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Abstract. In spite of significant efforts to improve image-guided ablation therapy, a large number of patients undergoing ablation therapy to treat cardiac arrhythmic conditions require repeat procedures. The delivery of insufficient thermal dose is a significant contributor to incomplete tissue ablation, in turn leading to the arrhythmia recurrence. Ongoing research efforts aim to better characterize and visualize RF delivery to monitor the induced tissue damage during therapy. Here, we propose a method that entails modeling and visualization of the lesions in real-time. The described image-based ablation model relies on classical heat transfer principles to estimate tissue temperature in response to the ablation parameters, tissue properties, and duration. The ablation lesion quality, geometry, and overall progression are quantified on a voxel-by-voxel basis according to each voxel's cumulative temperature and time exposure. The model was evaluated both numerically under different parameter conditions, as well as experimentally, using *ex vivo* bovine tissue samples undergoing *ex vivo* clinically relevant ablation protocols. The studies demonstrated less than 5°C difference between the model-predicted and experimentally measured end-ablation temperatures. The model predicted lesion patterns were within 0.5 to 1 mm from the observed lesion patterns, suggesting sufficiently accurate modeling of the ablation lesions. Lastly, our proposed method enables therapy delivery feedback with no significant workflow latency. This study suggests that the proposed technique provides reasonably accurate and sufficiently fast visualizations of the delivered ablation lesions. © 2018 Society of Photo-Optical Instrumentation Engineers (SPIE) [DOI: [10.1117/1.JMI.5.2.021218](https://doi.org/10.1117/1.JMI.5.2.021218)]

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1 Introduction

Cardiac ablation therapy entails the navigation of a catheter into the heart and delivery of RF energy to the tissue to locally heat and destroy a small tissue region, to electrically isolate the regions that generate or propagate arrhythmia. Several medical imaging modalities, including computed tomography (CT), magnetic resonance imaging (MRI), ultrasound (US), and most often x-ray fluoroscopy, are used to guide the catheter to the target location inside the beating heart.¹ Although ablation therapy has received extensive attention for the treatment of arrhythmic conditions, and, in particular, atrial fibrillation, reported ventures into the treatment of some cardiac arrhythmias have been somewhat discouraging. Successful treatment is accompanied by lengthy procedures and prolonged exposure to ionizing radiation.² Moreover, studies have also shown that 30% to 50% of patients undergoing ablation treatment require repeat procedures, whereas others may experience complications, such as cardiac perforation and tamponade, and atrioesophageal fistula formation when ablating in the posterior wall of the left atrium.^{3,4}

We hypothesize that two complementary factors have hampered the success of cardiac catheter ablation therapy: suboptimal visualization and navigation inside the beating heart to enable effective targeting of the desired sites; and lack of adequate monitoring of the thermal damage induced during ablation to ensure irreversible tissue injury. As a result, the tissue is instead only temporarily inflamed as a result of the ablation, and, in time, it regains its viability and the arrhythmia recurs, raising the need for a repeat procedure. In addition, catheter–tissue contact plays a major role in the delivery of fully developed, irreversible lesions toward achieving contiguous, transmural ablation patterns. Since electrode–tissue contact directly impacts the heat flux into the tissue, but contact area is difficult to measure, contact force has been used as a contact surrogate. There is evidence most specifically from the TOCCATA trial⁵ that contact force is a relevant measure, and the challenge of using contact force in a low-volume center (as described in Ref. 6) has also been raised. Regardless of how improved contact may be achieved and measured, the success of the ablation depends on characterizing lesion quality and ensuring the delivery of irreversible lesions, which are in turn impacted by the electrode–tissue contact. As such, here we consider the effects of the

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electrode–tissue contact together with lesion and tissue injury characterization.

Extensive research has been conducted on improving visualization and guidance to facilitate catheter navigation inside the heart. Examples include the registration of single-phase preoperative models to the intraoperatively reconstructed geometries,⁷ complemented with sparse two-dimensional (2-D) contours of the left atrium⁸ depicted using 2-D intracardiac echocardiography (ICE). A similar system was the advanced prototype platform for image-guided cardiac ablation therapy described by Rettmann et al. that incorporates dynamic, preoperative, subject-specific anatomical cardiac models, tracked electrophysiology data, and tracked real-time US imaging registered to the patient.^{9–11} Chu et al.¹² conducted EP studies and subsequent ablation procedures under ICE guidance; Dickfeld et al.¹³ and Lardo et al.¹⁴ leveraged the use of interventional MRI technology for intraoperative visualization of catheter navigation and monitoring of therapy delivery; and Razavi et al.¹⁵ demonstrated improved intraoperative visualization under combined MR-fluoroscopy guidance. Lastly, electroanatomical mapping (EAM) systems, of which three are routinely employed in the clinic (CARTO—Biosense-Webster Inc., Diamond Bar, California; EnSite NavX—St. Jude Medical, St. Paul, Minnesota; and Rhythmia—Boston Scientific, Marlborough, Massachusetts), complement fluoroscopy imaging with electroanatomical models intraoperatively generated using the tracked catheter. Nevertheless, these models are somewhat coarse, as not all endocardial sites can be reached and not all recorded locations truly belong to the endocardial wall.

Once on target, RF energy is delivered to the tissue. The ablation lesion represents the tissue region that has undergone irreversible injury, and its quantification entails accurate assessment of the thermal dose received by the tissue. If available in near real-time, such information could significantly enhance the cardiologist's ability to achieve optimal therapy outcome. Nevertheless, without the ability to adequately monitor the thermal dose delivered to the tissue to ensure irreversible damage, ablation is often incomplete. Hence, the delivery of often inadequate lesions is a direct result of current image-guided ablation systems not conveying sufficient intraprocedural information to characterize the delivered lesions.

Traditional EAM systems (Carto, EnSite NavX, Rhythmia, etc.) depict the delivered ablation lesions by marking the locations visited by the tracked catheter with spherical glyphs. However, neither the size nor the color of these generic markers correlates with the size or quality of the lesion,¹⁶ hence providing no information on the lesion quality. In addition to the glyphs that mark the “ablated” locations, extensions of the traditional EAM systems, such as the VisiTag technology,¹⁷ show the user a “gauge” whose color changes according to the ablation index estimated at a specific location based on power, contact force, and ablation time.¹⁸ This technology presents an improvement over the traditional EAM systems, as it provides feedback based on contact force and ablation duration. Although better contact and longer ablation are known to impact lesion transmural, the force-time gauge display is based on force and time thresholds empirically defined by the user according to their experience, instead of thresholds chosen according to validation experiments that confirm irreversible, complete ablation under these conditions.

Since direct temperature monitoring and lesion characterization is challenging, several direct sensing-based surrogate

methods have been proposed. Infrared thermometry is limited to surface visualization and provides no feedback or visualization on lesion transmural.¹⁹ Alternatively, several approaches have resorted to monitoring lesion formation using MR thermometry^{20–22} or delayed contrast-enhanced magnetic resonance imaging (DCE-MRI).^{23–25} However, these methods require access to interventional suites equipped with complex MRI infrastructure that infringe on the procedure workflow, its cost, and also on the wide clinical translation of these methods.

Elastography-based methods constitute concurrent alternative approaches that use tissue stiffness to characterize the extent of ablation relative to viable tissue. Current literature suggests that there has been progress on the front of magnetic resonance elastography (MRE),²⁶ acoustic radiation force imaging (ARFI), shear wave elasticity imaging (SWEI),^{27,28} and US-based intracardiac myocardial elastography²⁹ but no clear demonstration of their implementation. MRE requires extensive footprint including an MR scanner and a driver that would be workflow impractical, particularly with catheters in place.²⁶ ARFI and SWEI imaging have been demonstrated *ex vivo*, but their translation has yet to be shown.^{27,28} Although routinely used for catheter guidance, US imaging^{30–32} has shown limited success in imaging lesions^{33–35} as the echogenic properties of viable and ablated tissue are not sufficiently different to enable their differentiation. Moreover, while US elastography has been demonstrated *in vivo*,²⁹ it is best suited for left ventricular applications, mainly due to its inadequacy for thin left atrial wall tissue. Although at the forefront of the research field, elastography-based techniques also impose a significant technological footprint into the interventional suite. As such, their translation into clinical systems has yet to be demonstrated and is therefore still debatable. Thus, no direct sensing method is clinically available to provide feedback on lesion size, geometry, and extent of induced tissue damage during the ablation procedure.

Finite element models (FEMs) have been used to study lesion development in response to RF energy delivery. Several groups have developed mathematical and physiological modeling approaches¹ to investigate lesion development in response to RF energy delivery^{36,37} for cardiac arrhythmia treatment.^{38–41} Typical thermal ablation models take into account resistive heating near the electrode–tissue interface, conductive heating within the tissue, and convective cooling at the electrode–tissue interface. Other models are more complex, also incorporating the heat sink effect due to tissue perfusion when ablating highly vascularized sites.⁴¹ Moreover, most models mimic temperature-controlled ablation, where power delivery is modulated to maintain a preset target electrode temperature.⁴²

Despite their complexity and accuracy,^{43–45} most traditional RF ablation models⁴² have been hampered by their inability to compute lesion progression in real-time, rendering them as unusable for intraprocedural lesion monitoring. Moreover, although extensive research has been conducted on complex thermal modeling of RF ablation, little effort has been channeled toward the integration of these models into the clinical workflow to provide online feedback on tissue temperature distribution and induced tissue injury to avoid incomplete ablation. The slow progress has primarily been attributed to the challenge of developing sufficiently fast ablation models capable of computing and updating the tissue temperature distribution and lesion progression in real-time⁴² while also maintaining a high spatial and temporal resolution.

The premise of this work builds on (1) the need for thermal injury assessment/monitoring and lesion characterization to ensure complete ablation, (2) the limitations associated with current standard of care platforms as far as addressing these needs, and on the potential of nondirect sensing-based techniques, such as ablation lesion models for lesion characterization and visualization, to provide a means to monitor ablation lesion delivery.

Here, we propose a fast and accurate surrogate ablation model that relies on classical heat transfer principles to provide estimates of the temperature distribution, tissue damage, and lesion progression during ablation. Specifically, the paper describes an image-based ablation lesion model that (1) quantifies tissue temperature based on heat transfer principles and available input data and tissue parameters, (2) characterizes tissue injury and provides representations of lesion geometry and quality based on the estimated temperature and ablation duration (which are the factors that play into the thermal dose calculation, along with tissue properties), and (3) renders ablation lesions in a timely fashion for use during procedures. The methods are accompanied by both numerical verification against previously developed and disseminated ablation models, and also by validation experiments conducted in animal myocardial tissue samples in a convective saline bath at body temperature using direct temperature sensing probes for real-time temperature measurements and postablation assessment of the lesion.

Unlike previous studies, this paper does not describe the development of a thermal ablation model intended to substitute or compete with previously disseminated approaches that employ finite element methods to model the tissue and heat transfer phenomena in their full complexity.^{43,44,46} Rather, we describe a surrogate method that follows a fast, image-based implementation, leverages intraoperative information to characterize the induced thermal injury^{47,48} and generates visual representations of the created lesions. This lesion characterization and visualization tool could be potentially leveraged clinically by integrating it into existing platforms, such as VisiTag. This approach could represent an upgraded version of what VisiTag currently offers and provide simultaneous guidance on lesion delivery and characterization/monitoring during ablation.^{49,50}

2 Materials and Methods

2.1 Thermal Ablation Model

2.1.1 Governing principles

Tissue response to RF energy can be approximated with sufficient accuracy by a coupled resistive–conductive heat transfer process.¹ Our proposed surrogate ablation model incorporates a resistive component occurring at the electrode–tissue interface, and a conductive component responsible for energy diffusion into the tissue (Fig. 1). The heat transfer equilibrium in the tissue during ablation is governed by the bioheat equation⁵¹

$$\rho \cdot c \cdot \frac{\partial T}{\partial t} = \nabla \cdot (\kappa \nabla T) + q - Q_p + Q_m, \quad (1)$$

where ρ is the mass density (kg/m^3), c is the specific heat ($J/kg \cdot K$), κ is the thermal conductivity ($W/m \cdot K$); Q_p is the perfusion heat loss (W/m^3)—typically neglected for cardiac ablation,⁴⁵ unless the ablated area is highly vascularized, Q_m is the metabolic heat generation (W/m^3), which was shown to be insignificant,^{52,53} and, lastly, q is the heat source (W/m^3), representing the energy deposited into the tissue within a small

Fig. 1 Schematic diagram of the heat transfer processes during cardiac RF ablation. RF electrode is in contact with tissue resulting in resistive (Joule) heating at the electrode–tissue interface, and conductive heating further within the tissue. A fresh blood supply inside the cardiac chamber convectively cools the electrode–tissue interface. Moreover, provided large blood vessels are present in the vicinity, internal tissue perfusion might also contribute to heat dissipation from the ablation site. Adapted from Ref. 1.

radius around the active electrode surface and approximated by purely resistive and quasistatic Joule heating.

2.1.2 Model parameters, formulation, and image-based implementation

As opposed to solving the heat transfer equation on a discretized geometry using a traditional finite element mesh, the proposed ablation model was solved and implemented using an image-based approach, where the computational elements were the image voxels themselves: multiple image volumes, described later in more detail, are used to predict tissue temperature, extent of tissue damage, and lesion progression on a voxel-by-voxel basis during the ablation. The ablation electrode is represented by a virtual construct based on the physical properties of the catheter, and its interaction with the tissue is defined by means of voxel occupancy in the image volume.

Since energy dissipation within the tissue decreases rapidly with the distance from the electrode, the delivered power is absorbed within the first 1 to 1.5 mm from the electrode surface⁵⁴ through resistive heating. During clinical procedures, the energy delivered to the tissue is increased up to a set maximum power level, then adjusted to maintain the preset target electrode temperature⁵⁵ enforced to prevent char and coagulum formation on the electrode tip. Hence, as also suggested in Ref. 38, the power delivery to the electrode–tissue interface voxels can be modeled as a linear increase up to the maximum set power level. To maintain the preset target electrode temperature, our implementation modulates power delivery according to the gradient between the preset target electrode temperature and the dynamically updated electrode–tissue interface temperature. As such, the maximum prescribed power is delivered until the voxels in contact with the electrode reach the target temperature, at which point a power-damping factor is then applied to maintain the electrode voxels at or around the preset target temperature. Moreover, the endocardial blood–tissue boundary condition is modeled by updating the temperature of the blood pool voxels to 37°C prior to each computational cycle, mimicking a fresh blood supply at body temperature.

Tissue voxels remote from the catheter (i.e., beyond the resistive heating region immediately adjacent to the electrode) receive energy via heat conduction, modeled as a flow-based isotropic diffusion on the image volume. Tissue thermal conductivity has been traditionally treated as a constant parameter with a nominal value of $\kappa = 0.51 - 0.59 \text{ W/m} \cdot \text{K}$ for human myocardium.⁵⁶ Although this approach is a widely accepted approximation, in an effort to mimic the effect of temperature increase and tissue dehydration on conductivity, as suggested by Shahidi and Savard,⁵⁷ we also investigated the use of a temperature-variant tissue conductivity behavior as proposed by Valvano et al.⁵⁸ This approach uses a linear relationship $k = k_0 + k_1 \cdot T$, characterized by the k_0 and k_1 tissue-specific constants determined experimentally,⁵⁸ to describe tissue conductivity changes with temperature. Other parameters used in the model were assigned the literature-suggested average values: $\rho_{\text{blood}} = 1000 \text{ kg/m}^3$, $\rho_{\text{tissue}} = 1070 \text{ kg/m}^3$, $c_{\text{blood}} = 4180 \text{ J/kg} \cdot \text{K}$, and $c_{\text{tissue}} = 3500 \text{ J/kg} \cdot \text{K}$.⁵⁷

Unlike traditional FEM models, our proposed ablation model follows an image-based implementation. A preacquired anatomical three-dimensional (3-D) image—a subject-specific preoperative CT or MR image of the heart—is segmented into blood and tissue compartments, each of which is assigned its corresponding physiological parameters. After temperature initialization at 37°C, the following image volumes are used to evaluate tissue temperature, exposure (i.e., measure of tissue damage), and lesion progression during the ablation. The conductivity volume is defined by assigning all voxels in each image compartment—tissue and blood—their corresponding thermal conductivity values. An image volume of identical size is defined as the temperature volume by assigning each voxel its corresponding temperature value, first initialized at 37°C. The tissue exposure volume is a volume of the same size as the temperature volume in which each voxel is assigned its corresponding cumulative exposure. Cumulative exposure is defined as the area under the temperature–time curve or the cumulative sum of the temperature–time product during the ablation duration, as explained in the Sec. 2.1.3. Lastly, the tissue lesion volume is constructed based on the tissue exposure volume according to each voxel’s cumulative exposure relative to the cell-death cumulative temperature–time exposure threshold of 55°C for 5 s or longer, as explained in Sec. 2.1.3. Voxels of the tissue exposure volume are marked as part of either the irreversible lesion, provided their cumulative exposure exceeds the cell-death exposure threshold or reversible lesion penumbra otherwise.

2.1.3 Tissue damage and lesion characterization

Modeling studies have demonstrated that the ablation lesion—tissue rendered nonviable after RF ablation—is consistent with the volume enclosed by the 50°C isothermal surface.⁵⁹

A more traditional approach to quantify tissue damage, first introduced in Ref. 60, characterizes tissue injury [$\Omega(t)$] via an empirical function of the Arrhenius form Ref. 61

$$\Omega(t) = \int_0^t P \cdot e^{\frac{-\Delta E}{RT}} dt, \quad (2)$$

where T is the temperature (K) calculated at each point in the model region, R is the universal gas constant ($8.314 \text{ J/mol} \cdot \text{K}$), and P (s^{-1}) and ΔE (J/mole) are tissue-specific kinetic coefficients evaluated experimentally⁶² and validated against RF

ablation experiments.⁵³ According to this relationship, tissue damage accumulates linearly with time and hyperbolically with temperature, rendering tissue injury as reversible for $\Omega < 0.5$ and irreversible for $\Omega > 1$, resulting in 63% irreversible injury for $\Omega = 1$.

Labonté⁶³ demonstrated via computer simulations validated against RF ablation experiments⁵³ that the kinetic coefficients $P = 2.3921 \cdot 10^{92} \text{ s}^{-1}$ and $\Delta E = 5.8692 \cdot 10^5 \text{ J/mol}$ reasonably predicted tissue injury as reversible for $\Omega < 0.5$ and irreversible for $\Omega > 1.0$. Moreover, the critical cell-death temperature for heart tissue— $T_{\text{crit}} = \frac{\Delta E}{R \cdot \ln(P)}$ —computed using the above-mentioned kinetic coefficients is $\sim 58^\circ\text{C}$. These results confirm that, while extended exposure above 50°C would ensure irreversible damage, as suggested in Ref. 1, several seconds of exposure above 55°C would be sufficient to generate a well-developed lesion.

Nevertheless, tissue damage is known to be a function of both temperature and time⁴² and here we define cumulative tissue exposure as a measure of tissue injury. This new parameter quantifies and characterizes lesion irreversibility in a similar fashion to the Arrhenius-based tissue damage coefficient, however with a much higher computational efficiency, which is critical for the intended time-sensitive intraprocedural monitoring application.

Cumulative exposure is computed as the cumulative sum of the temperature–time product for each individual voxel in the temperature volume throughout the duration of the ablation. Cumulative exposure can be essentially interpreted as the area under the temperature–time curve for each voxel accumulated during the ablation cycle. However, not all tissue voxels exhibit sufficient exposure to undergo irreversible damage; as such, we estimate cumulative voxel exposure in two stages

$$E = \int_0^{t_{\text{cell-death}}} T(t) dt + \int_{t_{\text{cell-death}}}^{t_{\text{max}}} T(t) dt, \quad (3)$$

where $t_{\text{cell-death}}$ is the time at which a voxel reaches cell-death temperature ($T_{\text{cell-death}} = 55^\circ\text{C}$). The former term represents cumulative voxel exposure accumulated by a voxel prior to cell-death and is classified as reversible damage, rendering such voxels as part of the reversible lesion penumbra. The latter term represents the cumulative voxel exposure beyond cell-death (i.e., exposure to temperatures above the 55°C cell-death temperature for 5 s or longer) and labels the voxel as irreversibly damaged and a part of the irreversible lesion core.

In Sec. 3.1.2, we verify and confirm through numerical simulations that our proposed cumulative exposure criterion indeed yields irreversible tissue damage; both the Arrhenius and the 50°C isotherm tissue injury criteria yield consistent results with our proposed exposure criterion, yet at a higher computational expense.

To summarize, our image-based model implementation stores tissue temperature distribution in the time-variant tissue temperature volume, which constitutes the basis for estimating the tissue injury that is stored in the time-variant tissue exposure volume. Corresponding voxels in the tissue exposure volume are digitally marked in the tissue lesion volume according to their cumulative exposure relative to the cell-death threshold (55°C or higher for 5 s or longer) as either irreversible lesion or reversible lesion penumbra.

2.2 Model Robustness Evaluation

We conducted numerical simulations to assess the sensitivity of the model to several parameters, including tissue thermal conductivity, tissue damage criteria, and electrode-tissue contact. Moreover, we also conducted numerical simulations aimed at comparing the size and geometry of the irreversible lesion core predicted by each of the three tissue injury criteria: the Arrhenius-based tissue damage coefficient, the volume enclosed by the 50°C isothermal surface, and the computationally efficient cumulative exposure proposed here. Lastly, we also simulated a cooled versus dry electrode ablation, as described in Sec. 3.1.4.

2.3 Ex Vivo Experimental Evaluation

To validate our model against physical measurements, we conducted a series of ablation experiments using *ex vivo* bovine tissue samples. The 1-cm-thick muscle samples were submerged in a convective 0.9% saline bath. Endocardial blood flow was emulated using a variable flow-rate immersion circulator (VWR Scientific, Batavia, Illinois) that maintained the saline bath at a constant temperature at $36 \pm 0.5^\circ\text{C}$ with a 6 L/min flowrate. Ablation lesions were delivered using a 9F 4-mm temperature-feedback electrode catheter vertically displaced into the tissue by 1.5 mm, such that its tip achieved firm, hemispherical contact with the tissue. The RF power generator (EPT-1000 XP, Boston Scientific, Natick, Massachusetts) was operated in the temperature-control mode with a 60°C and 90°C target electrode temperature and 40-W maximum power level. To further emulate the clinical protocol, the ablation duration was set to 60 s and typical impedance values averaged 80 to 100Ω. Direct tissue temperature measurements, considered as ground truth, were recorded using focal 0.8-mm-diameter fiberoptic temperature probes (OpSens Inc., Quebec City, Canada) inserted in the samples at prescribed locations relative to the ablation electrode using a grid template (Fig. 2). Each sensor provided a precision of 0.1°C with a 50-Hz sampling rate. Three experiments were conducted at each experimental condition, and result variables were reported as mean and variance.

In lieu of an anatomical image, a synthetic 0.5-mm isotropic image was generated comprising two compartments, tissue and saline solution, labeled as tissue and blood, respectively. Material properties were assigned to each compartment according to the literature-suggested physiological parameters listed in Sec. 2.1.2. In addition to the tissue and blood temperature volumes initialized at 37°C, a tissue conductivity volume, exposure volume, and lesion volume of the same size as the tissue temperature volume were used to initialize the corresponding image volumes, all of which were subsequently updated during the ablation, yielding the estimated tissue temperature, conductivity, exposure, and ablation lesion. Convective cooling was modeled by reinitializing the blood temperature volume to 37°C at the start of each computational cycle.

3 Model Evaluation and Results

3.1 Numerical Verification

3.1.1 Temperature effects on tissue conductivity

Tissue conductivity has traditionally been treated as a constant parameter typically in the range of $\kappa = 0.51$ to $0.59 \text{ W/m} \cdot \text{K}$ range for human myocardium, with a nominal value of $0.55 \text{ W/m} \cdot \text{K}$.⁵⁶ However, to explore the effect of temperature increase and dehydration,⁵⁷ we investigated the temperature-variant tissue conductivity in our proposed model according to the linear criterion proposed in Ref. 58: $\kappa(T) = k_0 + k_1 \cdot T$, with the k_0 and k_1 tissue-specific constants determined experimentally.⁵⁸

We studied the effect of temperature-variant tissue conductivity by assessing tissue temperature at two different locations—1 and 3 from the catheter tip—following a 90-s temperature-controlled ablation at 90°C. Figure 3 shows that the constant tissue conductivity model across the 0.51 to $0.59 \text{ W/m} \cdot \text{K}$ range yielded an end-ablation temperature difference of less than 5°C at both locations compared to the linear temperature-variant tissue conductivity ($k = k_0 + k_1 \cdot T$) model. Similar studies conducted in Ref. 57 showed end-ablation tissue temperature variations of $\sim 2^\circ\text{C}$ at 1.1 and 3.3 mm from electrode

Fig. 2 Schematic diagram of the experimental setup for model evaluation during the RF ablation of *ex vivo* bovine samples. (a) Fiberoptic temperature probes were inserted at various locations within the tissue samples (x, y, x, w); (b) Experimental apparatus showing a tissue sample submerged in a convective saline bath maintained at 37°C. A grid template is used to position the fiberoptic probes used for direct temperature measurements at desired locations with respect to the ablation electrode.

Fig. 3 Model-predicted tissue temperature at 1 and 3 mm from electrode tip for constant tissue conductivity (0.51, 0.55, 0.59 W/m · K), as well linear temperature-variant tissue conductivity ($k = k_0 + k_1 \cdot T$). Minimal end-ablation tissue temperature variations (5°C or less) were observed, consistent with the findings disseminated in Ref. 57.

and insignificant differences in lesion dimension, suggesting that a constant tissue conductivity model is sufficiently accurate in predicting end-ablation tissue temperature distribution.

3.1.2 Tissue damage criteria

Since the intended application of the proposed lesion characterization and evaluation method is for intraprocedural monitoring use, computational efficiency is a key factor. To enable sufficiently fast computation and rendering of the lesion, we proposed the cumulative exposure metric as a surrogate to characterize lesion reversibility, as the estimation of the cumulative temperature–time product on a voxel-by-voxel basis is more computationally efficient than estimating the Arrhenius tissue damage coefficient. Moreover, although the 50°C isotherm damage criterion has been accepted as a sufficiently faithful lesion reversibility threshold, given the inherent uncertainties associated with the estimation of tissue temperature, the cumulative exposure threshold that enforces exposure to 55°C provides a more conservative criterion for ensuring lesion irreversibility.

To confirm the proposed cumulative exposure metric as a viable criterion for ensuring lesion irreversibility vis-a-vis the other established tissue injury criteria, we compared the geometry of the tissue injury characterized according to our cumulative exposure formulation (irreversible damage above 55°C for 5 s or longer), to the tissue injury geometry predicted by the traditional Arrhenius tissue damage coefficient (irreversible for $\Omega > 1$), and the 50°C isotherm criteria.

Figure 4 shows that the diameter of the irreversibly damaged tissue region predicted by the cumulative voxel exposure criterion is within 1 mm (the spatial resolution of the image data) from the irreversible region predicted by the Arrhenius and the 50°C isotherm criteria. Moreover, the latter two criteria show consistent irreversible lesion size, within 1 voxel difference.

3.1.3 Electrode–tissue contact

As suggested in Ref. 64, electrode contact is a direct indicator of the lesion size,⁶⁵ as suboptimal contact may lead to inadequately developed lesions.⁶⁶ We addressed the electrode–tissue contact by modeling three different cases of electrode penetration depth and assessed the corresponding lesion geometry (i.e., depth, width, and volume).

Figure 5 shows the lesion depth, diameter, and volume versus electrode penetration depth—a surrogate for electrode–tissue contact employed in Ref. 67—predicted by our model and confirms increasing lesion diameter, depth, and volume with electrode penetration depth.

3.1.4 Cooled versus dry electrode ablation

In addition to modeling traditional ablation catheters with no active cooling, we employed the approximation adopted in Refs. 68 and 69 to model a cooled ablation procedure and assessed its effects by characterizing the resulting lesion. The active cooling was modeled by setting a temperature boundary condition at the electrode surface equal to the temperature of the cooling/irrigation fluid. While we recognize the potential limitations of this model, the numerical studies have shown that this first-order approximation provides sufficient fidelity.

Figure 6 shows the model-predicted 90-s end-ablation tissue temperature at 1 and 3 mm away from the electrode under three

Fig. 4 (a) Comparison of the irreversibly injured tissue region assessed according to the three tissue injury criteria. Irreversible lesion diameter predicted by the three injury criteria; (b) the voxel exposure criterion proposed in our model predicts an irreversible lesion diameter within 1 mm of that predicted by the Arrhenius and the 50°C isotherm criteria.

Fig. 5 Model-predicted lesion depth, diameter, and volume versus electrode-penetration depth show consistent results with previous simulations reported in Ref. 67.

different conditions: no electrode cooling, cooled electrode ablation at 50°C, and cooled electrode ablation at 37°C. Both cooled ablations led to higher tissue temperatures at both internal locations than the noncooled ablation. Moreover, the added cooling at 37°C led to lower temperatures than the 50°C cooled ablation at the same internal locations and contributed to a shift of the temperature distribution deeper within the tissue. As shown in Fig. 6, the 50°C isotherm shifted from a ~6 to ~8 mm depth, and led to higher end-ablation tissue temperatures compared to the noncooled ablation.

Similar to the findings reported in Ref. 69, our modeling results suggest that the peak temperature regions lie farther from the electrode during cooled ablation than noncooled ablation, enabling the delivery of higher power without reaching the target electrode temperature, leading to deeper and larger lesions. Moreover, as also shown in Ref. 70, the catheter–tissue interface stays cooler than the deep tissue (Fig. 6).

Figure 7 shows the model-predicted lesion geometry at the end of a 90-s ablation with no electrode cooling (temperature controlled at 90°C), as well as the two cooled ablations at 50°C and 37°C. Lesion diameter, depth, and volume increased with electrode cooling, confirming that electrode cooling enables the formation of better developed lesions, as previously suggested in Refs. 69 and 70.

3.2 Experimental Ex Vivo Evaluation

3.2.1 Tissue temperature distribution

To validate the predicted tissue temperature and lesion geometry against actual measurements, we conducted ablation experiments using the setup and apparatus described in Sec. 2.3. One set of the experiments was conducted at a preset target electrode temperature of 90°C, and another set was conducted at 60°C. Four fiberoptic temperature probes were inserted into each tissue sample in a symmetric pattern with respect to the ablation electrode at a specified radial distance (R) and depth (D): two probes were inserted at 2.5 mm $R \times 3$ mm D with respect to the electrode, denoted as the proximal lesion location; the other two probes at 5 mm $R \times 3$ mm D , denoted as the distal lesion location. Temperature measurements were recorded simultaneously from all probes during the 60-s constant contact (1.5-mm electrode penetration depth) ablation procedures. The experiments were repeated three times on fresh tissue samples.

Tables 1 and 2 summarize the model-predicted and experimentally measured tissue temperatures at the sampled lesion locations at six time-points during the 90°C and 60°C target electrode temperature ablations.

Figure 8 shows the end-ablation temperature distribution in the axisymmetric plane of the modeled lesion, at the proximal

Fig. 6 End-ablation tissue temperature at 1 and 3 mm from electrode tip during a noncooled ablation, as well as two cooled ablations at 50°C and 37°C. Cooling leads to a lesion shift deeper within the tissue, as suggested by the shift of the 50°C isotherm from ~6 to ~8 mm.

Fig. 7 Lesion depth, diameter and volume achieved during a simulated noncooled, 90°C temperature-controlled ablation, as well as two simulated cooled procedures at 50°C and 37°C. The cooling effect leads to larger, better developed lesions, consistent with previous reports.^{69,70} In addition, cooled ablation may benefit from longer ablation times, whereas dry ablation lesions reach their maximum after 60 to 90 s.

Table 1 Model-predicted and experimentally measured tissue temperature (°C) at proximal (2.5 mm $R \times 3.0$ mm D) and distal (5.0 mm $R \times 3.0$ mm D) lesion locations during 60 s temperature-controlled ablation cycles at 90°C. The model-predicted temperature values were recorded at the nearest second to measurement time.

Time stamp (s)	Measured temp. (°C)	Predicted temp. (°C)	Measured temp. (°C)	Predicted temp. (°C)
	Proximal lesion location		Distal lesion location	
10	68.9 ± 4.9	63.8	37.2 ± 1.7	37.1
20	73.0 ± 5.2	70.7	39.0 ± 1.6	37.9
30	76.2 ± 4.8	74.9	39.8 ± 2.3	38.7
40	77.4 ± 5.3	77.2	40.6 ± 2.6	40.3
50	78.7 ± 5.4	78.8	40.9 ± 3.2	41.2
60	79.1 ± 4.9	78.9	41.5 ± 3.4	42.3

Table 2 Model-predicted and experimentally measured tissue temperature (°C) at proximal (2.5 mm $R \times 3.0$ mm D) and distal (5.0 mm $R \times 3.0$ mm D) lesion locations during 60 s temperature-controlled ablation cycles at 60°C. The model-predicted temperature values were recorded at the nearest second to measurement time.

Time stamp (s)	Measured temp. (°C)	Predicted temp. (°C)	Measured temp. (°C)	Predicted temp. (°C)
	Proximal Lesion location		Distal lesion location	
11.2	46.1 ± 2.1	49.5	36.8 ± 1.5	37.0
19.6	50.9 ± 2.6	52.8	37.6 ± 1.6	37.0
30.8	53.1 ± 3.1	54.6	38.4 ± 1.7	37.3
39.2	54.5 ± 4.3	55.0	39.2 ± 1.7	37.5
50.4	56.3 ± 3.5	56.2	39.1 ± 1.7	37.6
58.8	57.2 ± 3.4	56.9	39.3 ± 1.8	38.0

[Figs. 8(a) and 8(c)] and distal [Figs. 8(b) and 8(d)] locations for the 90°C and 60°C ablations, respectively. For the 90°C ablation, the proximal location [Fig. 8(a)] reached end-temperatures above cell-death, whereas the distal location [Fig. 8(b)] only experienced temperatures on the order of ~40°C. Hence, the model-predicted nonviable tissue region extends beyond the proximal location but does not include the distal location, placing the latter in the reversible lesion penumbra as opposed to the irreversible lesion core to which the proximal location belongs.

For the 60°C ablation, neither the proximal nor the distal sampled locations reached the cell-death temperature. Moreover, while the proximal site reached ~40°C, the temperature at the distal site did not rise above the ambient temperature of 37°C. Hence, the image voxels corresponding to the distal isotherm cannot be distinguished from the background (i.e., saline) at 37°C [Fig. 8(d)].

Figure 9 compares the model-predicted and experimentally recorded temperature profiles at both the proximal and distal sampled locations during the ablation cycles at 90°C and 60°C ablation, respectively, showing agreement between the model and direct temperature measurements.

3.3 Lesion Characterization

The model-predicted ablation lesions were characterized according to lesion volume, maximum lesion diameter, surface lesion diameter estimated as the width of the lesion at tissue surface (provided the lesion exposure threshold was reached), and surface exposure diameter, as summarized in Tables 3 and 4. Lesion geometry was defined according to the cumulative exposure tissue injury criterion proposed and validated in Sec. 3.1.2; voxels exposed to temperatures above 55°C for at least 5 s were classified as irreversible lesion and reversible lesion penumbra, otherwise.

For the 90°C target electrode temperature ablation, the model-predicted lesion volume reached ~100 mm³, the achieved maximum lesion diameter was ~6 mm and occurred at 2 mm below the tissue surface, the lesion diameter at the surface was ~5 mm, and the surface exposure diameter was ~9 mm. Similar trends, however of lesser magnitudes, were observed for the 60°C target electrode temperature ablation: the model-predicted lesion volume reached ~27 mm³, the maximum lesion diameter at end-ablation was ~4 mm and occurred at 1.5 mm beneath the tissue surface, the surface exposure

Fig. 8 Model-predicted end-ablation temperature distribution at (a) and (c) proximal (2.5 mm $R \times$ 3.0 mm D) and (b) and (d) distal (5.0 mm $R \times$ 3.0 mm D) sampled locations, along with voxels on the same isotherm (shown by the outlined voxels) for the (a) and (b) 90°C and (c) and (d) 60°C ablations. Note that the distal location did not rise above the saline bath ambient temperature during the 60°C ablation, as reported in Table 2, and hence its intensity is similar to that of the background.

Fig. 9 Temperature plots of the electrode model, as well as model-predicted and experimentally measured (mean $\pm$ standard deviation) temperatures at the proximal and distal locations for the 90°C and 60°C target electrode temperature ablations.

Table 3 Characterization of model-predicted tissue damage achieved at 90°C target electrode temperature according to lesion volume, maximum lesion diameter, surface lesion diameter, and surface exposure diameter.

Time stamp (s)	Lesion volume (mm ³)	Maximum lesion dia. (mm)	Surface lesion dia. (mm)	Surface exposure dia. (mm)
5	30.2	3.8	4.0	7.6
10	44.1	4.2	4.5	8.3
20	63.1	4.8	4.8	8.6
30	77.5	5.2	4.9	9.0
40	84.5	5.6	4.9	9.1
50	91.5	5.7	5.0	9.2
60	96.0	5.8	5.0	9.4
Experimental lesion (mm)	Final lesion dia.		Final surface exposure dia.	
	5.5 $\pm$ 1.3		9.5 $\pm$ 2.3	

Table 4 Characterization of model-predicted tissue damage achieved at 60°C target electrode temperature according to lesion volume, maximum lesion diameter, surface lesion diameter, and surface exposure diameter.

Time stamp (s)	Lesion volume (mm ³)	Maximum lesion dia. (mm)	Surface lesion dia. (mm)	Surface exposure dia. (mm)
5	6.4	3.0	—	6.7
10	14.4	3.4	—	7.0
20	21.9	3.8	—	7.3
30	23.0	3.9	—	7.6
40	25.1	3.9	—	8.0
50	25.6	4.0	—	8.3
60	26.2	4.0	—	8.8
Experimental lesion (mm)	Final lesion dia.		Final surface exposure dia.	
	3.8 $\pm$ 1.5		8.3 $\pm$ 2.9	

diameter was on the order of 8 mm, but no lesion was detected at the surface, due to the limited amount of RF energy delivered given the lower target-electrode temperature, combined with the effect of convective surface cooling.

Following ablation, the samples were removed from the saline bath and analyzed according to the diameter and depth of the region marked by tissue discoloration, suggested to correspond to the region enclosed by the 50°C isothermal surface.⁴²

The lesion characterization measurements predicted by the model were consistent with those observed in the digital images of the tissue samples (surface and section views) acquired post-ablation (Figs. 10 and 11).

The model-predicted maximum lesion diameter was ~6 mm for the 90°C ablation and 4 mm for the 60°C ablation, respectively, while the experimentally observed maximum lesion diameters were 5.5 ± 1.3 and 3.8 ± 1.5 mm for the 90°C and

Fig. 10 Cross sectional rendering of the model-predicted tissue damage at 5, 10, and 60 s during 90°C ablation. The bright discolored regions are suggestive of irreversible damage, surrounded by lesion penumbra. Postablation images of a typical sample: a surface exposure diameter of ~9 mm and a maximum lesion diameter of ~6 mm, consistent with the model-predicted dimensions.

Fig. 11 Cross sectional rendering of the model-predicted tissue damage at 5, 10, and 60 s during 60°C ablation and postablation images of a typical sample: a surface exposure diameter of ~8 mm and a maximum lesion diameter of ~4 mm, consistent with the model-predicted dimensions.

60°C ablations, respectively. Similarly, the model-predicted surface exposure diameter was ~9 mm for the 90°C ablation and 8 mm for the 60°C ablation, comparable to postablation measurements of 9.5 ± 2.3 mm for the 90°C ablation and 8.3 ± 2.9 mm for the 60°C ablation, as summarized in Tables 3 and 4.

Figures 10 and 11 also show axis-symmetric renderings of the model-generated lesion (irreversible core region surrounded by lesion penumbra) at 5, 10, and 60 s.

4 Discussion

We have described the implementation, numerical verification, and *ex vivo* validation of a 3-D image-based ablation model designed to provide monitoring and visualization of induced tissue injury and lesion progression during RF cardiac ablation. The model uses the voxels of traditional medical images as input, relies on the physiological parameters of tissue and blood, and characterizes the extent of tissue injury according to the exposure temperature and time. The methods are accompanied by both numerical verification against previously developed and disseminated ablation models, and also by validation experiments conducted in animal myocardial tissue samples in a convective saline bath at body temperature using direct temperature sensing probes for real-time temperature measurements and postablation assessment of the lesion. This work has demonstrated that the proposed approach provides a sufficiently accurate surrogate model that enables fast, real-time estimates of tissue temperature distribution, tissue damage, and lesion characterization.

The formulation proposed here is designed for intraoperative implementation and integration with an image guidance and navigation platform for cardiac ablation therapy. While this work does not provide a clinical evaluation of a proposed system, we believe it demonstrates the potential of developing a method to quantify, characterize, and visualize ablation lesions via nondirect sensing approaches.

We explored the effects of temperature-dependent tissue conductivity, electrode penetration depth, electrode cooling, and compared the proposed, computationally efficient lesion irreversibility criterion based on cumulative exposure to two other tissue injury criteria previously validated in the literature. The experimental *ex vivo* studies also demonstrated agreement within 5°C between the model-predicted and experimentally measured end-ablation temperatures, which is imperative to ensure sufficient exposure to achieve irreversible tissue damage. To account for uncertainties in the positioning of the fiberoptic probes at the desired locations, the measured temperature was assessed against model-predicted measurements over a 1.0×1.0 mm (with 0.5-mm computational spatial accuracy) region. The experimentally recorded temperature matched the model-predicted temperature within a 1.0 mm^2 region. Lastly, the model-predicted lesion dimensions were also in agreement with those observed postablation within 0.5 to 1 mm. While the model-predicted results were fully reproducible, the experimental postablation lesion dimensions were measured by visual inspection and were associated with higher uncertainty when identifying the lesion border.

We have identified several potential limitations of the current work in the context of its long-term intended application to the intraoperative guidance of cardiac ablation therapy. While an *in vivo*, beating heart assessment is preferred, it would entail a very invasive process of implanting fiberoptic temperature probes inside the *in vivo* animal heart tissue for direct online monitoring

of tissue temperature, acquiring the necessary preoperative images, conducting the ablation, and ultimately, sacrificing the animal for postprocedure evaluation. To avoid these challenges, other groups have conducted studies using bovine *ex vivo*³⁹ and porcine *in situ*⁷¹ skeletal and myocardium muscle samples, as well as in *in vivo* canine thigh muscle.⁷² Since the objective of this study was to evaluate the proposed image-based surrogate ablation model and validate the predicted tissue temperature and lesion development, we conducted the experiments in a well-controlled environment using bovine tissue samples and literature-suggested physiological parameters. Nevertheless, we plan to conduct *in vivo* investigations as part of our future evaluation studies.

Another possible limitation is cardiac motion and its effect on the electrode–tissue contact. As suggested in Refs. 64 and 65, contact contributes to the size of the created RF lesion, and sub-optimal contact may lead to inadequately developed lesions and gaps in the ablation line, requiring revision.⁶⁶ We also acknowledge that electrode–tissue contact is a critical factor in ablation. The entire premise of the VisiTag approach is that higher contact (force) leads to better developed, transmural, irreversible lesions. In this work, instead of treating it as an independent factor, we are considering it in the context of lesion quality assessment, as contact (area) directly impacts heat flux into the tissue and, in turn, it impacts the transmural and irreversibility of the lesion. Contact force affects lesion development by directly affecting the resistive (Joule) heating at the electrode–tissue interface: better contact increases the energy flux into the tissue. While resistive heating is only responsible for the initial heat transfer into the electrode–tissue interface,¹ the much larger contributor to a fully developed lesion is the heat diffusion into the tissue via conduction. These principles were demonstrated in the numerical experiments reported in their work, which showed that lesion volume, depth, and width increased with electrode penetration depth, hence leading to better developed lesions.

While force may serve as an acceptable surrogate for contact, it does not provide an objective measure of the extent of the contact. For example, same applied force deflects stiffer tissue less than softer tissue, yielding a smaller contact area. Contact area, on the other hand, is a more direct measure, as it directly impacts heat flux into the tissue and lesion size. We have conducted several experiments aimed at establishing the correlation among electrode penetration depth, contact area, contact force, and approach angle.⁷³ We used polyvinyl alcohol cryogel (PVA-C) tissue mimicking phantoms that exhibited different stiffness⁷⁴ to control the electrode–tissue contact by varying the applied contact force and recorded the electrode depression and contact area. We employed real-time US imaging to quantify the interaction between the ablation electrode and tissue to accurately estimate contact, to help us better understand the effect of catheter pose and position relative to the tissue. We simultaneously recorded the tracked position of the catheter tip, the contact force reading, and US images of the electrode–tissue interface at different catheter orientations. The study showed that, with increased material stiffness, equivalent contact force readings led to shallower electrode penetration as recorded by the tracked catheter and a smaller contact area, both depicted from the US images, as well as quantified based on the electrode radius and penetration depth, implying varying lesion quality. Figure 12 shows the correlation among the electrode depression (measured using the tracked catheter), contact area (estimated from US images and quantified based on the electrode depression and

Fig. 12 Plots showing the direct correlation between the estimated contact area (estimated based on electrode radius and measured electrode penetration depth) versus contact force measured at 90°C and 60°C catheter orientation using three PVA-C gels of different stiffness.⁷³

electrode tip radius), and contact force (recorded by the build-in force transducer).

We will conduct additional experimental studies that take into account electrode–tissue contact by varying the electrode depression into the tissue and experimentally replicate the current numerical results. The contact area inferred from the force–displacement–contact area data will serve as a contact surrogate. We will leverage this information in a future study to incorporate the effect of contact on the modeled ablation lesion and validate the method accordingly. Moreover, as an alternative to relying on recently introduced force-feedback catheters,⁷⁵ real-time US imaging^{76–78} may also be employed to estimate electrode–tissue contact.

With respect to the efficacy of “point by point” and “drag” methods for ablation, the 2017 HRS/EHRA/ECAS/APHSR/SOLAECE Guidance Documents Expert Consensus Statement on Catheter and Surgical Ablation of Atrial Fibrillation⁷⁹ recognizes that there are “several relevant variables that impact lesion size including catheter stability, contact force, power output, temperature, and duration.” At this time, there is no specific guidance suggesting one ablation approach (point-by-point or drag) versus the other: while Yokokawa et al.⁸⁰ indicated better outcomes for dragging, a larger study was reported by Nagahori et al.,⁸¹ which showed the same outcome for both approaches. The long-term goal for this work is to effectively incorporate electrode–contact information into the online ablation lesion modeling and visualization techniques regardless of the ablation approach used.

Another limitation of this study may be its applicability to modeling the ablation of thin tissue, such as the left atrial wall. The described model and validation experiments were conducted on 10-mm myocardial tissue samples, but tissue thickness is a relevant parameter. The recent publication by Chikata et al.⁸² also confirms that tissue thickness is important. Our model can account for tissue thickness (and tissue composition) parameters in a manner similar to Ref. 82 by using a CT or MRI scan to segment out the tissue, including its thickness. On the other hand, the LA wall is fairly thin, making it nearly impossible to place thermocouples into an *ex vivo* sample of the LA wall without completely destroying the tissue. Instead, we chose to measure the temperature using a larger piece of tissue, but at different depths, one of which was at 2.5 mm below tissue surface, similar to the LA wall thickness. In its current form, the model is much more representative of a ventricular ablation.

However, we argue that the modeled temperature measurements are sufficiently accurate, as shown in this paper, and provided the model could be enhanced to incorporate tissue thickness as a modeling parameter, which would be the focus of future work, this technique could help visualize and assess lesion progression for both ventricular and atrial ablation applications.

In its current form, the model is implemented using a 0.5-mm isotropic voxel spacing and 1-s temporal resolution. Other groups have investigated the use of finer spatial resolutions via traditional finite element meshes, as well as finer computational time steps. We have also explored the use of oversampled images with higher spatial resolution (0.5, 0.25, and 0.1 mm isotropic voxels) and finer computational time steps of up to 0.1 s. These computational parameters led to less than 3°C differences in the estimated tissue temperature distribution and less than 0.5 mm difference in the estimated lesion diameter as the 0.5 mm and 1 s simulations; however, at the expense of longer computational times, on the order of several minutes for a 60- to 90-s ablation cycle. Moreover, given the typical resolution of clinical-quality images and provided the position and the accuracy of electromagnetic tracking systems ranging from 1⁸³ to 2.5 mm,⁸⁴ a 0.5-mm voxel size was identified as most appropriate for this application. Furthermore, in light of the intended use for intraoperative lesion monitoring, the 0.5-mm isotropic voxel spacing and 1-s temporal resolution provides the optimal trade-off between accuracy and computing efficiency.

The intended clinical application requires that the model provides lesion visualization and monitoring with minimal latency, in the real-time of the procedure. The computational speed of the image-based implementation presents a significant advantage over computationally prohibitive finite element approaches. The proposed model was implemented on a standard desktop PC (Intel Core2 Quad 2.5 GHz processor with a 8 GB DDR2 RAM) and yielded updates of the tissue temperature distribution and rendered a model-predicted ablation lesion during a 60-s ablation cycle in ~1 min. The computational efficiency can be further improved via parallel multithread computing and GPU implementation, enabling lesion monitoring with no workflow latency.

This lesion characterization and visualization tool could be leveraged clinically by integrating it into existing platforms, such as VisiTag. As such, this could be an upgraded version of what VisiTag currently offers. Once integrated with an

image guidance platform for left atrial ablation therapy, the proposed model has the potential to provide the cardiologist with online characterization, visualization, and monitoring of the delivered RF lesions. In concert with the catheter position provided by the tracking system, the model-predicted lesions will be displayed as superimposed on the electroanatomical model used for guidance. Clinicians will have access to dynamically updated lesion maps during therapy that indicate the location and size of the lesions that need to be delivered to avoid the presence of gaps in the ablation pattern.

5 Summary and Future Directions

This work describes the implementation, numerical verification, and *ex vivo* experimental validation of a fast and accurate method to model, predict, characterize, and visualize ablation lesions. These studies demonstrated less than 5°C difference between the model-predicted and experimentally measured end-ablation temperatures. The predicted and observed lesion patterns were also in agreement, suggesting sufficiently accurate modeling of the ablation lesions. Lastly, our proposed method enables therapy delivery feedback with minimal latency.

Given the support provided by new methodologies of planning, simulation, and training, this work demonstrates an effective and fast extension of a theoretical model into a practical tool. Following further development and validation, including *in vivo* evaluation in animal models and integration into an existing EAM platform, this technology has the potential to allow for quantitative monitoring of ablation therapy with integrated guidance.^{50,85}

Future efforts will focus on extending the model to include tissue thickness as a modeling parameter, estimating electrode-tissue contact, and incorporating it into the model formulation as a means to modulate the heat flux at the electrode-tissue interface. We will also evaluate the model-predicted lesions against postprocedural lesion assessment as characterized via tetrazolium chloride tissue staining and/or DCE-MRI. Knowledge of the true ablation sites and their characterization as necrotic or endemic via DCE-MRI²⁴ is of significant importance for the *in vivo* validation of the thermal model. Lastly, we will explore several approaches⁸⁶ for optimal visualization and display of the model-predicted ablation lesions, including volumetric overlays, multiparameter contours showing the representative isotherms, and 3-D visualizations using combined volume rendering of the lesion and substrate tissue.

Disclosures

No conflicts of interest, financial or otherwise, are declared by the authors.

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Biographies for the authors are not available.