A PATIENT-SPECIFIC COMPUTATIONAL STUDY OF TRANSVENOUS DEFIBRILLATION

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\textbf{Abstract}- The goal of this study is to assess the predictive capacity of computational models of transvenous defibrillation by comparing the results of patient-specific simulations to clinically determined defibrillation metrics. Solutions for seven patient-specific models have been completed. The 3-D models of the thorax and in situ electrodes were created from segmented CT images taken shortly after implant. Each of the 3-D models was created by defining each voxel in the segmented data set as a volume element in the computational model. The electric field distribution during defibrillation was computed using the finite volume method. The critical mass hypothesis was used to define a successful shock and to determine the defibrillation metrics from the calculated field distribution. Simulated defibrillation thresholds yielded good estimates of the clinically determined thresholds in 4 of the 7 patients examined. The model-predicted impedances correlate well with the clinical measurements. These results are promising and provide preliminary support to the potential utility of this modeling approach for patient-specific surgical planning of cardioverter defibrillator implantation and for evaluating new electrode configurations.

\textbf{Keywords} - implantable defibrillator, defibrillation modeling, finite volume method, patient-specific, critical mass

I. INTRODUCTION

Ventricular fibrillation (VF) is a severe heart arrhythmia that can lead to sudden cardiac death if not treated promptly. The only effective clinical intervention to cease VF is electrical defibrillation, which consists of passing a large electric current through the heart. The Implantable Cardioverter Defibrillator (ICD) is an electronic device implanted in the left pectoral region. The ICD detects VF and delivers electric shocks via intracardiac leads to reset the heart to the normal sinus rhythm. The defibrillation threshold (DFT) energy, defined as the lowest shock energy that achieves defibrillation, varies from patient to patient and is not known prior to ICD implantation. Clinical DFT testing follows standard protocols, which require repetitive inductions and extinctions of VF during implant procedure. These protocols impose an immense strain on the patient’s heart which might be reduced if an accurate estimate of the individual DFT is available prior to implant. Previous computer modeling studies \cite{1}, based on a single average model of a human torso, have shown a good correlation with the overall mean of reported clinical defibrillation metrics (DFT energy, DFT current \( I_{th} \), DFT voltage \( V_{th} \) and impedance \( Z \)). These findings suggest that patient-specific computer models might be able to predict the defibrillation energy requirements for individual patients. Thus, the goal of this study was to assess the predictive capacity of patient-specific computational models of transvenous defibrillation, by comparing simulated and clinical defibrillation metrics.

II. METHODS

A. Clinical DFT Testing

Seven patients were recruited for ICD implantation (in accordance with the guidelines established by the Human Research Committee at the Brigham and Women’s Hospital). Clinical DFT testing was performed using a step-down procedure. VF was induced by applying a pulse of alternating current and the defibrillation shock (biphasic waveform) was delivered ten seconds later. The trials started at 20J and decremented until VF was no longer terminated. The lowest energy that defibrillated was the DFT energy.

B. Image-Based Model Construction

All patients were imaged on a spiral CT scanning system post-implant, with the catheter electrodes in place. Each of the patient-specific numerical models was constructed directly from the segmented CT images, using a structured meshing algorithm. Each voxel in the segmented image data set was defined as a volume element in the computational model.

C. Computational Approach

In the quasistatic approximation, the electric potential \( \Phi \) is governed by

\[
\nabla \cdot (\sigma \nabla \Phi) = 0
\]

subject to boundary conditions: i) constant potential on the electrodes and pulse generator can (Dirichlet); ii) no current flux on the thorax surface (Neumann). Electrical conductivities were assigned to six tissue regions as follows: \( \sigma_{\text{myocardium}}=2.5 \text{mS/cm}, \quad \sigma_{\text{muscle}}=2.5 \text{mS/cm}, \quad \sigma_{\text{blood}}=8 \text{mS/cm}, \quad \sigma_{\text{lung}}=0.7 \text{mS/cm}, \quad \sigma_{\text{fat}}=0.5 \text{mS/cm}, \quad \sigma_{\text{bone}}=0.1 \text{mS/cm} \). Equation (1) was solved numerically by the finite volume method using I-DEAS software (Structural Dynamics Research Corporation, Milford, OH, USA).

D. Solution Interpretation

For each patient-specific simulation, the critical mass hypothesis was used to define successful defibrillation with minimum delivered energy: a successful shock must expose fields equal to or greater than the inexcitability threshold \( E_{th} \). According to Ideker \cite{2} we used a critical mass of 95\% and an \( E_{th}=3.5 \text{V/cm} \) (biphasic pulse).

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III. RESULTS

The patient-specific clinical and model-predicted defibrillation metrics are compared in Fig. 1a-d. The patient’s labels are shown on the horizontal axis. Current pathways during a simulated defibrillation shock are shown in Fig. 2 (patient SM).

![Fig. 1. Simulated and clinical defibrillation metrics: a) impedance; b) DFT energy; c) DFT voltage, Vth; d) DFT current, Ith.](image)

IV. DISCUSSION

The model-predicted impedances correlate well with the clinical measurements. Simulated defibrillation thresholds yielded good estimates in four of the seven patients examined. It is significant to note that the respective clinical DFTs for these patients spanned an approximately 2-fold range, suggesting that the goodness of the achieved match was not due to the similarity of the patients examined but in fact are reflections of their differences. The patients whose clinical metrics were not well matched by the model-predicted values exhibited clinical anomalies (e.g. patient EV has large infarct regions). Our modeling approach can only capture geometry-based differences in DFTs reflected in the patient population. Hence, patients with altered cellular electrophysiology may not be well modeled by the defibrillation assumptions applied in extracting the defibrillation parameters, i.e. $E_{th}$ and/or critical mass may differ. These drawbacks might also partially explain the failure to predict DFTs for patients SM, EV and MA.

V. CONCLUSION

This paper presents comparisons between simulated and clinical defibrillation metrics determined for individual patients. The correspondence between the predicted and measured DFTs observed in four of the seven patients is encouraging and provides preliminary support to the potential utility of the modeling approach for presurgical planning and for evaluation of new electrode configurations.

REFERENCES
