

Catheter Contact Geometry Affects Lesion Formation In Radio-Frequency Cardiac Catheter Ablation

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Abstract—One factor which may be important for determining proper lesion creation in an atrial ablation procedure is catheter-endocardial contact. Little information is available that relates geometric contact, depth *and* angle, to ablation lesion formation. We present an electrothermal computer model of ablation that calculates lesion volume and temperature development over time. The Pennes bioheat equation was coupled to a quasistatic electrical problem. This method simulates importantly, not just catheter penetration depth, but also several different incident catheter angles as may occur in practise. Results show that for deeply penetrating tips, greater catheter angles reduce the rate of temperature buildup, allowing for larger lesions to form before temperatures become dangerous. It was also found that greater penetration may not lead to greater transmural penetration in lesion formation. We conclude that catheter contact angle plays a significant role in lesion formation, and the time course must be considered. This is clinically relevant because it makes proper identification and prediction of geometric contact variables a necessity in order to improve ablation efficacy and safety.

I. INTRODUCTION

Radio-Frequency (RF) ablation is a minimally invasive interventional technique that has seen a steady rise in popularity and can be effectively used to treat many different conditions. Of particular interest is the demonstrated efficacy in treating cardiac arrhythmia, specifically atrial fibrillation (AF) [1]. In the procedure, a physician advances the catheter to the target site in the heart through a venous access point. The catheter tip is maneuvered to the target endocardial site with the aid of fluoroscopy. Once in place, an RF current is applied which flows between the catheter and a dispersive patch electrode placed on the surface of the patient's body. This current heats the myocardial tissue, via the Joule effect, to temperatures in excess of 50°C at which point cellular necrosis occurs (caused by protein denaturation of the cellular membrane) which results in a permanent loss of electrical excitability [2]. In the case of atrial ablations, the goal is often to isolate the pulmonary veins and disrupt the macro-reentrant circuits that AF requires.

Unfortunately, the success of this procedure is entirely dependent on full transmural lesion formation [3] for electrical isolation. If too much energy is applied, there are the serious

risks of perforating the atrium or creating an embolism [4]. 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 [5].

The purpose of this study is to relate catheter angle and penetration depth by constructing an electrothermal computer model of atrial ablation and generating temperature distributions for various catheter angles and penetration depths.

II. METHODS

A. Model Construction

Both the electrical and thermal portions of the models were constructed using SEMCAD (Schmid & Partner Engineering AG, Switzerland) . A homogeneous slab of tissue $100\text{mm} \times 100\text{mm} \times 4\text{mm}$ thick was suspended in a blood solution measuring $150\text{mm} \times 150\text{mm} \times 75\text{mm}$ with the catheter incident upon the center of the slab with a dispersive patch electrode 4mm below the tissue. The catheter modeled is a standard clinical 7F, 4mm 485kHz ablation catheter. Coupled electrical and thermal simulations were run for catheter/tissue interface angles of 15° , 30° , 45° , 60° , 75° and 90° and penetration depths of -4mm (in the blood pool), 0.04mm , 0.8mm , 1.2mm , 2.4mm , 3.2mm and 4mm (fully engaged in the tissue). Catheter angles were defined relative to the surface normal so that an angle of 0° was perpendicular to the surface.

B. Electrical Problem

A Finite Element (FE) based low frequency solver was used to solve the electrical problem. Since the dimensions of the problem are small relative to the wavelength, the

Electroquasistatic assumption was used, allowing decoupling of the electric and magnetic fields by setting the time varying magnetic field to zero. This assumption allowed the transition from a FDTD solution to a frequency domain FE solution. All that is required is to solve the Laplacian (equation 1):

$$\nabla \cdot \tilde{\epsilon}_r \nabla V \quad \text{where : } \tilde{\epsilon}_r = \epsilon_r \epsilon_o - j \left(\frac{\sigma}{\omega} \right) \quad (1)$$

No flux boundary conditions were imposed at all edges and the problem space was large enough that boundary effects were not observed. Permittivity and conductivity of the tissue and blood were calculated using multiple Cole-Cole dispersions[6], evaluated at 485 kHz (see table I) and used in simulations.

C. Thermal Problem

To solve the thermal problem, a conventional FDTD solver is used to solve the Pennes bioheat equation (equation 2).

$$\rho c \frac{\partial T}{\partial t} = \nabla \cdot k \nabla T + q - Q_p + Q_m \quad (2)$$

Where ρ is the density, c is the specific heat capacity, k is the thermal conductivity, q is the source term from the RF power deposition. The blood perfusion heat loss (Q_p) in the myocardial tissue is modelled using a perfusion rate of 1130 ml/min/kg [7] and the metabolic heat generation rate (Q_m) is taken at 9W/kg. The physical material properties used in the model are shown in Table I and were taken from literature [8][9][10].

D. Thermal Boundary Conditions

The initial temperature in the entire model was set to 37°C. Convective film coefficients were used to model the interface between the catheter tip and the surrounding blood pool (h_{elec}) and the interface between the myocardial tissue and the blood pool (h_{tiss}). These are required to adequately

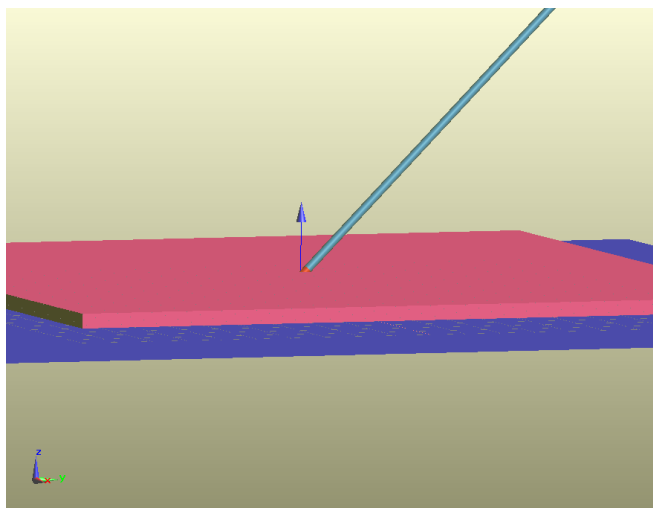


Fig. 1. **Sample ablation model setup.** Dispersive patch electrode is on bottom in blue, catheter body is in light blue, catheter tip is red and myocardial tissue is pink. Blood fills the rest of the volume. Scenario shown has catheter at 3.2mm depth and 45° angle (calculated from the tissue surface normal).

TABLE I
MATERIAL PROPERTIES USED IN ELECTRICAL AND THERMAL PROBLEMS

Material	c J/Kg·K	k J/Kg·K	ρ J/Kg·K	σ (J/Kg·K)	ϵ_r J/Kg·K
Pt-Ir	132	71	21.5e3	4e6	NA
Blood	4180	0.543	1000	0.7459	4227.12
Myocardium	3200	0.585	1200	0.2790	3332.32
Polyurethane	NA	NA	NA	4.7e-4	2.54

TABLE II
CONVECTIVE FILM COEFFICIENTS USED IN MODEL

Interface	Symbol W/m ² ·K	h
Catheter tip/blood pool	h_{elec}	5748.1
Myocardium/blood pool	h_{tiss}	5350.0

consider the rate of cooling that occurs due to the blood flow on both the endo- and epicardial surfaces of the heart during an ablation. Many previous works use h_{tiss} film coefficients derived from typical blood flow velocities measured in the heart, or from measured film coefficients taken from physical rubber and plastic models, with values in the range of 44-2500W/m²K [9][10][11][12]. These values are quite low and found using calculations depending on laminar flow, which is not applicable in the left atrium. We used a value of 5350 W/m²K as determined by Tangwongsan et al. [13] from measurements of swine endocardial convective heat transfer coefficients taken on the lateral wall of the left atrium. To determine the convective coefficient of the electrode/blood pool interface, standard methods were implemented:

$$h_{elec} = \frac{Nu \cdot k}{d} \quad (3)$$

Where k is the thermal conductivity of blood, d is the diameter of the catheter and Nu is the Nusselt number:

$$Nu = 0.683 \cdot \left(\frac{\rho \cdot v \cdot d}{u} \right)^{0.466} \cdot \left(\frac{c \cdot u}{k} \right)^{0.333} \quad (4)$$

Where v is the flow velocity of blood (estimated at 24.4 cm/s [11]), and u is the viscosity of blood. The convective film coefficients used in the model are shown in Table II.

E. RF Ablation Mode and Analysis Metrics

A constant voltage ablation mode was implemented with the peak voltage set to 40V. The simulation was run for 60 s, the maximum rated time of the catheter modeled (St. Jude Therapy™Cool Path™Duo). Since myocardial injury begins to occur at $\approx 50^\circ\text{C}$ 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 when the maximum temperature calculated in the tissue volume was greater than 100°C after which the 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 (T_{max}), and the time taken to reach 100°C .

III. RESULTS

Over all depths and catheter angles simulated, the plots shown in Figure 2 are representative of results. These plots consider lesion volume (Figures 2(a) and 2(b)) and T_{max} (Figures 2(c) and 2(d)) as a function of time taken at two different catheter depths: 0.8mm and 4mm (fully engaged). At catheter penetration depths less than 0.8mm it was found that T_{max} barely exceeded the 50°C lesion formation temperature, even after a 60s ablation period. Correspondingly, the lesions were *far* too small ($< 35mm^3$) to offer the electrical isolation that was sought.

As can be seen in Figure 2(a), catheter/endocardial surface contact angle plays a significant factor in lesion volume, particularly as the ablation time increases. This effect is not nearly as apparent for T_{max} (Figure 2(c)) where the angle related temperature difference is merely a few degrees. Another thing to note is that for a catheter penetration depth of 0.8mm T_{max} never reaches 100° and a transmural lesion (a full 4mm lesion) is never formed (plot not shown).

When the catheter is fully engaged (4mm penetration depth) a transmural lesion is achieved at all catheter/endocardial surface angles. Also, the lesion volume (Figure 2(b)) and T_{max} (Figure 2(d)) plots continue to show a fairly strong angle dependency. Perhaps most telling however, is the fact that T_{max} reaches 100° nearly 8 seconds sooner at 30° than at 90°. This restricts the lesion formation at 30° since ablation must be stopped before T_{max} exceeds 100°C, which is well before the maximum lesion size.

Comparing Figures 2(a) and 2(b), it is clear that penetration depth also has a significant effect on lesion volume as expected. A deeper penetration leads to a larger and more transmural lesion.

IV. DISCUSSION

It is apparent how important catheter contact geometry is for determining lesion formation. A minimum penetration depth of 0.8mm is required for transmural lesion formation. At greater depths, catheter angle plays a significant role in determining both the resulting lesion volume and maximal temperature in the tissue.

For ablations where the catheter is at the same penetration depth the lesion volume and T_{max} , can be significantly different depending on angle. Given the comprehensive set of simulations run, it has been demonstrated that while T_{max} increases markedly with increasing catheter penetration depth, catheter angle does contribute significantly and that this increase in T_{max} does not necessarily equate to higher lesion volume. In fact, when the catheter is fully engaged with the myocardium at an angle of 30°, the lesion volume only reaches 51.75 mm^3 before T_{max} exceeds 100°C. This is compared that to a lesion volume of 114.5 mm^3 for a fully engaged catheter at 90° when T_{max} exceeds 100°C. This study directly challenges the common perception that increased catheter penetration depth unequivocally leads to increased lesion volume. Despite only being at a penetration depth of 0.8mm, the resulting lesion volumes ranged from 52.66 mm^3 at a catheter angle of 75° to 76.86

mm^3 at a catheter angle of 15°; both larger than that of the fully engaged catheter at 30°.

Furthermore, this study shows that the time course of the lesion formation must be followed in order to properly determine the maximum lesion volume which can be safely obtained. Considering only steady state values does not disclose when the temperature reaches 100°C and ablation must stop. Even though the final lesion size may be larger if a current had been applied indefinitely, it may be severely limited in the period over which it may be applied due to temperature buildup.

Admittedly, when T_{max} 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 run away quickly, and that this rate depends strongly on catheter contact angle.

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.

V. CONCLUSIONS

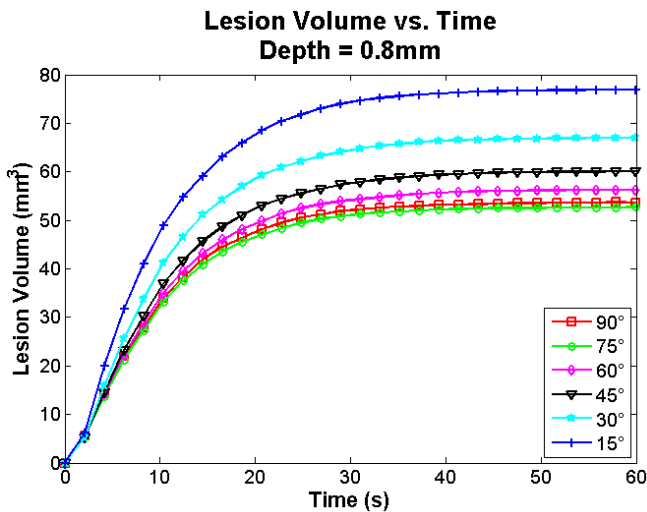
It was found that catheter contact angle (not just penetration depth) plays an integral role not just in lesion formation, but also in patient safety. Generally, as the angle that the catheter makes with the tissue becomes more acute (approximately 30° is optimal), the maximal temperature in the tissue 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.

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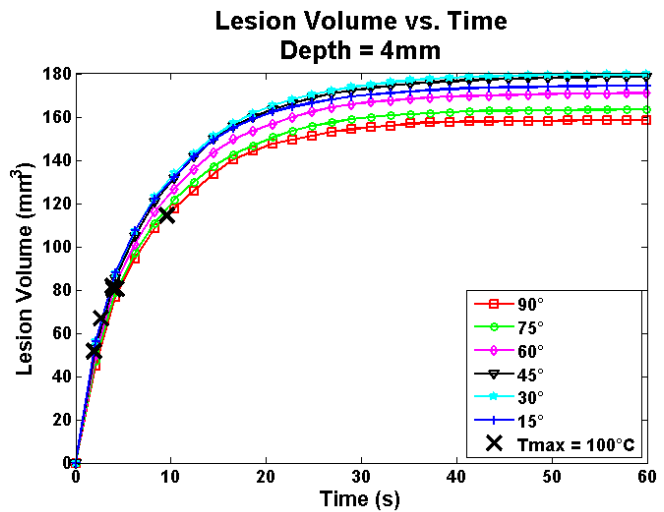
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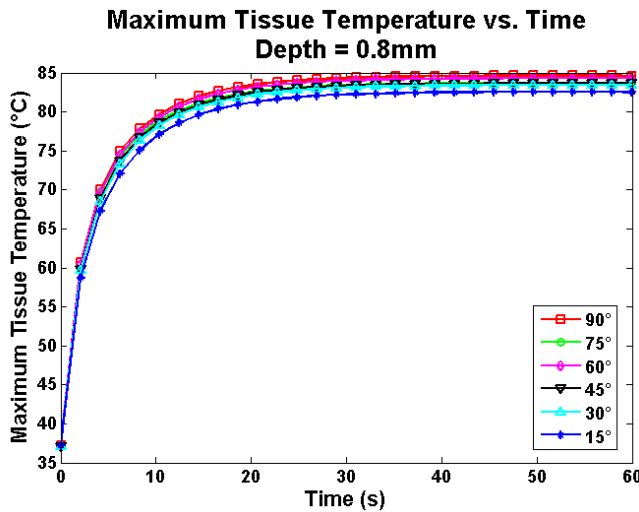
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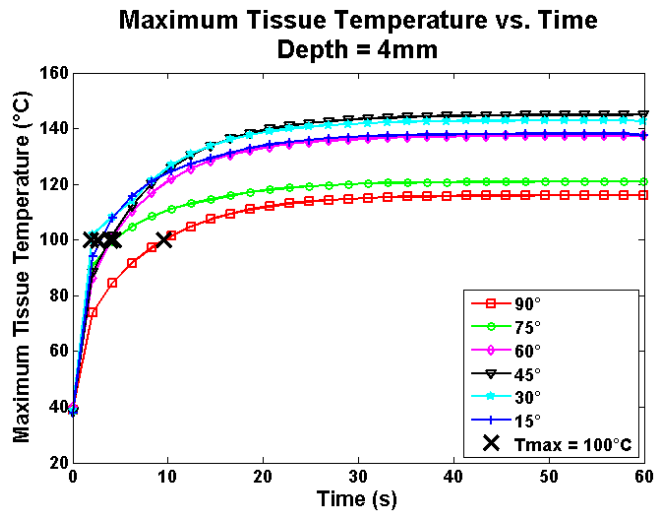
(a) Plot of lesion volume vs. time when catheter penetration is 0.8mm.



(b) Plot of lesion volume vs. time when catheter penetration is 4mm.



(c) Plot of Tmax in the tissue vs. time when catheter penetration is 0.8mm.



(d) Plot of Tmax in the tissue vs. time when catheter penetration is 4mm.

Fig. 2. Lesion volume and maximum tissue temperature depend on catheter angle and penetration depth. Plots showing Lesion Volume and maximum temperature in the tissue vs. time at difference catheter contact angles. 2(a) and 2(c) are from ablations with a catheter penetration depth of 0.8mm while 2(b) and 2(d) are from ablations where the catheter is fully engaged (4mm). Small black 'x' marks indicate the time at which Tmax inside the tissue has reached 100°C.

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