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A Patient-Specific Model of Virtual Ablation for Atrial Fibrillation

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Abstract— **The purpose of this study was to propose a patient-specific model of atrial fibrillation (AF) and apply it to virtual radiofrequency ablation (RFA). We obtained patient-specific geometries of the left atrium (LA) from CT data and constructed three-dimensional (3D) simulation models. A bidomain Courtemanche model was used to simulate the 3D electric waves on the LA surface, and an S1–S2 protocol was applied to induce AF in the model. To identify scar areas in the models, we converted clinically measured voltage data on the LA surface to the scar maps of the simulation model. Then, after initiation of AF, we applied the virtual ablation scheme to the model and investigated whether the AF was terminated by the scheme. The computed results of AF and ablation were similar to those of clinical observation, providing a clinically important simulation method for preclinical virtual trials of AF treatment.**

I. Introduction

Atrial fibrillation (AF) is defined as a fast, irregular atrial rhythm [1]. It causes rapid atrial contraction that leads to blood stagnation [2]. AF increases the risk of stroke four to five fold across all age groups, accounting for 10% to 15% of all ischemic strokes and nearly 25% of strokes in people older than 80 years [3]. Thus, various studies on the underlying mechanism of AF have been performed [4]. Along with experimental and clinical studies, computer simulation methods have become popular in the analysis of AF [5,6]. Despite substantial progress in the simulation of AF, only a few studies on the clinical application of virtual atrial ablation have been conducted.

In this study, we developed a patient-specific model of AF based on CT data of patients. For this purpose, the atrial geometry of the patient was constructed from CT data, and an electrophysiological protocol for the geometry was applied to generate electric wave conduction in tissue. We used an S1–S2 protocol to induce AF and applied virtual ablation to the AF model. With this virtual ablation technique, we found optimal ablation sites and checked that the treatment could terminate the chaotic AF phenomenon. Computed results were compared with clinical data. Moreover, we developed a graphical user interface (GUI) including a semi-automatic virtual ablation procedure for patients.

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II. Geometry and Method

A. Geometry

A patient's left atrial geometry constructed from CT data is shown in Fig. 1(a). This was used to create a three-dimensional (3D) model for a finite-element method (FEM) (Fig. 1(b)). We only considered the left atrium (LA), although electric wave propagation during AF in the right atrium (RA) is not trivial. However, because the effective refractory period of LA is shorter than that of RA, most of the AF drivers are present within the LA. Hence, electric waves in the LA are critical from a clinal perspective. A total of 1,000,000 nodes and 1,000,000 finite elements were generated in the LA model.

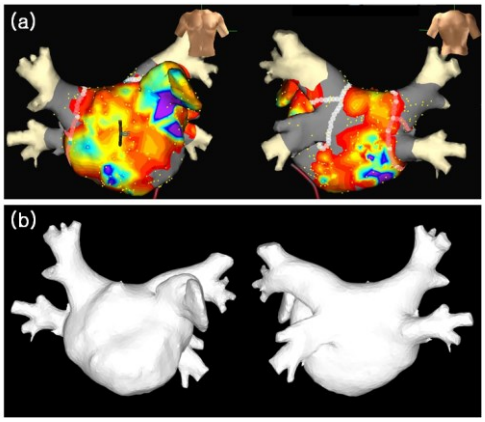

Figure 1. The original surface geometry of the left atrium. *(a) Clinical image with scar areas, (b) computational model.*

B. Human atrial cell model

The computational approach to cellular components is based on the electrophysiological model of the human atrial myocyte proposed by Courtemanche et al. [7] (CRN model). This model was implemented to simulate atrial electrophysiology and uses well-established experimental data on human atrial cells and calcium dynamics.

C. Governing equation for electric wave propagation in 3-dimensional atrium

To simulate electrical wave propagation in LA tissue, we used an FEM. Atrial tissue in the model was spatially divided into two regions: intracellular and interstitial spaces. Each area

acts as a volume conductor that has its own volume-averaged potential fields, current, and conductivity tensor. The ionic currents pass from one area to another through the cell membrane. We assumed spatial variation in the electric potential inside the intracellular and extracellular spaces and therefore used a bidomain method [8,9] for the tissue model. The governing equation of the 3D cardiac tissue model can thus be described by the following partial differential equations in reaction–diffusion form:

$$\nabla\cdot\left(\left(\sigma_i+\sigma_e\right)\nabla\phi_e\right)=-\nabla\cdot\left(\sigma_i\nabla V_m\right)+I_{s1}$$

$$\nabla\cdot\left(\sigma_i\nabla V_m\right)+\nabla\cdot\left(\sigma_i\nabla\phi_e\right)=A_m\left(C_m\frac{\partial V_m}{\partial t}+I_{ion}\right)-I_{s2}$$

where V_m is the transmembrane voltage; ϕ_e is the extracellular voltage; t is time; A_m is the cellular cross-sectional area of the atrial cell; C_m is the membrane capacitance of the ventricular cell; σ_i and σ_e are the diffusion coefficients of electric wave propagation in the intracellular and extracellular directions, respectively; I_{s1} and I_{s2} are the externally applied stimulus currents to the intra- and extracellular spaces; and I_{ion} is the sum of all transmembrane ionic currents. A forward Euler method was used for the temporal discretization of the bidomain equation, and an FEM was applied for spatial discretization.

III. Result

We plotted the shape of the action potential of the modified CRN model. The action potential showed the characteristic sharp spike shape of an atrial cell with a resting potential of -81 mV (Fig. 2). To shorten the action potential duration (APD) in the case of fibrillation, we increased the conductance of the fast inward sodium current by 50% and decreased the conductance of the L-type calcium current by 50%. The APD in the modified mode was about 225 ms, whereas the conduction velocity in atrial tissue was about 0.52 m/s.

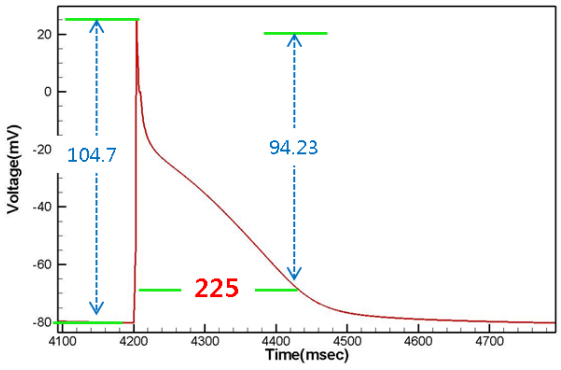

Figure 2. Transient shape of action potential of atrial cell

We generated AF for the 3D geometry of the patient's LA. To identify scar areas in the models, we converted clinically measured voltage data on the LA surface to the scar maps of the simulation model. An S1-S2 protocol [12] was used to create re-entry waves on the LA, and instability in the rotor of the re-entry wave then occurred, inducing wave break-up phenomena. Fifty patients were tested using this protocol. Figure 3 shows the chaotic wave pattern of the first patient's LA generated by the protocol. The same phenomena were also observed for the second patient (Fig. 4). To assess the feasibility of the virtual ablation technique, we virtually ablated some sites on the LA surface and then determined whether the ablation at these sites could permanently terminate AF.

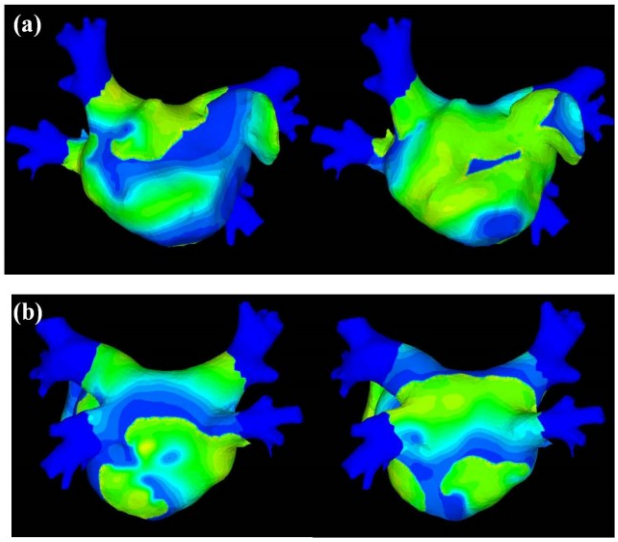

Figure 3. Simulated AF voltage map of a patient 1 *(a) anterior, (b) posterior.*

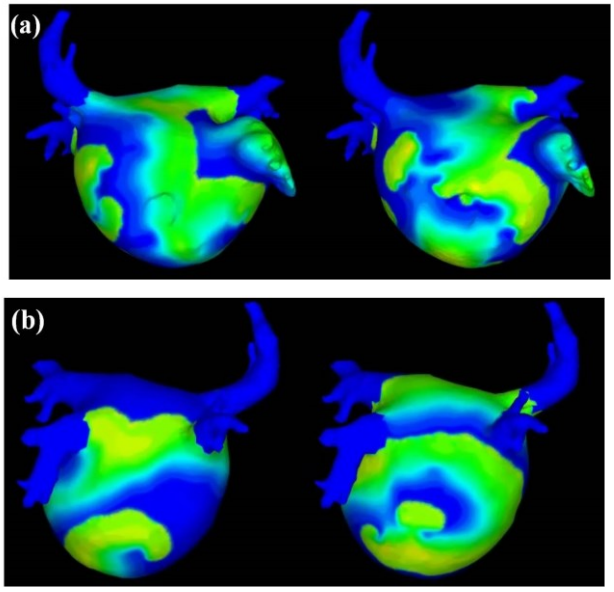

Figure 4. Simulated AF voltage map of patient 2 *(a) anterior, (b) posterior.*

Figure 5(a) shows the clinical data for the potential map, where the white dotted lines represent ablation sites. Figure 5(b) shows the ablation sites in the computational model. Electric wave patterns after virtual ablation are shown in Figure 6. Here, the chaotic waves eventually disappeared. We applied this ablation protocol to all patients in the computer simulation model and verified that the ablation sites were

effective in terminating AF. This is also confirmed in the action potential graph shown in Figure 7. Virtual ablation was applied at 20,000 ms, and the membrane potential was then maintained at the resting potential (-81 mV).

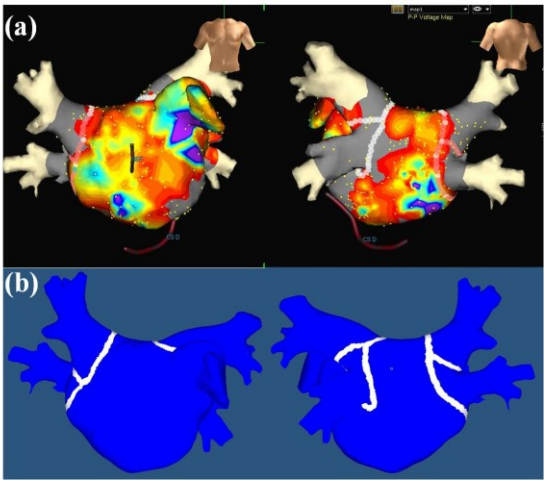

Figure 5. Virtual ablation zone. *(a) clinical image with scar regions, (b) computational model.*

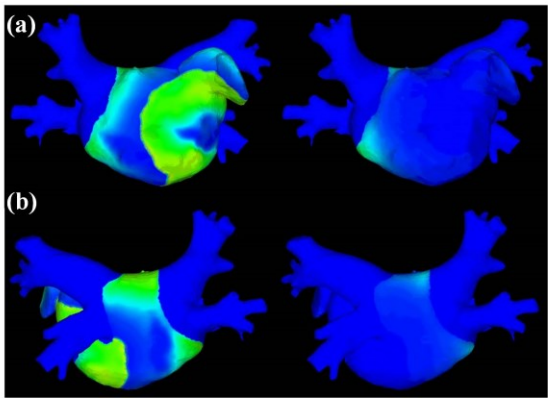

Figure 6. Simulated AF voltage map after virtual ablation. *(a) anterior, (b) posterior.*

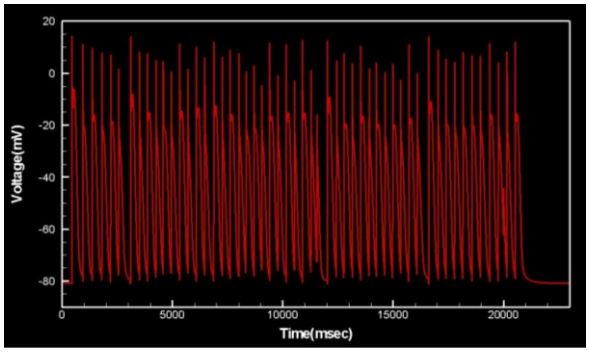

Figure 7. Action potential patterns in AF

The GUI for virtual ablation is shown in Figure 8. All procedures required to create a patient-specific model of AF and ablation are manageable within the program.

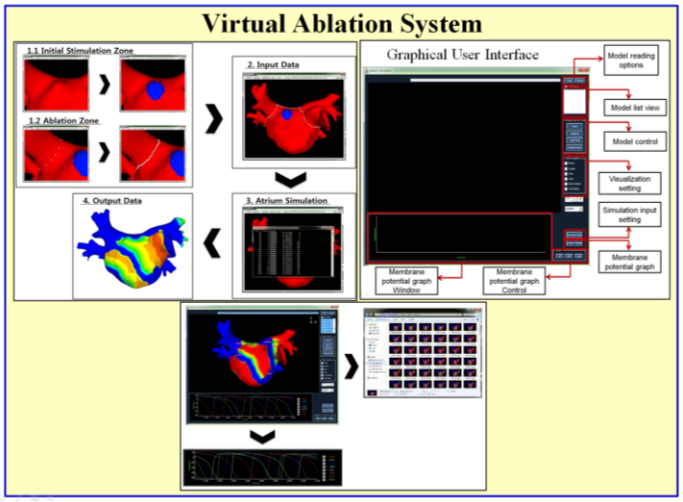

Figure 8. Graphical user interface of the virtual ablation system

IV. Discussion

In this study, we delineated the effect of virtual ablation on AF wave dynamics using a computational model comprising an electrophysiological model of 3D atrial tissue based on an existing cellular model and a bidomain method implemented in a 3D FEM. Using the CRN model, the typical shape of the cellular action potential was accurately reproduced, showing the spike-dome pattern of a human atrial cell. Additionally, the calcium dynamics and major ionic current profiles matched the original results of Courtemanche et al. [7]. The re-entrant wave propagation initially resulted in a regular re-entrant wave in the LV tissue, but it showed spiral breakup, and AF developed. To test the effect of ablation on atrial re-entrant wave dynamics in the LA, we applied ablation treatment to a specific tissue part. Ablation of LA tissue resulted in a partial conduction block, and the AF was terminated to a regular sinus rhythm with a resting potential of -81 mV.

Atrial ablation is a well-recognized therapy that is capable of transforming a chaotic re-entrant wave into a regular sinus rhythm. Although this has been frequently performed clinically, no theoretical study of virtual ablation has been carried out. The main contribution of the present paper to this field is that it provides a method of assessing the effect of ablation on AF.

Although the present study had meaningful results, several limitations still exist. First, we did not investigate the complex structure of fibrosis involved in the diseased LA tissue. We elucidated the virtual ablation effect only on the geometrical aspect of the patient-specific model. Also, in the computational model, the LA tissue did not have a complex muscle layer with fiber anisotropy and heterogeneous muscle thickness. However, these limitations were not expected to greatly alter the main contributions of this study.

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