameters that describe the catheter’s contact with the tissue. From these studies several extremely important conclusions can be drawn:

- Resistance is the dominant contributor to the measured impedance. Changes in reactance are small and are a mirror image to changes in resistance; meaning that in a sensing capability they don’t provide any additional information for
characterizing the catheter contact.

• Impedance magnitude increases exponentially with increasing penetration depth. This effect is truncated if the catheter is close to penetrating the tissue. As the penetration depth becomes close to the thickness of the tissue, the impedance begins to "round off" and even decay as the catheter becomes closer to the saline pool and dispersive patch electrode. If the tissue is thicker, the impedance will continue to increase exponentially as penetration depth increases.

• Impedance magnitude decreases as the frequency of the current increases. Also of note, the change in impedance due to catheter interaction with the tissue decreases as frequency increases.

• At deeper penetration depths, the effect that changing the catheter angle has on impedance magnitude increases. This increase is attributable to larger changes in contact area at deeper penetration depths.

• Impedance parameters are directly proportional to catheter contact with the tissue. The particular combination of catheter depth and angle required to attain a particular contact percentage is largely unimportant; if the contact is the same, the impedance will be the same.

In the end, the research conducted in this chapter provides some extremely valuable insight into how contact might be assessed by the practitioner. It is known that the measured impedance is directly proportional to the catheter contact area and that higher impedance means that a higher percentage of the catheter is in contact with the tissue meaning that the coupling between the catheter and the tissue has also improved. In the next chapter, we will be investigating whether or not this increased contact with the tissue manifests itself thermally when the ablation begins.
Chapter 4

Computer Modelling of Thermal Problem

In order for the results presented in the previous chapter to be truly pertinent, the analysis must come full circle and consider what implication a given catheter contact has on how heat propagates through the tissue and how the resulting lesion forms. Obviously, if a lesion forms independently of the measured electrical impedance (which would be highly suspicious), then knowing the contact would be largely irrelevant. Therefore, the logical next step for this research is to consider: how a given catheter contact geometry (angle and depth) affects lesion formations? Again, this is a relatively straightforward question but one that has quite a few hurdles to overcome before any reasonable conclusions can be drawn. This chapter will cover the description of these challenges as well as the ways in which they have been addressed.

4.1 Thermal Model Selection

The problem posed in a typical endocardial ablation is thermally quite complex due to the anatomy in which the procedure takes place. The radiofrequency currents cause direct heating in the tissue which, through conduction, propagates throughout the atrial wall. Inside the atrium there is significant blood volume that is continuously
exchanged causing variable amounts of convective cooling. This is variable because many patients with atrial fibrillation have greatly reduced ejection fraction or other physical changes to the atria that cause their hearts to not beat efficiently. On the epicardial side of the atrial wall there is a different situation altogether where varying amounts of fat and a thin layer of pericardial fluid contribute to a different conduction/convection boundary altogether.

Since the problem is so complex, the proper choice of bio-heat models is paramount. Careful consideration must be given to the model’s ability to resolve the temperature distribution spatially as well as temporally. When modelling biological heat transfer, there are two main approaches:\textsuperscript{59}

- The Vascular Model: Blood vessels are taken into consideration as small tubes buried in the the considered tissue. Generally, only the major vessels are considered, since representing each capillary would be extremely costly computationally and would not likely yield any significant difference for this application.
- The Continuum Model: Continuum models treat the considered tissue region as if there are no blood vessels present. The effect of blood flow in the tissue is averaged and treated using an additional perfusion term in the conduction equation. These models are obviously simpler to use than vascular models as they do not require complex approximations of vascular networks; information that may not be known. Obviously, this simplification also presents a limitation as the potential for vast differences in blood flow do exist as the concentration of vessels and rate of blood flow in a given tissue varies spatially.

The vascular model approach is mainly used in applications where focal temperature non-uniformities due to the presence of large blood vessels in the region of interest
are important (ablation of tumours in the liver). In the case of the thin walled left atrium, due to the lack of large vessels at the ablation sites, the dominant effects will be the convection due to the blood volume in the atrium and the conduction within the tissue. Therefore, due to its simplicity and applicability, a continuum model is chosen.

Having chosen the type of model to represent the conductive heat flow throughout the tissue, the set of governing equations required to satisfy the model must be selected. Generally in biomedical heat transfer applications, and particularly as it concerns Radio-frequency (RF) ablation, the Pennes bio-heat equation (a parabolic bio-heat equation where the heat conduction term is based on Fourier’s theory) is employed. However, in 2008 J.A. López Molina et al. presented an alternative to the parabolic bio-heat equation, proposing instead a hyperbolic version. At the crux of the matter is the fact that the parabolic bio-heat equation assumes infinite thermal energy propagation speed, which may not be valid for all application of RF ablations (particularly those with very short heating times). Effectively, the hyperbolic equation introduces a tissue specific relaxation constant that takes into account the time delay between heat flux and temperature gradient. The group found that there was some difference between the two approaches, particularly in cases where knowing the temperature rise within the relaxation time of the tissue was important. However, the steady state temperatures in both methods were quite similar. Due to the lack of experimentally valid data on relaxation properties of cardiac tissue and the fact that for the majority of scenarios considered in this study a long time course will be used, a steady state type solution will likely be sufficient. Therefore, the parabolic Pennes bio-heat equation will be implemented.
4.1.1 Pennes Bio-heat Equation

In order to solve the thermal problem and get the resulting temperature distribution map, the Pennes bioheat equation (equation 4.1) was solved everywhere in the tissue.

\[ \rho c \frac{\partial T}{\partial t} = \nabla \cdot (k \nabla T) + q - Q_p + Q_m \]  

(4.1)

\( \rho \) is a tissue’s density, \( c \) is its specific heat capacity, \( k \) is its thermal conductivity and \( q \) is the source term from the RF power deposition; the selection of these quantities will be discussed at greater lengths in sections 4.1.2 and 4.1.5 respectively. The values chosen for the heat loss due to blood perfusion in the myocardial tissue \( (Q_p) \) and the metabolic heat generation rate \( (Q_m) \) terms will be discussed at greater length in section 4.1.4.

The premise underlined by this equation is that the time dependant rate of temperature change is a function of the spatial gradient of the temperature distribution in the tissue, the heat deposited during the ablation, the natural metabolic and perfusion processes and the very properties of the tissue itself. This clearly covers the thermal conduction within the tissue aspect of the problem but there remains one more issue to cover: how is the convective cooling on the blood-endocardium interface accounted for? Without it, the temperature inside the tissue would certainly accumulate far quicker than found clinically, therefore, the careful consideration that this problem requires is covered in 4.1.3.
4.1.2 Material Properties

As with most heat transfer problems, the accuracy of the analysis is often limited by the accuracy of the material constants chosen and the assumptions made about them. This section will describe the rationale behind the choice of each parameter.

Density

The Density ($\rho$) of both the tissue and the blood factor prominently into the solution of the thermal problem. Unfortunately, density depends on a myriad of factors not limited to temperature, pressure, water volume of the sample, body posture etc. In the myocardial tissue for example, water accounts for approximately 77.8% of the tissue per unit volume.\textsuperscript{61} This undoubtedly will have some impact as the tissue begins to heat and water concentration changes (more significant at extremely high temperatures such as the temperatures towards the upper limit of this study). Common values used for the density, $\rho$, of myocardium tissue range from 1060 kg/m$^3$ to 1200 kg/m$^3$ while those of whole blood range from 1000 kg/m$^3$ to 1060 kg/m$^3$ at 37°C. Due to an important discovery of the dependence of blood density on body posture,\textsuperscript{63} the measured density in the supine position, 1047 kg/m$^3$ will be used.

Specific Heat Capacity

Specific Heat Capacity (c), is another important parameter for the model. Simply put, specific heat capacity refers to the amount of heat required to change the temperature of the material by a single degree. It also is temperature, pressure and water volume dependant. Common ranges for specific heat capacity in myocardium are 3111 J/Kg K to 3600 J/Kg K while in blood the range used is from 3760 J/Kg K to 4180 J/Kg K.
Thermal Conductivity

Thermal Conductivity ($k$) is a parameter that shows up throughout the model, and as the name would imply, is a measure of a material’s ability to conduct heat. It is particularly difficult to model because of the strong temperature dependance of this parameter. Generally, when tissues are warmer than $10^\circ C$ it is prudent to represent $k$ as:

$$k = k_0 + k_1 T$$  \hspace{1cm} (4.2)

For instance, in a study of swine myocardium it was found that the thermal conductivity went from 0.5367 W/m K at $37^\circ C$ to 0.5833 W/m K at $76^\circ C$.\(^{42}\) Most models however implement constant thermal conductivities for simplicity. Common ranges for thermal conductivity in myocardium are 0.512 W/m K to 0.7 W/m K while in blood the range used is from 0.492 W/m K to 0.543 W/m K.\(^{41}\)

The material properties chosen for use in this study are listed in table 4.1.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>$\rho$ (kg/m$^3$)</th>
<th>$c$ (J/kg K)</th>
<th>$k$ (W/m K)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>Chosen</td>
<td>Range</td>
</tr>
<tr>
<td>Atrial Tissue</td>
<td>1060-1200</td>
<td>$1063^{64}$</td>
<td>3111-3600</td>
</tr>
<tr>
<td>Blood</td>
<td>1000-1060</td>
<td>$1047^{63}$</td>
<td>3600-4180</td>
</tr>
<tr>
<td>Catheter Tip</td>
<td>-</td>
<td>$21.5 \cdot 10^3$</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 4.1: Thermal material parameters used in simulations. Catheter tip material used is Pt-Ir.

4.1.3 Convection in the Model

The largest and most important form of natural cooling that is present during endocardial ablation is the convective cooling that occurs due to the blood volume inside
the atria. The convective cooling described manifests itself in two ways:

- Forced convection caused by the interaction of the catheter tip that is exposed to the flowing blood in the atrium.
- Forced convection that occurs on the interface between the endocardial surface that is exposed to the blood in the atrium.

This cooling is generally considered helpful in an ablation procedure because the RF current density is highest in the area immediately surrounding the catheter tip (as shown in Figure 4.1); without any cooling, the temperature around the tip would spike so quickly that it would not have time to propagate deeper into the tissue. The endocardial surface would likely burn very quickly resulting in the formation of dangerous coagulum and char. The lesions formed in this scenario would be too shallow to provide useful electrical isolation. As a result of this, catheter makers have experimented with increasing the size of catheters\textsuperscript{11,39,43} and artificially cooling the interface using different irrigation techniques,\textsuperscript{12,43,66} with quite a bit of success. The key finding was that the more the interface is cooled, the deeper the heat is able to propagate, resulting in deeper lesions. In light of these findings, for any thermal model to meaningfully represent the propagation of heat in a realistic manner, it is obviously important to properly account for both types of convective cooling that occur naturally. While the field of RF catheter ablation as a whole has gone to some form of irrigation, be it open (irrigant being pumped through open holes at the distal end of the catheter tip to directly cool the tissue) or closed (where the irrigant is recirculated inside the catheter and does not directly exchange fluid with the outside environment), this study will focus on how heat created by resistive heat of the RF currents propagates and how lesions form in the natural environment (without
irrigation and subject to natural convection). The decision was made to limit the analysis to this because it is uncertain how catheter contact (which in practice can be largely unknown) affects heat flow in the tissue.

In a thermal model, a convective boundary condition is generally implemented anywhere there is a change in medium. At an endocardial ablation site, convection occurs at the boundaries between tissue and liquid. There are two such interfaces in the model (illustrated in figure 4.2):

1. $h_{elec}$: Convection occurring between the ablation catheter tip and the blood volume.
2. $h_{tiss}$: Convection occurring between the tissue surface and the blood volume.

So now the problem becomes quite evident: high current densities near the tip of the electrode cause high localized temperatures in the tissue. The resulting tem-
Figure 4.2: Sample ablation model illustrating the two convective interfaces that exist.

Temperature gradient will eventually conduct deeper in the tissue if given the proper opportunity to do so. Heat will also conduct extremely quickly in the catheter tip and the amount of cooling caused by the tip will be some function of the resulting temperature gradient between the tip and the blood pool and the exposed tip area. This relationship is mathematically represented in equation 4.3.

\[
-k \frac{\partial T}{\partial x} \bigg|_{x=0} = h \left( T \bigg|_{x=0} - T_\infty \right) \tag{4.3}
\]

On the conduction side of the equation, \( k \) is the thermal conductivity of the solid (either tissue or Platinum Iridium) and \( x \) refers to distance travelled in a simplified one-dimensional representation of the model \( (x = 0^- \text{ being just on the solid side}) \).
of the interface while $x = 0^+$ would be just on the liquid side of the interface).

On the convection side of the equation, $h$ is the convection film coefficient and the subtraction (in brackets) refers to the temperature difference between the interface surface temperature ($T_{\mid x=0}$) and the temperature of the blood ($T_\infty$). The rate of heat loss due to the convective interface is then implemented as a boundary condition (shown in equation 4.4).

$$q_{1-\infty} = hA(T_1 - T_\infty)$$ (4.4)

Where $q_{1-\infty}$ is the heat flux from region 1 to the boundary, $h$ is the film coefficient, $A$ is the surface area of the interface between regions and $T_1 - T_\infty$ is the temperature difference between the considered region and the boundary bulk temperature.

While it would seem that all the parts to the puzzle are finally together at this point, further investigation reveals that this could not be further from the truth! In fact, there is much debate about how to effectively calculate or measure the convection coefficient $h$, and as a result, a large range of convective film coefficients have been used. This uncertainty is for any of several good reasons:

- Blood flow in the atria is pulsatile and turbulent.
- The convective film coefficient at the ablation site is variable and difficult to measure.
- In a normal, healthy heart, the blood inside the left atrium is rhythmically exchanged at sinus rate as this blood volume is pumped into the left ventricle, leading to a functional pre-stretch of the ventricle. Patients that have undergone prolonged bouts of paroxysmal AF or persistent AF often show significant remodelling of the chambers frequently with blood flow to the body decreasing between 15% and 30%. Therefore, the blood flow rate and overall exchange
rate is highly variable between patients, trials and even individual ablation sites. Accordingly, as some value needs to be taken for $h$ in order to complete the thermal model, a rigorous analysis was performed to evaluate the comparative merits of a range of methods. The assumptions made and thought process that leads to the eventual selection is outlined in the proceeding section. As it is the end goal to implement convective boundary conditions on the endocardial/blood ($h_{tiss}$) as well as the catheter tip/blood ($h_{elec}$) boundaries to properly represent the amount of heat lost during the ablation, a thorough survey of the topic and an attempted calculation and thought exercise will follow.

**Endocardial-Blood Volume Convective Film Coefficient ($h_{tiss}$) Selection**

To date, researchers have implemented the $h_{tiss}$ boundary condition with values ranging from 44 to 6090 W/m$^2$·K depending on the site and material properties that they used.$^{38,46–48}$ Many of these tried to calculate the coefficient assuming laminar blood flow.$^{47,68,69}$ There have also been attempts to measure $h_{tiss}$ using an in-vitro$^{46}$ physical swine model constructed of silicone rubber and a blood substitute; this experiment gave $h_{tiss}$ values ranging from 510 to 4800 W/m$^2$·K. Citing the obvious problems that arise when building a functioning model of a heart, Tangwongsan et al. performed some much needed in-vivo$^{70}$ measurements in pigs.

Such a wide range of coefficients used for such an important parameter undoubtedly confirms that there is extremely high variability in this measure due largely to differences between subjects. As a starting point, an attempt will be made to compute $h_{tiss}$ then some discussion must follow before a value is chosen; ultimately, a range will be used to establish the dependence of critical ablation metrics on this highly variable parameter.
The simplest way to get a baseline of the potential convective film coefficient $h_{tiss}$ is to simply consider the case where there is no blood flow at all. Aside from the obvious limitation of neglecting blood flow, there is an issue with this approach since the calculation of natural convection from a flat plate depends on the orientation of the "plate" considered but offers some insight into the problem nonetheless. Accordingly, both the horizontal and vertical cases will be computed just to get some additional perspective. In both cases, the Grashof Number ($Gr$) (an approximation of the ratio of buoyancy to viscous force) must first be computed:

$$Gr = \frac{\beta g \rho^2 L^3 \Delta T}{\mu^2}$$  \hspace{1cm} (4.5)

where $\beta$ is the expansion coefficient (the expansion coefficient of water will be used), $g$ is acceleration due to gravity, $\rho$ is the density, $L$ is the characteristic dimension, $\Delta T$ is the temperature difference between the surface and the bulk blood and $\mu$ is the viscosity of blood. The characteristic length for this analysis is the height of the surface therefore it is logical to select 2.33mm, the diameter of the catheter. The temperature difference chosen will be a modest 5°K in order to capture what the coefficient would be given a small temperature differential in order to act as a proper baseline (obviously a larger gradient would lead to a larger film coefficient). The next step is then to calculate the Rayleigh Number ($Ra$) (shown in equation 4.6) which is a measure of buoyancy driven flow in natural convection.

$$Ra = Gr \times Pr$$  \hspace{1cm} (4.6)

Where $Pr$ is the Prandtl Number ($Pr$) (for blood it is approximately 25). Using
the properties described in section 4.1.2, the Rayleigh number is 1475.9. From here, the Nusselt Number (\(Nu\)) and finally the baseline convective film coefficient for a vertical plate can be estimated (shown in equation 4.7). Simply, \(Nu\) relates thermal conduction with thermal convection in the fluid (the ratio of diffusive resistance and convective resistance).

\[
Nu = \left( 0.825 + \frac{0.387Ra^{1/6}}{[1 + (0.492/Pr)^{9/16}]^{8/27}} \right)^2 \tag{4.7}
\]

\[
h_{\text{vertical}} = \frac{Nu k}{L} \tag{4.8}
\]

For the case of a vertical plate and natural convection, the Nusselt number is 4.3729 giving a convective film coefficient of 922 W/m²·K.

Repeating the procedure for a horizontal plate is similar with the only difference coming when computing the Nusselt number. In equation 4.9, \(Nu_1\) is for a Nusselt number when the hot side of the plate is facing up, while the formulation \(Nu_2\) is for when the hot side is facing down. As neither scenario is totally accurate, the true value would likely lie somewhere in between.

\[
Nu_1 = 0.54Ra^{1/4} \quad Nu_2 = 0.27Ra^{1/4} \tag{4.9}
\]

These convective coefficients are predictably much smaller with hot side up case giving a value of \(h = 491\) W/m²·K while the hot side down had a value of \(h = 245\) W/m²·K. On the whole, these results compare quite well with ones obtained in similar fashion\textsuperscript{70} and are in the range used by many of the works cited. Undoubtedly, the
accuracy of these results is not impressive but they serve as a baseline for ablation sites that are sheltered from direct blood flow (such as in the recess underneath the mitral valve leaflets).

For the high flow cases, some consideration must be given to the measurements taken of sites in in-vivo pigs. These measurements yielded values of 5350 W/m²·K on the lateral wall of the LA, 7000 W/m²·K on the LA medial wall and 9500 W/m²·K floating in the middle of the LA. These values are high compared to other literature values and some of that is no doubt due to the fact that pigs have higher heart rates and blood pressures than humans; however, their cardiac output is quite similar. Given these facts, it would seem reasonable to use these values as upper limits to the study.

**Catheter Tip-Blood Volume Convective Film Coefficient ($h_{elec}$) Selection**

With ranges for $h_{tiss}$ established, all that is left to do is calculate the remaining coefficient that describes the rate of heat transfer from the surface of the catheter tip that is exposed to the passing blood volume. Any computation of $h_{elec}$ will depend largely on the fluid flow profile that the catheter is exposed to. While there is much variability here as well due to varying patient health and physical structure of the organ itself, blood velocities have been measured in humans both in the pulmonary veins and near the tip of the mitral valve leaflets (to obtain the highest velocity) using transesophageal echo. Due obviously to the pulsatile flow, the maximal average flow rate measured in the pulmonary veins (during ventricular systole) was 0.41 m/s while the minimal flow was 0.18 m/s (during atrial contraction). The velocity recorded at the mitral leaflets during atrial contraction was 0.44m/sec. Given these flow rates in the pulmonary veins and using measured vein cross-sectional data an estimate
of the Reynolds Number ($Re$) for each vein (using an elliptical pipe assumption) is computed as follows:

- The primary ($a$) and secondary ($b$) radius of each vein is calculated as:

$$a = \frac{D}{2} \quad b = \frac{A}{(\pi \times a)} \quad (4.10)$$

Where $A$ is the particular vein’s cross-sectional area in $mm^2$ and $D$ is its diameter in $mm$ taken at the ostium.

- Next, the perimeter of the ellipse is estimated using the infinite series:

$$P = 2a\pi \left(1 - \sum_{i=1}^{\infty} \frac{(2i)!^2}{(2^i \cdot i!)^4} \cdot \frac{e^{2i}}{2i - 1}\right) \quad (4.11)$$

Where $e$ is the eccentricity of the ellipse, defined as:

$$e = \frac{\sqrt{a^2 - b^2}}{a} \quad (4.12)$$

- It is then required to calculate the hydraulic diameter of the pipe (since it is noncircular) in order to compute the Reynolds number.\textsuperscript{73} The hydraulic diameter is computed as:

$$D_h = \frac{4A}{P} \quad (4.13)$$

- Finally, the Reynolds number is calculated as:

$$Re = \frac{\rho V_{avg} D_h}{\mu} \quad (4.14)$$

Where $\rho$ is the blood density, $V_{avg}$ is the average blood velocity and $\mu$ is the
dynamic viscosity.

<table>
<thead>
<tr>
<th>Pulmonary Vein</th>
<th>Mean Diameter (mm)</th>
<th>Mean Cross-Sectional Area ($mm^2$)</th>
<th>Reynolds Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right Superior</td>
<td>11.9</td>
<td>132.2</td>
<td>2637</td>
</tr>
<tr>
<td>Left Superior</td>
<td>10.0</td>
<td>106.5</td>
<td>2339</td>
</tr>
<tr>
<td>Right Inferior</td>
<td>12.7</td>
<td>142.6</td>
<td>2747</td>
</tr>
<tr>
<td>Left Inferior</td>
<td>9.4</td>
<td>98.0</td>
<td>2233</td>
</tr>
</tbody>
</table>

Table 4.2: Listing of calculated Reynolds number for each Pulmonary Vein.

As can be seen in Table 4.2, the flow in each vein is in (or is near) the transitional flow region, defined as $2300 \lesssim RE \lesssim 4000$. The significance of this is that with all four pulmonary veins filling the left atrium in (or near) the transition region to turbulent flow, then experiencing a sudden expansion (coupled with the fact that the atria itself is constantly contracting) the whole scenario causes flows that are quite turbulent. When flows are highly turbulent, the turbulence acts as a mixer, in effect cycling the fluid that contacts the immediate interface much more often. This increased mixing leads to enhanced rate of heat transfer (this relationship is illustrated in Figure 4.3 for the case of fluid flow along a plate).

Accordingly, an appropriate correlation must be chosen that can effectively fit turbulent flow data for the case considered. As such, the Churchill and Bernstein correlation for forced convection over a cylinder in cross flow (in this model the catheter tip is the cylinder) for both laminar and turbulent flows is widely used and will serve as the basis for the computation of $h_{elec}$.

The Reynolds number is a measure of the ratio of a fluid’s inertial and viscous forces. Basically, as Re increases, the fluid velocity and boundary film thickness
Figure 4.3: Variation of convective film coefficient for flows over a flat plate as flow changes from laminar to turbulent. V depicts the direction of flow, $h_c$ is the convection coefficient, $t_s$ denotes the surface of the plate and $x_{cr}$ is the length of the laminar region. Note the spike in convection rate when the flow transitions from laminar to turbulent. Reprinted from Solar Energy, Vol 80, Ernani Sartori, Convection coefficient equations for forced air flow over flat surfaces, 1063-1071, Copyright (2006), with permission from Elsevier.

(thermal insulating layer) decreases; leading to a higher convection coefficient.

\[
Re = \frac{u_\infty D \rho}{\mu} \tag{4.15}
\]

Equation 4.15 computes the Reynolds number of the fluid flowing around the catheter. In this equation $u_\infty$ refers to the stream flow, $D$ is the catheter’s diameter and $\mu$ is the dynamic viscosity of blood.

Equation 4.16 computes the Prandtl number.

\[
Pr = \frac{c \cdot \mu}{k} \tag{4.16}
\]
As can be observed, the Prandtl number is dependant only on properties of the fluid (unfortunately, these properties are pressure and temperature dependant where \( c \) is the specific heat capacity, \( \mu \) is the dynamic viscosity and \( k \) is the thermal conductivity). Simply put, the Prandtl number is a descriptor of how the fluid transfers heat (low Pr indicates a fluid that is conductive transfer strong while high Pr indicates that the fluid is convective transfer strong).

Equation 4.17 shows the Churchill-Bernstein correlation for calculating the Nusselt number. The Churchill-Bernstein correlation is used in scenarios that have a wide range of Reynolds numbers, Prandtl numbers and in cases where the flow is turbulent.

\[
Nu = 0.3 + \frac{0.62Re^{1/2}Pr^{1/3}}{\left[1 + (0.4/Pr)^{2/3}\right]^{1/4}} \left[1 + \left(\frac{Re}{282000}\right)^{5/8}\right]^{4/5}
\]  

\text{(4.17)}

Finally, the convective film coefficient \( h_{elec} \) is computed (Equation 4.18).

\[
h_{elec} = \frac{Nu k}{L}
\]

\text{(4.18)}

All of the work described in the preceding section can effectively be summed up in Table 4.3.

<table>
<thead>
<tr>
<th>Convection Film Coefficient</th>
<th>Literature Range</th>
<th>Values Chosen for Study</th>
</tr>
</thead>
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<tr>
<td>( h_{tiss} )</td>
<td>Low 44  High 5350</td>
<td>Low 268  High 5350</td>
</tr>
<tr>
<td>( h_{elec} )</td>
<td>Low 721  High 6090</td>
<td>Low 1749  High 7920</td>
</tr>
</tbody>
</table>

Table 4.3: Convection film coefficients implemented in the thermal model. Both low and high flow cases are presented. Note: All convection film coefficients listed have units of \( W/m^2 \cdot K \).
4.1.4 Perfusion and Metabolic Heat Generation Rates

The perfusion heat loss rate ($Q_p$) and metabolic heat generation rate ($Q_m$) are potentially important remaining terms in the bioheat equation (equation 4.1). As discussed, when it comes to continuum heat transfer models, the perfusion term is an extremely controversial one. If the site considered is significantly vascularized or there are large vessels nearby, this term can often be quite high. When modelling ablation in the liver or other tissues with high degrees of perfusion, $Q_p$ can play a significant role. Generally, in the case of atrial ablation (and other non vascular tissues), this term is ignored since its effect is negligible.\(^{40}\)

Similarly, the metabolic heat generation term is always ignored in cardiac ablation modelling because its effect has also shown to be insignificant for ablation.\(^{44}\)

4.1.5 RF Power Deposition

The last thing to discuss is how the RF power, $q$ in equation 4.1, will be deposited into the system. Heating is generally related with an electrical power density $P \ [W/m^3]$.\(^{75}\)

$$P = j \omega \tilde{\epsilon} E \cdot E \quad (4.19)$$

Where $\omega$ is the angular frequency (rad/s), $\tilde{\epsilon}$ is the complex permittivity of the medium ($\tilde{\epsilon} = \epsilon_0 \epsilon_r - j \frac{\sigma}{\omega}$) and $E$ is the electric field. Expanding this expression gives equation 4.20.

$$P = (\sigma + j \omega \epsilon_0 \epsilon_r) |E|^2 \quad (4.20)$$

The real term, $\sigma |E|^2$ represents the heating due to the currents flowing through the resistive tissue (translational motion of ions). Interestingly, the imaginary term,
\( j \omega \varepsilon_0 \varepsilon_r |E|^2 \) represents the heat dissipation caused by molecular rotation and vibration of bound charge carriers. The reality of RF ablation is, however, that the RF current has a frequency of (or in the order of) 485 kHz, meaning that given the extremely short distance of interest and long wavelengths, the problem is effectively quasistatic (see section 3.1.3). The end ramification of this is that the complex heating term is neglected and equation 4.20 reduces to equation 4.21.

\[
q = \sigma |E|^2 \quad \rightarrow \quad q = J \cdot E
\]  

(4.21)

To sum up, in RF cardiac ablation, the mechanism through with myocardial injury is induced in the tissue is through the Joule effect characterized by the flow of RF currents through the tissue causing resistive heating.

### 4.2 Implementation of Thermal Model

The solution of the bioheat equation is simply to solve Poisson’s equation while implementing the boundary conditions, as described, throughout the solution region. Some solvers implement “Fast Poisson Solvers” (which are based on Fourier Transformations of the differential equation) to make this possible; yet they are not suitable due to interest in observing the tissue heating and lesion progression over time. Therefore, a stepwise method for solving these equations must be implemented. For this, SEMCAD uses a variant on the Finite Difference Time Domain (FDTD) method for a non-uniform grid. This functionality allows the use of the grids generated when solving the electrical problem, eliminating the need for an interpolation scheme which introduces error into the model.
Given the specifications made in the previous section of this chapter, the final form of the bioheat equation as implemented by SEMCAD is given in equation 4.22.

\[ \rho c \frac{\partial T}{\partial t} = \nabla \cdot (k \nabla T) + \sigma |E|^2 \]  

(4.22)

When implementing any model, there are stability criteria that must be observed. For time-domain simulations, these considerations extend past the spatial step size and also include the time step size. Included with the software, SEMCAD deduces the time step using a von Neumann stability analysis.\(^76\) Equation 4.23 gives a simple representation of how the stable time step is computed for uniform meshes (meshes where the spatial step size in all planes is uniform).

\[ \Delta t \leq -\frac{2}{\left( \frac{\rho c \omega}{c} \right) - 4 \left( \frac{k}{\rho c} \right) \Delta x^2} \]  

(4.23)

For non-uniform meshes, convective boundary conditions and thin structures the calculation is significantly more complicated. This was a major consideration when implementing the thermal model because of the high thermal conductivity of the catheter tip. As can quickly be observed, even in the simplified equation shown in 4.23, the stable time step is proportional to the ratio of thermal conductivity \(k\) and spatial step \(\Delta x\). The worst case scenario then is when the thermal conductivity is high and the spatial step size is very small; this is exactly the case around the tip of the catheter. Small spatial step size is required for stability in the electrical analysis and also to effectively model the conduction effect that wicks heat from the hot spot (directly under the catheter tip where current density is greatest) to the blood pool where convection occurs. Unfortunately, due to the fineness of the mesh the thermal
simulation requires days to complete since the time step becomes so small. To get around this, the catheter tip was implemented as a Platinum-Iridium surface instead of a volume. This approximation is not unreasonable because, not only is that how the catheter is actually built, but due to the high thermal conductivity, heat would conduct so quickly along the surface of the catheter that any inconsistency would likely be extremely small. With the catheter modelled as a surface, the problem can run in a very reasonable 30 minutes for 60 seconds of elapsed simulated ablation time.

4.2.1 Model

The ablation model for the thermal study is similar to the model presented in 3.1.1. Once again, the model consists of a 7 French, 4mm standard ablation catheter, a dispersive patch electrode (which serves as a reference plane), a homogeneous slab of tissue (myocardial tissue to be ablated) and a blood volume that encompasses the entire region. The catheter tip’s depth and angle are once again varied as described in section 3.2. The individual components of the model will be treated slightly differently (using distinct boundary conditions) but the same physical elements will be maintained; therefore, the mesh inside the tissue and catheter tip will be preserved from the electrical model.

4.3 Ablation Scheme

There are quite a few ablation schemes that are commonly used: constant temperature (supplied power is modulated in order to maintain a constant temperature at the thermistor embedded in the catheter), constant power (voltage is modulated to
maintain constant power), constant current and constant voltage. Clinically, constant temperature and constant power are the schemes most commonly used. For this study, a constant voltage ablation mode was implemented. This decision was made for two reasons:

1. Natural ablation phenomena are not obscured by compensatory schemes. This makes prevalent trends easier to identify during analysis.

2. Ease of implementation

Accordingly, the peak voltage was set to 23V. This voltage was chosen since the higher impedances measured at 485 kHz were between 100 and 120Ω, which give an approximate starting power of 5W (a fairly safe power level by more advanced schemes) at the deeper penetrations.

The simulation was run for 60 s, the maximum rated time of the catheter modelled (St. Jude Therapy Cool Path Duo). Since myocardial injury begins to occur at \( \approx 50^\circ C \) and there are no data that define the time/temperature relationship above 50°C, the 50°C isotherm was defined as the lesion boundary. Ablation was also considered finished if the maximum temperature calculated in the tissue volume was greater than 100°C after which tissue begins to dangerously “pop”.

The metrics used to qualify lesion formation with respect to catheter geometry were lesion volume, width, depth, maximal temperature reached in tissue (Tmax), and the time taken to reach 100°C.

### 4.4 Results

As covered earlier in this chapter and in chapter 2, the goal of any ablation procedure is to achieve full electrical isolation of the atria from the ectopic focus (usually in
the Pulmonary Veins) preventing them from acting as a substrate for self-sustaining reentry. To accomplish this, a full transmural lesion is required. That being said, the lesion depth is not the only factor that matters as excess temperatures cause immediate (and potentially traumatic) health concerns while excessively thin lesions have the potential problem of not being continuous with the next ablation site; effectively leading to gaps in the attempted isolation ring. Therefore, the key findings of the thermal analysis presented in this chapter will include the lesion depth, width and volume as well as the maximal temperature calculated inside the volume as the ablation progresses at both the high and low flow conditions discussed (See Table 4.4 for values corresponding to different flows).

4.4.1 Maximum Temperature vs. Time

Considering the potential danger associated with ablating using the simple constant voltage algorithm, the logical first parameter to investigate is the maximal temperature calculated within the solution region. The progression of the maximal temperature detected in the tissue over time is presented at each penetration depth studied, with each curve corresponding to a different catheter angle, at low flow (Figure 4.4) and high flow (Figure 4.5) conditions.

In the low flow case, it becomes immediately clear that the maximum temperature can attain dangerous levels extremely quickly if there is sufficient catheter penetration. In fact, the subplots showing very shallow depths of 0.04mm and 0.8mm are the only ones that manage to go the entire simulation time course (60s) without exceeding 100°C. Subplots showing penetration depths of 1.6mm, 2.4mm, 3.2mm and full penetration (4mm) exceed 100°C according to the depth (∼ 34-43s, 17-21s, 1.6-9.7s
and 2.9-5.7s respectively) with the time range corresponding to angle related temperature differences. It was discovered in chapter 3 that due to different configurations of catheter penetration depth and angle, the change in surface contact area was proportional to resulting electrical impedance. Given that a constant voltage ablation algorithm is used, one would then expect to see lower power at higher surface contacts (which is shown in table B.3) yet the thermal problem is a complex one since it really depends on the amount of power dissipated in the tissue. This is worth mentioning because the overall power dissipated for simulations with higher contact is less. This
is a function of having impedance proportional to contact and constant voltage. The bulk of resulting current at lower contact however, flows through the blood (because of its relatively high conductance).

Higher temperatures, albeit very slightly higher in most cases, are consistently observed at the lower catheter angles, the exception being at 3.2mm and at full penetration. At 3.2mm penetration depth, the temperature spikes when the catheter is at 15° taking only 1.6s to reach 100°C while the other angles take nearly 10s to reach the same. This can likely be explained by the 4% increase in catheter contact at this angle that forces just a little bit more current though the tissue. At 4mm penetration depth however, the times to 100°C are longer than the 3.2mm at 15° case (2.9-5.7s instead of 1.6s) and the angles 45° and 30° get to 100°C the quickest. This can be explained by the fact that these angles also have the highest surface contact (and impedance) of that depth set and therefore, likely have the most power dissipated within the tissue. This is consistent with the finding in the 3.2mm plot as the impedance in the 15° scenario is higher than any angle at 4mm.

In contrast with the low flow case, the high flow ablation temperatures cluster much closer together and are much slower to exceed 100°C. In fact, when the catheter’s penetration depth is less than 0.04mm, even the maximal temperature in the tissue doesn’t exceed 50°C. Temperature profiles at high flow seem much more stable and indicate that an equilibrium has been reached between the rates of input heat and the rate that the heat is being carried away by the blood volume. At a depth of 2.4mm, 100°C is exceeded for the first time, although after getting very close (<90°C for ~33s) it takes nearly 54s for this to occur. At 3.2mm and 4mm 100°C is exceeded at 19.12-34.01s and 21.1-31.12s respectively. Once again, when the catheter is at 15°, the
Figure 4.5: Maximal temperature detected in the tissue given a high flow condition. Temperature tends to be the highest with the exception coming at full penetration. This can no doubt be explained by the fact that the measured resistance at full penetration depth is as follows:

Table 4.4 shows that the maximum temperature, and rate of maximum temperature increase in the tissue is strongly correlated to the initial measured resistance. The only exception being at 45° but the times shown are so close that even the minute fraction of the catheter that is not touching the tissue (note that the decimal places after the 100 indicate that rounding to 100% has been done) may result in added
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<table>
<thead>
<tr>
<th>Depth (mm)</th>
<th>Angle (°)</th>
<th>Resistance (Ω)</th>
<th>Contact Area (%)</th>
<th>Time to Reach 100°C (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>15</td>
<td>105.0872</td>
<td>100.000</td>
<td>26.07</td>
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<td>-</td>
</tr>
</tbody>
</table>

Table 4.4: Comparison of resistance and time to reach 100°C calculated for full penetration depth at high flow.

cooling that led to the 0.21s difference in time to reach 100°C. In general, at both high and low flows, the maximum temperature calculated inside the tissue tracks very well with whichever configuration has the highest impedance. In nearly all cases, this implies that highest temperatures detected belonged to the configurations at 15° and temperature decreased as angle increased accordingly for each depth set.

### 4.4.2 Lesion Depth vs. Time

Lesion depth is perhaps the second most important parameter during an ablation, and the most important when it comes to successful procedural outcomes. Figures 4.6 and 4.7 show the lesion depth progressing throughout time at each catheter penetration depth and angle considered in the study for low and high blood flows respectively.

In the low flow case we can see that a transmural lesion (a full 4mm lesion) forms at quite low penetration depths (beginning at 0.8mm) after sufficient time (∼45s). At deeper penetration depths they generally occur sooner and there is not a whole lot of angle related difference. At 3.2mm penetration depth however, since the 100°C threshold is reached so quickly at 15° compared to the other catheter/tissue angles, a full 4mm thick lesion is not necessarily reached. At full penetration, a transmural
lesion occurs at every angle.

Of note is this “staircase” shape of the lesion depth plots. This is a function of having only a finite number of points in the mesh that discretizes the tissue. The observed temperature transition is so rapid that the lesion progression is also quite rapid; confined only by the tissue’s ability to conduct heat. Since an FE model was used to study this problem, an interpolation could have been done to find the precise location of the 50°C isotherm however this approach has its own shortcomings:

- The solution is only evaluated at the grid points therefore the accuracy of the interpolated solution depends on how well the chosen interpolant function approximates the true behaviour in the region. SEMCAD offers no flexibility when it comes to applying different functions.
- Using this interpolated solution increases the post-processing complexity.
- Using the 50°C isotherm to demarcate the lesion boundary often overestimates the true lesion.
- The grid was suitably fine to provide a curve that does not suffer badly from staircasing that useful information cannot be extracted.

Given all of this, it made more sense to simply use the nearest grid point that was at 50°C as the depth boundary. Despite the “staircasing” the trend is quite clear from the data.

In the high blood flow case, a slightly different landscape emerges. At 0.04mm, the temperature in the tissue never exceeds 50° therefore there is no lesion possible at this depth. At 0.8mm penetration depth, a thermal equilibrium is reached when the lesion is only about 2mm deep, with the 15° and 30° cases achieving slightly deeper lesions, ~2.1mm compared to 1.8mm for the other cases. Lesions get progressively
deeper at 1.6mm and 2.4mm penetration depth but given the high convection levels, the lesion generally remains within 1.3mm of the deepest point of the catheter. This is likely due to the fact that in the model, both the endo- and epicardial sides of the tissue experience the same rate of convection. Implemented this way for the sake of numerical ease, physiologically this is not the case and likely introduces some key shortcomings in the analysis.

At 3.2mm penetration depth, a transmural or near transmural lesion is achieved for all but the 75° and 90° cases, those with the lowest resistance (see Table 4.4). Not
surprisingly, at full penetration a fully transmural lesion is achieved for all angles.

### 4.4.3 Lesion Volume vs. Time

Lesion volume is a sort of amalgamate measurement that gives the end impression on how effectively power is transferred from the catheter to the tissue. Some works consider the lesion depth and lesion width and then make calculations assuming that the lesion formed is perfectly elliptical or an ellipsoid that has been sliced at the top yet Figures B.1-B.4 show that the lesions formed, particularly at catheter angles other than 90° are not perfectly elliptical or even symmetric. For this reason, lesion volume is computed numerically as the sum of voxels that have a temperature exceeding 50°C.

Figure 4.8 shows how the ablated lesion volume progresses with time in the low blood flow case. The first thing that is evident from even a cursory glance at each subplot is that a thermal equilibrium has not been reached. Clearly, the convective rate is so low that the rate of incoming heat supplied by the catheter is totally overwhelming. Conduction plays an increased role in lesion growth for all low flow studies, as can be seen by the increased lesion width (Figures B.1-B.4) and increased lesion depth (Figure 4.6). In fact, even after 60s the lesion volume is still increasing for all depths. Once again, the trend continues where a more acute catheter angle corresponds with increasing lesion volume. As penetration depth increases, generally so to do the lesions. However, when the physiological limit of 100°C is imposed, things really begin to get interesting!

At a penetration depth of 1.6mm the temperatures inside the tissue begin to exceed 100°C after about 35s. At a penetration depth of 2.4mm this pattern repeats
itself but the time taken is only about 17s, once again, the largest lesion being at an angle of 15°. At 3.2 mm penetration depth however, the temperature spikes so quickly (after 1.6s) that the heat applied doesn’t have sufficient time to propagate deeply into the tissue. The resulting lesion is much smaller than not only the lesions formed at the same depth but even the lesions formed at a depth of 0.8mm. Table 4.5 shows the lesion volumes for all angles at penetration depths of 3.2mm and 4mm.

<table>
<thead>
<tr>
<th>Depth (mm)</th>
<th>Angle (°)</th>
<th>Resistance (Ω)</th>
<th>Contact Area (%)</th>
<th>Time to Reach 100°C (s)</th>
<th>Lesion Volume (mm³)</th>
</tr>
</thead>
<tbody>
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</table>

Table 4.5: Comparison of resistance, surface area, the time to reach 100°C and lesion volume calculated for 3.2mm and full penetration depth at low flow.

Predictably, at higher flow rates, the lesions tend to be smaller. Inspection of the shapes of the curves at all penetration depths (Figure 4.9) indicates that the rate of deposited heat related lesion expansion reaches an equilibrium with the rate of heat dissipation due to convection. While not visible in the time scale shown in any of the low-flow plots (Figures 4.4, 4.6 and 4.8), that equilibrium does occur, just at much higher temperatures, lesion widths, depths and volumes. Once again, the lower angles
(with the higher impedances) have larger lesion volumes as their electrical coupling with the tissue is no doubt better. Due to the lower maximal temperatures, the heat has more time to penetrate deeply into the tissue and generally the ablations can continue longer than in the low flow cases. In fact, despite the huge difference in convection rates, we can see in table B.3 that at the deepest penetration depths the lesion volumes are often larger at the high flow rates!

4.4.4 Contact Area Related to Key Lesion Indicators

In what could be defined as the crux of this work, the final progression was to show the correlation between the previously presented data and the catheter contact area for both blood flow cases. Shown in Figures 4.10 and 4.11, the key lesion formation indicators (lesion volume, depth, maximum temperature and ablation power) are compared with catheter contact area with all values taken at the study’s endpoint (either 60s elapsed ablation time or the time which $T_{\text{max}}$ reaches 100°C). In the low flow case, the lesion volume trend (Figure 4.10(a)) seems to be the most confusing: volume increases with contact until a depth of 1.6mm is reached and then lesions actually begin to get smaller, with some of the smallest lesions of all being produced at a depth of 4mm. Here it is important to remember that the $T_{\text{max}}$ first exceeds 100°C at a depth of 1.6 mm and the study is terminated before the natural, steady-state maximum temperature would be reached. Compare this result to the one shown in Figure 4.11(a) and we see an that lesion volume has an increasing relationship with contact area until very deep penetration depths.

In Figures 4.10(b) and 4.11(b), we see the relation of the maximum tissue temperature and catheter contact area in the low and high flow cases respectively. Inter-
estingly, in both cases (until the catheter is very deep into the tissue) the maximum
temperature recorded in the tissue doesn’t seem to have much angle dependence.
That is to say, at each grouping (according to penetration depth) the maximum tem-
perature varied little (only a few degrees) while the contact area variation was as
much as 19% (for a depth of 1.6mm). This would seem to indicate that the maxi-
mum temperature depends much more strongly on the absolute penetration depth,
the degree with which the catheter is engaged with the tissue, than the contact area.
Also, it was observed that maximum temperature was at (or over) 100°C for just
about all contact arrangements where depth was greater than 1.6mm for the low flow
case but doesn’t exceed 100°C until \( \approx 86 \% \) for the high flow case.

Figures 4.10(c) and 4.10(c) show how lesion depth varies with catheter contact
area. The low flow case is not very interesting in itself, as long as the contact area
was greater than \( \approx 20 \% \) then a transmural lesion was all but guaranteed, but its
juxtaposition with the high flow case tells a much more complete story. In the high
flow case, the lesion depth increases quickly with catheter contact, which is not at
all surprising considering that the lesion depth cannot really be any less than the
penetration depth. This assertion however, must be qualified because when real-
world tissue deformation is accounted for this unlikely to be a certainty as seen in the
model. Catheter advancement depth would deform the tissue and cause it to retreat;
not necessarily leading to increasing penetration depth as in the model. Clearly, the
flow rate and the catheter depth are important factors in the overall depth of the
lesion, but importantly, the difference between the penetration depth and the final
lesion depth does vary with respect to contact angle.

Finally, in Figures 4.10(d) and 4.11(d) the relationship between ablation power
and contact area is shown for the low and high flow cases. Not surprisingly, these curves are identical because the ablation power depends only on electrical parameters, which for the purpose of this study, are temperature invariant. Interestingly, the ablation power decreases with respect to increasing catheter contact area (the exception being when the catheter is fully engaged due to penetration effects explained previously in Section 3.3.5). While the absolute amount of ablation power, as seen at the generator, does decrease, the amount of power dissipated in the tissue has actually increased in the cases with greater contact area. This is a subtlety that is lost when constant current or temperature algorithms are used: despite the fact that lower generator powers are being used, the lesions formed are larger, deeper and hotter when contact area is increased.

Of note is the comforting fact that despite belonging to different penetration depth sets, simulations that similar contact areas returned nearly identical values for lesion volume, maximum temperature, lesion depth and ablation power. This would be also be expected to manifest itself experimentally.

### 4.5 Analysis of Results

It was found that catheter contact angle (not just penetration depth) plays an integral role in lesion formation and temperature response. It is easy to imagine that this would have implications in patient safety. Generally, as the angle that the catheter makes with the tissue becomes more acute the maximal temperature in the tissue spikes. Clinically, the final catheter contact geometry is largely unknown, therefore, in order to maximize both procedural efficacy and patient safety, more must be done to ascertain not just catheter penetration depth but also the angle, and tailor the
procedure accordingly to the geometric reality. In both the low and high flow cases, it is clear that there is a strong angle related dependence for all key procedural indicators. This angle related dependence is a direct result of the increased impedance caused by increased catheter surface area contact with the endocardium. The effective increase in electrical coupling between the catheter and the tissue leads to more current flowing through the tissue, despite the larger impedance, and generally larger lesions. This perhaps the most significant finding of the above analysis, particularly because of the constant voltage ablation scheme used; constant voltage is nice because the amount of power supplied decreases if the impedance increases. A potential disadvantage of the constant voltage scheme is that the applied power will increase throughout the procedure as the temperature dependent conductivity increases in the heated regions. While a constant voltage ablation scheme may not be the best way to safely ablate the atrial tissue responsible for atrial fibrillation clinically, the effect that catheter angle has on energy deposition in the tissue shown in this study cannot be neglected and would no doubt manifest itself regardless of the ablation mode chosen. In light of this, the information presented is used to make some inferences regarding the other schemes.

When the key lesion formation parameters were related to the catheter contact area, a few really interesting key points became instantly apparent:

- While lesion volume and depth increased with catheter contact angle, the maximum tissue temperature seemed to vary more with penetration depth. This is something that could be exploited if a technique were developed to calculate the penetration depth from the measured impedance.
- All lesion indicators were highly sensitive to the blood flow rate. Clinically,
this would be a value that would not only be unknown but also constantly in flux. While the value used for the convective film coefficient for the low flow case is quite low, it is not physiologically impossible. Therefore, an opportunity to further improve lesion formation and control runaway temperatures would have to include some kind of feedback to quantify this number. Potentially, a solution could involve a simple system identification problem where the surface temperature (as measured by the catheter) and input power are used to approximate what the maximum temperature would be at a certain depth in the tissue. With this information the convection rate could be approximated and a power delivery strategy calculated.

- Linear penetration depth depends heavily on the catheter penetration depth. However, the difference between the catheter and the straight line boundary of the lesion does also depend on catheter contact area. It would be very interesting to compute this plot of normalized lesion depth with a smaller convective film coefficient applied to the epicardial surface.

- Despite providing steadily decreasing ablation power output at increasing contact levels during a constant voltage ablation, the lesions formed were larger and hotter. A constant power algorithm would provide still larger and hotter lesions at the higher contact levels and given the continually varying depth of atrial tissue, much care must be taken with this approach.

While catheter contact is largely unknown, it is an important parameter to know and has significant ramifications on key factors that could decide whether or not a procedure is successful.

Table B.3 shows that the catheter configurations that have give the highest tem-
temperatures, widest and deepest lesions are the ones with the highest impedance, despite having the lowest input power! Clinically, this is relevant because it is very common to ablate using a constant power scheme. In this scenario, convection rates are always unknown and the knowledge of catheter contact is vague at best. Applying the same power to all contact geometries is not ideal for a few reasons. At deeper penetration depths where the electrical coupling is already quite high, using a power level used at the other depths would likely result in even higher temperatures. Obviously this creates an unpredictable situation considering the huge range of convective rates that exist and fluctuate constantly. Constant power has shown to be a nice solution to combat shifting conductances over the time of the procedure.

Applying a constant current scheme would provide a protective benefit since the tissue’s impedance is inversely proportional to its temperature, implying that as the procedure went on, a protective effect would decrease the supplied power. That being said, ablating in all scenarios with constant current would again lead to similar safety concerns as the constant power algorithm.

In the end, this study has shown that the impedance measured is directly proportional to the catheter’s ability to impart electrical energy into the tissue (this can be both good and bad). It also directly challenges the common perception that increased penetration depth unequivocally leads to increased lesion volume. Higher convection rates allow heat to propagate more deeply into the tissue which results in better lesions. It is therefore no wonder that irrigated ablation catheters have much more success than dry catheters alone. It is clear that no single procedure can be blindly applied in all cases and improved contact assessment is imperative for this treatment option to achieve the required safety and success rates.
4.6 Limitations

This study was designed to ascertain the effect that angle has on thermal parameters linked to successful ablation outcomes more than to predict the exact lesion characteristics during a procedure. In that, it was very successful. As with any study, there are going to be shortcomings to address in future work or to acknowledge going forward. Since the thermal problem is so complex there were quite a few simplifications made to ease the computational requirements or enhance stability. For example, it is known that nearly all parameters including specific heat capacity, thermal conductivity, density, viscosity and electrical conductivity change with temperature. In this study, these changes were all neglected except the change in electrical conductivity with temperature. To date, most studies implement an increase of 1-2%/°C in electrical conductivity. This study implemented a 1%/°C change using a linear fit to the exponential equation. Given the extremely flat slope this seems reasonable in the temperature range although at some temperatures the linear fit will overestimate the conductivity while at others it will be underestimated.

Admittedly, when the maximum temperature exceeds 100°C clinically, lesion formation would not suddenly stop as in this study. It is more likely that even if the procedure were terminated the instant excess temperatures were measured, the overall lesion volume created by the more deeply engaged catheter would continue to grow for some time afterwards. This does not however, change the fact that temperatures within the tissue can reach dangerous levels very quickly and that this rate depends strongly on catheter contact angle.

Another perhaps more significant shortcoming of this study is that there is no consideration for how the tissue may be mechanically deformed when a catheter
presses on it. To be certain, the tissue will not completely submerge the catheter (as a liquid might) when pressed in fully (Figure 4.12). In a real life scenario, a thin tissue such as the atrial wall would almost certainly be extended outwardly. Deformation of the tissue around the catheter tip would be prevalent but one thing is for certain: advancing the catheter 4mm would not result in the catheter being fully submerged in the tissue! It is also unknown how the mechanical deformation of the tissue might change thermal and electrical parameters that are crucial in the model...

One final shortcoming arises in the assigning of the convective boundary conditions. In this study, the same convective boundary was assigned to both the endo- and epicardial surfaces. This introduces some definite bias into the thermal results since the low flow rates would be largely unaffected by having both surfaces the same; in all likelihood the epicardial convective film coefficient is something similar to the low flow condition assigned. The high flow case, on the other hand, suffers because the heat is being quickly drawn from the tissue through both sides. This lowers the conduction which prevents the heat from accumulating deeper in the tissue (required for transmural lesions). Future studies will implement a different boundary layer on the epicardial surface and potentially a small layer of fat which is present (in variable thicknesses) on the epicardial surface.
Figure 4.7: Lesion depth vs. time in the tissue given a high flow condition.
Figure 4.8: Progression of lesion volume vs. time in the tissue given a low flow condition. Note: All plots have units of $mm^3$ for lesion volume.
Figure 4.9: Progression of lesion volume vs. time in the tissue given a high flow condition. Note: All plots have units of $mm^3$ for lesion volume.
(a) Plot of lesion volume vs. catheter contact area.

(b) Plot of maximum temperature vs. catheter contact area.

(c) Plot of lesion depth vs. catheter contact area.

(d) Plot of ablation power vs. catheter contact area.

Figure 4.10: Lesion formation indicators and their relationship with catheter contact area. Plots showing Lesion Volume, maximum temperature, lesion depth and ablation power with respect to catheter contact area. Plots shown here are for the low flow case. The legend shown at the bottom of Figure applies to all plots.
Figure 4.11: **Lesion formation indicators and their relationship with catheter contact area.** Plots showing Lesion Volume, maximum temperature, lesion depth and ablation power with respect to catheter contact area. Plots shown here are for the high flow case. The legend shown at the bottom of Figure applies to all plots.
Figure 4.12: Sample ablation model illustrating the lack of deformation around the catheter tip at full penetration.
Chapter 5

Experimental Results

In Chapter 3 an electrical model of the atria was constructed and complex impedance values extracted were found to be in the range of typical measurements made at 20kHz. The next step is to compare the in-silico results with actual measurements taken in the nearest physical representation in order to determine if the real-world phenomena match what is demonstrated in the model.

While the results from the model won’t be in perfect agreement, the important questions will be: how similar are the model and experimental data and what can we learn from the differences? As George E.P. Box famously said: "Remember that all models are wrong; the practical question is how wrong do they have to be to not be useful."

5.1 Description of Electrical Experiment Setup

The physical model was setup much the same way that the computer model was. That is to say, it consists of the same components: dispersive patch electrode, "blood" bath (saline), tissue slab and ablation catheter. While this is a simple way to reconstruct the complex geometry of the left atrium, it still consists of the same fundamental pieces and helps paint a picture of how contact depth and angle relate to the measured impedance.
The dispersive patch electrode was a stainless steel disc that had a diameter of 6.5 inches and was a quarter inch thick. The patch electrode serves as the reference, return current path. This was submerged in a saline bath that was 0.9% saline diluted with deionized water until a conductance of 6.5mS was reached. The tissue was taken from preparations of cow ventricle from the butcher and there was no perfusion. The tissue slab was placed in the centre of the patch electrode, endocardial side up, and secured with clamps. The ablation catheter, a Coolpath Duo 7 French 4mm (St. Jude, MN) was placed in a mill arm to precisely control its angle and depth. Angles of 0°, 30°, 60° and 90° and depths of 0mm (in the bath), 2mm and 4mm were considered. The depth was measured as the length that the mill arm travels when the catheter is on the verge of touching the tissue. A ablation generator set to approximately 0.6W and research version of the EnSite Contact (St Jude, MN) unit were used to generate and measure the tiny sensing currents demanded in this study.

Each individual 'measurement', was actually a three second data recording taken by a USB-6259 data acquisition board and LabVIEW software (National Instruments, TX USA), with a sampling frequency of 2048Hz. This was done in order to assess the variability of each measurement; in the end it gives a sense of consistency and repeatability of each measurement. All of the data collection and experimentation has been performed on site by St. Jude atrial fibrillation technicians under the supervision of Dr. Israel A. Byrd. To date, 36 experiments have been conducted.

5.2 Results of Electrical Contact Assessment Experiments

The raw data collected in the 36 trials described above is compiled in Table C.1. This data is consolidated in Table 5.1 to the twelve separate geometries that the 36 trials
The recorded impedance when the catheter is in the bath (not engaged with the tissue) the impedance is approximately $89 - 3.46\,\Omega$ while the model reports one of approximately $86\,\Omega$; this starting point offers quite good agreement. As the catheter becomes more engaged in the tissue, the impedance increases both in its real form (resistance) and imaginary form (reactance becomes more negative) which was predicted by the model.

In Table C.1 it is encouraging to observe that at each depth the standard deviation for each measurement was relatively low (less than 0.25Ω resistance and 0.04Ω reactance). For each true measurement, for which there are over 6000 samples taken, the measured impedance doesn’t fluctuate very much. This would indicate that each measurement is quite repeatable. In Table 5.1 we can see that the standard deviation of measurements taken between preparations is quite a bit higher. This is no

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<th>Reactance (Ω)</th>
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Table 5.1: Consolidated Experimental results
doubt due to the extremely low sample size, in this case only three samples per category. The variability did not seem to be related to the depth or the angle, meaning that with more samples the variability would likely decrease. At any rate, it is clear that before any statement can be made with any sort of statistical certainty, a more complete set of experiments must be conducted.

5.3 Analysis of Results of Electrical Contact Assessment Experiments

The results of simulation are compared with the set of experimental data, preliminary though it may be. This data is presented in Figure 5.1.

![Plot of Resistance vs. Penetration Depth](image1)
![Plot of Reactance vs. Penetration Depth](image2)

(a) Plot comparing resistance determined in experiment and simulation.
(b) Plot comparing reactance determined in experiment and simulation.

Figure 5.1: Comparison of experimental and simulated data.

The first thing that is immediately notable is that the simulation data agrees quite well with the experimental data for shallow penetration depths and agrees quite poorly with the experimental data when the catheter is pushed deeper into the tissue.
Also, the agreement between the reactance component of the model and experimental data is worse than the resistive component. Both of these observations are quite likely artifacts of comparing ideal simulations and experiments. It is likely that the main contributor of the discrepancy between the results is the amount of mechanical deformation of the tissue that occurs the deeper the catheter is pushed into the tissue. In the simulations, the blood/tissue interface acts like a liquid mechanically, that is to say that doesn’t deform the tissue in any way. If the catheter is advanced 2mm deeper into the tissue, then 2mm more of the catheter tip will be embedded in the tissue. In the experiments however, when the catheter is advanced, a gradual sloping pit forms around the high pressure point as a result of the surface tension. Therefore, in real terms, if the catheter is pushed 2mm deeper into the tissue, it may only incrementally increase its surface contact area with the tissue. This is an extremely important distinction since the model indicated that there was an exponential relationship between catheter contact area and impedance!

This hypothesis seems to be in small way validated by the fact that the simulations are in such good agreement between 0 and 2mm penetration depth and that this agreement disappears as the catheter gets deeper. This makes sense as the hemispherical tip of the catheter would plunge quite well (at least a shallow distance) into the layer of epithelial cells on the endocardium. The body of the catheter however, would have a harder time making contact with the tissue particularly at more obtuse angles.

The second observation was that the experimentally measured reactance didn’t seem to agree quite as well with the model as the resistance. While this is also a function of the difference in actual contact area, here the differences in material prop-
erties also account for some of the deviation between the simulated and experimental results. The conductance of the saline bath was adjusted until it was similar to that of blood, this was done by diluting saline. This makes the conductivity of water a good approximation of that of blood however there is no effort made to match the permittivity of the saline bath to that of the blood. It is known that the permittivity of water is quite a bit different than that of blood\textsuperscript{78} and it is this difference that likely makes up the reactive component of the measured impedance.

The final observation was that the experimental results didn’t exhibit the same angle dependency as the theoretical ones. Once again, the mechanical deformation that the tissue undergoes in a real-life scenario is likely responsible for this discrepancy. Due to the fact that the tissue used in the experiments was stiffer ventricular tissue, as the catheter was advanced into it, no more than the hemispherical tip would contact the tissue without it retreating and deforming. This finding is consistent with the theoretical ones which also show that when only the hemispherical tip is in contact with the tissue, little angle dependent impedance variations are observed.

In the end, the set of experimental results offered excellent agreement with the model. The data obtained showed that the results obtained by the model were reasonable and within plausible physiological range.

5.4 Future Experimental Work

The first and foremost step to solidify the findings of the model is to have a greater sample size of each contact situation. More angles and more depths need to be considered to achieve a higher statistical power of the results. One of the best findings of the experiments was the strong agreement of the experimental data and the simulated
data at more shallow penetration depths. That being said, more experiments must be done in between 0mm and 2mm to further validate this point.

Perhaps the most important unanswered question of this work is: despite the divergence of the measurements taken by the two methods at deeper penetration depths, does the measured experimental impedance still tell the operator how much of the catheter is touching the tissue? It is possible that the measured impedance still corresponds to the catheter contact area, there is just less contact in a real-world scenario then in the modelled one. This would have to be verified using some kind of optical imaging study that calculates exactly how much of the catheter is contacting the tissue during any particular measurement. Another approach would be to modify the model to take into account the real world effects of mechanical deformation. Finally, and probably more pertinent of all, how does a particular contact area (taking into account the tissue deformation tissue) compare thermally to the modelled ones? Will lesion volume, depth, width etc. still be similar to the modelled ones? These are the real questions that remain to be answered, but this analysis was still able to afford a huge step forward in terms of knowledge gained and provides a direction for future research.
Chapter 6

Conclusions and Future Work

The objective of this work was to determine how catheter interaction geometry with the endocardial surface effects the outcomes of catheterized ablation for the treatment of atrial fibrillation. While it is known that catheter contact and incident angle had an impact on the size of lesion produced during an ablation, it was unknown exactly how catheter depth and angle interacted to create each lesion and whether or not the contact itself could be predicted using simply measured values. The bulk of the research was conducted using a simplified three dimensional computer model of a representative slab of tissue from the left atrium. Simply stated, the goal was to precisely control the angle and depth of the catheter, measure the resulting impedance, and identify any trends that should arise. The modelling results strongly suggest that both the electrical and thermal responses of the model depend heavily on the amount of the catheter, which is a function of both angle and depth, that is in contact with the tissue at any given time. The impedance measured during experiments hasn’t exhibited an angle dependency to the same degree as the theoretical ones however this is likely a byproduct of mechanical deformation that occurs in the tissue; more experiments are needed to investigate this further. This work serves to provide a framework going forward in improving the efficacy of the procedure: good contact is crucial in order to deliver quality lesions and that contact can be assessed using
measured electrical impedance. In the end, the specific catheter penetration depth
and angle matter less than the resultant amount of contact made.

6.1 Significant Findings

The prevailing wisdom in the ablation community is that it is catheter penetration
depth that affects measured impedance and lesion formation yet angle and catheter
orientation is rarely considered. This is a glaring omission considering the challenges
inherent in positioning the catheter. In Chapter 3, a simple model of the left atrium
was developed and the catheter’s position was permuted through a representative set
of angle and penetration depths and the resulting complex impedance was measured.
As expected, the impedance increased similarly to other publications but what was
perhaps the most interesting was that the angle that catheter makes can contribute
significantly to the measured electrical impedance. Also, it was found that the strictly
angle dependent impedance variation increases as the penetration depth increases.
This strictly angle related change ranges from less than 1% when the catheter is in
the blood pool to over 20% when the catheter is deeper into the tissue. It turns
out that these differences in measured impedance, seemingly due solely to changes in
angle (taken at a particular penetration depth), can be attributed to the change in
the catheter contact area with the tissue. The greater the contact, the greater the
impedance. This idea is further reinforced in simulations where the contact is held
roughly constant but have different combinations of angle and depth (to make the
same contact percentage) were run: it was found that they have the roughly the same
impedance.

In all simulations performed, both the magnitude and phase shift were calculated.
This was done in order to establish whether or not the precise catheter angle and depth could be calculated from the extra information provided by the real and imaginary components of the calculated impedance. However, the same increasing exponential trend with catheter area was observed in the real and imaginary components meaning that no extra useful data can be gleaned from studying both components. The larger dynamic range of the real component of the impedance makes it easier to detect when a change in contact has occurred.

Another interesting finding of this study was when the catheter was fully engaged in the atrial tissue, the impedance was found to deviate from the increasing exponential trend with contact area that was observed at lesser penetration depths. This was theorized to be an artifact of the catheter being close to penetrating the tissue making it both physically closer to the dispersive electrode and closer to the epicardial blood pool (where the resistance is lowest). This theory was confirmed when additional simulations were run with a thicker tissue slab. In these simulations, the impedance continued to increase exponentially until the same penetration effect was observed at a corresponding deeper depth. This finding is significant because complex structures present on the endocardial surface of the atria: trabeculated spaghetti like structures on a paper thin wall.

Experimental results taken using real tissue show that the values presented in the computer modelling study are in a reasonable range and that impedance does increase with increased contact. Unfortunately, there wasn’t enough experimental data gathered to say this conclusively and more experiments are pending.

While some of the results presented outlining the relationship between catheter contact and electrical impedance have been inferred or suggested in other works,
there has been precious little work studying the effect that catheter contact has on a lesion’s thermal progression. In Chapter 4, the electrical solution presented in the previous chapter was re-purposed in order to calculate important thermal statistics such as width, depth, volume and maximum temperature in the solution region. The simulations show that the angle that the catheter makes with the tissue makes a significant impact on the created lesion (volume, width and depth) and maximum temperature reached in the tissue. This effect that angle has is unchanged by the huge range of potential convection rates that can exist physiologically however, the lesions progress more rapidly when the heat is dissipated at a lower rate.

As with the electrical results, it was found that the angle dependency was mostly due to the amount of contact made with the tissue. As catheter contact area increases, the recorded lesion volume increased and ablation power decreased (during a constant voltage procedure). However, unlike in the electrical simulations and in what is perhaps the most significant finding of the study, there was an interesting exception from this trend: the maximum temperature inside the solution volume did not increase with increasing catheter contact area as much as with increasing depth. That is to say that catheter angle didn’t really have much of an impact on the maximum temperature compared to that seen when increasing the penetration depth. This is a finding that would have to be confirmed experimentally but could have some interesting clinical applications if a modality to extract the precise catheter depth and angle from an impedance (or other) measurement.
6.2 Clinical Relevance

The work presented in this thesis was predominantly a theoretical biophysical modelling study. Despite not including extensive experimental data, new insights arise that may be transferable to a clinical setting. In this study, a framework for being able to predict the contact that a catheter makes with the endocardial surface is presented. Being able to predict the contact and what the resulting lesion will be using electrical impedance would be an extremely important breakthrough in helping this procedure to become more effective at treating AF. If a physician were armed with the knowledge of the current state of contact, they could adjust the tip until suitable contact were obtained or an algorithm could be developed to automatically adjust the applied power such that conditions were optimal for transmural lesion formation (and resulting electrical isolation). This would lower the degree of difficulty of the procedure which would speed up procedures, increase success rates and keep patients safer.

The concept of impedance based contact assessment is an elegant one: faced with an electrical problem, it makes sense that an electrical parameter be best suited to determining how the catheter is coupled to the physiology, not to mention that the electrical impedance is a parameter that is already being measured. However, given the difficulty of establishing this relation in in-vivo and in-vitro trials, some research groups have shifted their focus to other modalities such as force based contact sensing. Once again, the complex structures of the atria conspire to make this a tough nut to crack. Here again, the ability of measured impedance to give an idea of the amount of the catheter shines in a way that force couldn’t: scenarios abound in which the force could be small and yet contact be high. Couple this reality with the fact that force
transducers dramatically increase procedural costs and the walls of the atria are so thin that perforation is a very real risk and the clinical advantage of using impedance to establish contact is evident.

The added understanding presented in this work of the thermal behaviour of the tissue given a proposed catheter configuration is significant, particularly when coupled with a potential scheme to approximate the contact. During a typical procedure, temperature is a value that is constantly monitored yet it is measured at the surface of the tissue. This is the point that is exposed to the greatest degree of cooling and is consequentially several degrees cooler than the maximum temperature detected in the volume. With a more accurate contact assessment and understanding of how lesions form at a given contact, the procedure could be tailored to keeping the peak temperatures below dangerous levels.

As a result of performing all simulations at a constant voltage, it was seen that despite having lower power, simulations with higher surface contact area created larger lesions and higher temperatures. This is a significant finding clinically because constant power ablation algorithms are quite common. Had the power been forced to be constant for all contact scenarios, the ones with higher contact would have had even larger lesions and higher temperatures. Given that contact can vary quite a bit due solely to the angle that the catheter is at and that angle and depth are difficult to control, using a constant power algorithm is akin to using a sledgehammer to swat a mosquito: fun but not necessarily the right tool for the job.
6.3 Limitations of the Study

The model described in Chapter 3 is motionless and uses properties that are valid only at a particular temperature. The actual procedure takes place on tissue that has neither luxury. As the tissue moves, it is possible (even likely) that the contact would change during a single ablation. The contact would have to be continuously assessed for more realistic applicability. One thing that makes this particularly difficult is that the electrical and thermal properties of the tissue change as it is heated up.

Another factor that limits the applicability of the experimental results is that the contact area cannot be precisely controlled as in simulation. The mechanical compliance of the tissue causes a sloping pit around the catheter, reducing the amount of the catheter that directly touches the tissue. Also, the surface of the tissue is actually quite complex in real life, it is quite prone to embedding itself in different surficial ridges or troughs in the tissue.

A better thermal model would include a separate boundary for the epicardial surface. As there isn’t constant blood exchange at the epicardial surface, heat would be dissipated at a slower rate. This could have a potentially serious impact on the recorded lesion size and depth (particularly at shallow catheter depths) as higher temperature would be allowed to migrate deeper into the tissue.

Of course with any simulation study, experimental results would be required to confirm the results. Thermal experiments are particularly difficult to achieve precise agreement with the model because only single point temperature readings can be taken and the tissue would have to be destroyed in order to do so. That being said, some experiments to show that the temperature progresses in a manner similar to that projected by the models is crucial verification to assert the angle dependence of
the thermal problem.

6.4 Future Work

Firstly, more experimental results must be performed in order to validate the existing simulation data. Using an optical technique, the area of catheter contacting the tissue can be quantified and the experimental impedance results compared against the simulated ones with proper contact areas.

Another advancement would occur on the model side, one that takes the mechanical deformation of the tissue into account. With a better representation of the tip/tissue interface, better accuracy of results can be obtained and it will be easier to compare experimental and simulated results (both electrical and thermal).

The tissue in a real-life ablation is moving and being heated throughout which would make the initial contact assessment invalid and any subsequent calculation could also be invalidated due to changing material properties. Experiments should be done to measure the complex impedance of the tissue during an entire ablation. Given that the imaginary term of the complex impedance is due to the capacitive structure of the cell membrane, it is possible that the capacitance will change as the tissue becomes progressively damaged. We know that the irreversible loss of electrical excitability that the tissue experiences at high temperatures is due to the denaturation of the cellular membrane; it would be interesting to see if the resistance to reactance relationship would remain the same. Answering this question would be relatively straightforward experimentally and could potentially unlock the ability to continuously track the catheter’s contact in the physically and thermally dynamic environment.
Another interesting application for the research presented in this study is an optimization problem wherein an algorithm for optimal energy delivery can be developed given a certain catheter contact. With the measured impedance as an input, the algorithm could vary parameters such as voltage, current limit, duty cycle and temperature settings in order to give the supplied heat enough time to flow deeper into the tissue (and not simply sear the surface) at a given contact.

The reality is, despite the significant amount of new insights presented by the models in this thesis, we have only begun to scratch the surface of what can be done in this field!
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Appendix A

Model Discretization and Minimization Analysis

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Table A.1: Statistics of the different gridding schemes in the case with catheter penetration depth set to 1.6mm and incident angle of 90°
(a) Resistance and Reactance as a function of grid discretization density.

(b) Impedance magnitude and phase shift as a function of grid discretization density.

Figure A.1: Plots of complex impedance quantities vs. grid discretization density. Test case shown is when catheter is at 1.6mm penetration depth and at 90°.
(a) Resistance and Reactance as a function of grid discretization density.

(b) Impedance magnitude and phase shift as a function of grid discretization density.

Figure A.2: Plots of complex impedance quantities vs. grid discretization density. Test case shown is when catheter is at 3.2mm penetration depth and at 15°.
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Table A.2: Statistics of the different gridding schemes in the case with catheter penetration depth set to 3.2mm and incident angle of 15°

Where BR refers to reduced baseline resolution, NL uses no special local grid for the catheter tip, RD1,2 and 3 have successively reduced discretization on the catheter tip while RD4,5 and 6 use fewer voxels to resolve any curvature in the model. SR and WR change the way that any slot or width span are resolved; there is no change due to either of these due to the lack of slots or spans in the model.
Appendix B

Key Indicators of Lesion Formation

Lesion Width
Figure B.1: Progression of lesion width (in the catheter’s axis of rotation) vs. time in the tissue given a low flow condition.
Figure B.2: Progression of lesion width (in the catheter’s axis of rotation) vs. time in the tissue given a high flow condition.
Figure B.3: Progression of lesion width (perpendicular to the catheter’s axis of rotation) vs. time in the tissue given a low flow condition.
Figure B.4: Progression of lesion width (perpendicular to the catheter’s axis of rotation) vs. time in the tissue given a high flow condition.
## Compiled Table of Important Lesion Indicators

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<th>$T_{max}$ (°C)</th>
<th>Lesion Width (mm)</th>
<th>Lesion Depth (mm)</th>
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| 0.04      | 90        | Low  | 7.1875              | 62.51          | 4.1726             | 2.7313            | 27.8565                 |
|           |           | High |                     | 46.44          | 0                  | 0                 | 0                       |
|           | 75        | Low  | 7.1517              | 62.65          | 4.2751             | 2.7175            | 28.589                  |
|           |           | High |                     | 46.49          | 0                  | 0                 | 0                       |
|           | 60        | Low  | 7.1212              | 62.61          | 4.4613             | 2.7175            | 30.192                  |
|           |           | High |                     | 46.50          | 0                  | 0                 | 0                       |
|           | 45        | Low  | 7.0893              | 62.40          | 4.6138             | 2.7122            | 32.6765                 |
|           |           | High |                     | 46.50          | 0                  | 0                 | 0                       |
|           | 30        | Low  | 7.0403              | 62.59          | 5.0145             | 2.7109            | 39.2218                 |
|           |           | High |                     | 46.69          | 0                  | 0                 | 0                       |
|           | 15        | Low  | 6.9756              | 63.41          | 5.6915             | 3.2975            | 52.7438                 |
|           |           | High |                     | 47.12          | 0                  | 0                 | 0                       |

Table B.1: Lesion parameters before $T_{max} = 100^\circ C$ is reached. Penetration depths -4mm and 0.04mm.
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Table B.2: Lesion parameters before $T_{max} = 100^\circ C$ is reached. Penetration depths 0.8mm, 1.6mm and 2.4mm.
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Table B.3: Lesion parameters before $T_{\text{max}} = 100°C$ is reached. Penetration depths 3.2mm and 4mm.
# Appendix C

## Experimental Data

Raw experimental data as collected at St. Jude research facility.

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<th>Angle (°)</th>
<th>Resistance (Ω)</th>
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Table C.1: Experimental results
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Table C.2: Experimental results continued.
Appendix D

Publications & Presentations

Throughout this research project, several conference presentations and posters were created. This appendix features presentations and conference publications and a brief description of what is covered in each one.

Gallagher et al. (2010) Alberta Biomedical Engineering Conference


The methods and part of the results, as described in Sec. 3, was presented in poster form.

Gallagher et al. (2011) NFSI & ICBEM Conference


The methods and part of the results, as described in Sec. 3, were published as part of the conference proceedings. Also, a talk by the same name was given at the conference.

**Gallagher et al. (2011) EMBC Conference**


The methods and some of the results, as described in Sec. 4, were published as part of the conference proceedings. Also, a poster by the same name was presented at the conference.

**Gallagher et al. (2011) Alberta Biomedical Engineering Conference**


Further refined methods and results, as described in Sec. 4, was presented in poster form.