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Feasibility of using patient-specific models and the “minimum cut” algorithm to predict optimal ablation targets for left atrial flutter

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Abstract

BACKGROUND—Left atrial flutter (LAFL) occurs in patients after atrial fibrillation ablation. Identification of optimal ablation targets to terminate LAFL remains challenging.

OBJECTIVE—The purpose of this study was to use patient-specific models to simulate LAFL and predict optimal ablation targets using a novel approach based on flow network theory.

METHODS—Late gadolinium-enhanced cardiac magnetic resonance scans from 10 patients with LAFL were used to construct atrial models incorporating fibrosis by investigators blinded to procedural findings. Rapid pacing was applied in silico to induce LAFL. In each LAFL, we represented reentrant wave propagation as an electric flow network and identified the “minimum cut” (MC), which was the smallest amount of tissue that separated the flow into 2 discontinuous components. In silico ablation was applied at MCs, and targets were compared to those that terminated LAFL during catheter ablation.

RESULTS—Patient-specific atrial models were successfully generated from patient scans. LAFL was induced in 7 of 10 models. Ablation of MCs terminated LAFL in 4 models and produced new, slower LAFL morphologies in the other 3. For the latter cases, flow analysis was repeated to identify MCs of emergent LAFLs. Ablation of these MCs terminated emergent LAFLs. The MC-based ablation lesions in simulations were similar in length and location to ablation targets that terminated LAFL during catheter ablation for these 7 patients.

CONCLUSION—Personalized atrial simulations can predict ablation targets for LAFL. These simulations provide a powerful tool for planning ablation procedures and may reduce procedural times and complications.

Keywords

Patient-specific atrial model; Left atrial flutter; Ablation target; Network theory; Fibrosis

Introduction

Left atrial flutter (LAFL) is a common form of arrhythmia recurrence in patients after atrial fibrillation (AF) ablation, occurring in up to 31% of AF ablation cases.^{1,2} Sustained by a reentrant circuit in the left atrium, LAFL has an AF equivalent risk of thromboembolic events, is difficult to manage with antiarrhythmic drugs or rate control therapy, and recurs frequently after cardioversion.^{2,3} The preferred strategy for treatment of this arrhythmia uses catheter ablation to render the critical tissue responsible for sustaining the reentry nonconductive.⁴

Traditional strategies for determining LAFL ablation targets include entrainment and activation mapping during invasive clinical electrophysiologic study (EPS).⁴ Entrainment mapping uses programmed electrical stimulation to locate the slow conducting zone in the reentrant circuit that perpetuates LAFL.⁵ This approach is tedious and can be technically challenging when there are multiple circuits that switch with entrainment maneuvers.⁶ Activation mapping plots the time sequence of reentrant wave propagation during LAFL, but this method is encumbered by a time-consuming, nonsystematic, point-by-point procedure that requires hundreds of distinct acquisition points.⁷ The limitations of these treatment strategies thereby result in long procedural times, which may increase complication rates and fluoroscopy exposure,⁶ underscoring the urgent need to develop novel strategies for identification of the optimal LAFL ablation targets.

The purpose of this study was to explore the feasibility of using noninvasive, patient-specific atrial models to predict the optimal LAFL ablation targets.

Methods

Construction of patient-specific atrial models

Detailed methods of patient-specific model construction are provided in the Online Supplementary Material. In brief, late gadolinium-enhanced cardiac magnetic resonance (LGE-CMR) scans were acquired from 10 patients with LAFL presenting for catheter ablation (Figure 1I, A). After fibrotic regions were segmented using an image intensity ratio algorithm⁸ (Figure 1I, B), finite-element meshes of the atria were constructed (Figure 1I, C).⁹ Atrial fiber orientations were assigned (Figure 1I, D) using a validated approach,¹⁰ as previously used in the atria.⁹ Membrane kinetics in non-fibrotic regions was represented by the Courtemanche-Ramirez-Nattel model of the human atrial action potential (Figure 1I, E, gray), modified to fit intracardiac electro-physiologic data.^{11,12} Membrane kinetics in fibrotic regions were represented by the aforementioned modified Courtemanche-Ramirez-

Nattel model with additional changes to reflect electrophysiologic consequences of fibrotic remodeling, namely, incorporating reduced inward rectifier potassium current (50%), L-type calcium current (50%), and sodium current (40%) (Figure 1I, E, green).¹³ Tissue conductivities were chosen such that conduction velocity was within the range recorded during atrial flutter.¹⁴

Simulation protocol

Electrical wave propagation was governed by the monodomain formulation. Finite-element simulations were executed with the CARP software package (Johns Hopkins University, Université de Bordeaux, Medizinische Universität Graz).¹⁵ In each patient-specific model, 30 pacing sites were distributed uniformly throughout the atria using a point repulsion algorithm. A decremental pacing protocol consisting of 14 pulses with cycle lengths decreasing from 300 to 150 ms in 25-ms intervals was delivered at each site to induce LAFL. LAFL was categorized as inducible in a given atrial model if self-sustained reentrant activity persisted throughout the duration of the simulation after the last pacing stimulus was delivered.

Identification and ablation of minimum cuts in reentrant wave propagation

We developed a novel methodology to automatically identify, based on the simulated reentrant circuits, the optimal ablation target for each LAFL morphology. We represented wave propagation during 1 cycle of reentry as a flow network. A flow network is a mathematical graph that models directional movement between interconnected objects. Interconnected objects are represented as vertices in a graph; links between adjacent pairs of vertices, where flow can pass through, are called edges; and the maximum possible flow between two adjacent vertices is termed edge capacity. The minimum cut (MC) in a flow network represents the number of edges that, when removed, separate the network into 2 disconnected components (Figure 1 II).

In this study, each vertex in the flow network corresponded to an element in the patient-derived mesh. Two vertices in the network were defined to be adjacent to each other if they (1) corresponded to elements that shared a common face and (2) if the difference in activation time between these elements was <20 ms. An edge in the flow network corresponded to the shared face between adjacent vertices. The capacity of a network edge was defined to be equal to the cross-sectional area of the corresponding face. The MC was the minimum set of faces between finite elements in the mesh that partitioned the flow network into 2 disconnected components and was determined using the Boykov-Kolmogorov algorithm.¹⁶ The MCs are the *in silico* equivalent of the minimum set of ablation lesions needed to terminate LAFL. Additional information about the advantages of the MC can be found in the Online Supplementary Materials and Supplemental Figure 1. *In silico* ablation was performed by rendering all atrial tissue within 2 mm of the MC nonexcitable.

Comparison of *in silico* and clinical LAFL and ablation targets

Each patient in this study underwent clinical EPS to treat LAFL. For each patient, the critical isthmus location of the reentrant circuit in LAFL found by entrainment mapping was

compared to the one found in LAFL simulations. Additionally, in each patient, the lengths and locations of ablation lesions that terminated LAFL were compared to the MCs that were ablated in the patient-specific models. This comparison was retrospective but was conducted in a double-blinded fashion: researchers who conducted simulations were blinded to procedures and clinicians who performed procedures were blinded to simulations. Detailed information about clinical EPS can be found in the Online Supplementary Material.

Results

Patient characteristics

The baseline characteristics for all 10 patients are given in Online Supplemental Table 1. Mean patient age was 67 ± 13 years (3 female, 7 male). Mean left atrial volume in each patient was 42.9 ± 10.3 mL. Every patient had a previous pulmonary vein (PV) isolation procedure to treat AF. The reconstructed atrial models with individualized representations of fibrosis derived from LGE-CMR for all 10 patients are shown in Figure 2. The mean proportion of fibrotic tissue in the atria was $24.5\% \pm 12.89\%$, and most of the fibrotic tissue ($56.61\% \pm 29.37\%$) was located in the posterior left atrium (PLA).

In silico LAFL reentrant morphologies

In silico LAFL was successfully induced in 7 of 10 models. In these 7 models, a single stable macroreentrant circuit was induced, which was unique to the model. Compared to the models not inducible for arrhythmia, the 7 LAFL-inducible models contained a significantly greater proportion of fibrotic tissue ($30.0\% \pm 11.5\%$ vs $12.0\% \pm 3.3\%$, $P < .05$). In the LAFL-inducible models, there were 3 types of persistent reentrant morphologies: peripulmonary vein reentry, perimitral reentry, and reentry around fibrotic tissue in the PLA. Figure 3 shows transmembrane voltage (V_m) maps of peripulmonary vein reentry at 3 instants of time in models 1 and 2. In model 1, a single wave rotating clockwise around the left inferior pulmonary vein (LIPV) sustained LAFL (Figure 3A). A figure-of-eight reentry perpetuated LAFL in model 2 (Figure 3B), in which 1 clockwise reentrant wavefront propagated around the LIPV and the other adjacent counterclockwise reentrant wavefront propagated in the PLA. Examples of perimitral reentry in models 3 and 4 are shown in Figure 4. In both models, a figure-of-eight reentry, with 1 clockwise reentrant wavefront around the mitral valve and an adjacent counterclockwise reentrant wavefront in the PLA, sustained LAFL. Figure 5 depicts a clockwise reentry that propagates around fibrotic tissue in the PLA, sustaining LAFL in model 5. The reentrant morphologies of 2 additional patients are presented in the Online Supplementary Materials and Online Supplemental Figures 3 and 4. Online Supplemental Table 2 gives a summary of the clinical and in silico cycle lengths for the 7 in silico inducible patients.

Ablation of MC

In each model inducible for LAFL, the MC corresponding to the specific reentrant morphology was determined and ablation delivered as described in Methods. The MC was unique to each model. Figures 6A, 6B, and 6C illustrate the MCs (cyan) in models 2, 3, and 4, respectively, overlaid on the LAFL activation map. In silico ablation at the MC terminated LAFL in these 3 models, as demonstrated in Figures 6D, 6E, and 6F.

Figures 7A and 7B show the MCs in models 1 and 5. In silico ablation of these MCs uncovered additional LAFLs with different reentrant morphologies. In the original LAFL morphology in model 1, a counterclockwise wavefront split from the reentrant wave near the LIPV and extinguished upon collision with a clockwise wavefront that emanated from the reentrant wave in the posterior inferior left atrium (Figure 3A). Ablation of the MC prevented the emergence of the counterclockwise wavefront, so the clockwise wavefront subsequently propagated along excitable tissue inferior to the LIPV and caused the formation of a sustained, figure-of-eight reentry in the PLA with a cycle length of 460 ms (Figures 7C).

In the original LAFL morphology in model 5, a counterclockwise wavefront split from the reentrant wave near the right inferior PV. This wave collided with a clockwise wavefront that broke off from the reentrant wave at an earlier time point near the LIPV. The resulting collision caused both wavefronts to terminate (Figure 5). Ablation of the MC prevented the formation of the counterclockwise wavefront, so the clockwise wavefront led to a sustained reentrant circuit with a cycle length of 475 ms around the LIPV (Figure 7D).

The MCs in the emergent postablation reentrant morphologies are shown in Online Supplemental Figures 2A and 2B for models 1 and 5, respectively. Ablation of these MCs terminated the arrhythmia in both cases (Online Supplemental Figures 2C and 2D). Information detailing MC locations and ablation outcomes in the remaining models 6 and 7 is provided in the Online Supplementary Results and Online Supplemental Figures 3–4.

Comparison between in silico and clinical LAFL critical isthmus locations

Online Supplemental Table 3 details the comparison between LAFL critical isthmus locations found in patient-specific models and clinical EPS. Entrainment mapping in clinical EPS and in silico simulations identified the critical isthmus of LAFL reentrant circuits to be located in the same regions of the atria in patients 1 (LIPV ostium), 3 (mitral isthmus), 4 (PLA), 5 (PLA), 6 (mitral isthmus), and 7 (mitral isthmus). In patient 2, simulations identified the critical isthmus of the LAFL reentrant circuit to be located near the ostium of the LIPV. In clinical EPS for this patient, multiple reentrant circuits with different activation sequences were observed, and LAFL was not successfully entrained. However, LAFL terminated upon application of ablation lines connecting right and left PV isolation lesions, thus suggesting the region between the right and left PVs was involved in the reentrant circuit.

Comparison between in silico and clinical ablation targets

Online Supplemental Table 4, Figure 8 (models 1–5), and Online Supplemental Figure 5 (models 6 and 7) present comparisons of the lengths and locations of the ablation targets that terminated LAFL in simulations and in clinical EPS for all patients. The ablation targets in simulation were the MCs in the flow network of reentrant wave propagation in silico LAFL, and the ablation targets in clinical EPS were determined from entrainment mapping during the procedure. Simulation ablation targets were similar in location but smaller in length than clinical ablation targets (2.8 ± 1.5 cm in simulation vs 4.8 ± 1.7 cm in EPS, $P < .05$).

In model 1, the simulation ablation targets included 2 segments of atrial tissue (1.40 cm and 1.82 cm in length), which both extended from the LIPV to fibrotic tissue in the PLA (Figure 8A, i). In clinical EPS for this patient, a region of tissue (lesion dimensions 2.75 cm $\times$ 0.93 cm) inferior to the LIPV was ablated to terminate LAFL (Figure 8A, ii).

The in silico ablation targets in model 2 were 2 segments (0.96 cm and 1.56 cm in length) that both extended from the LIPV to fibrotic tissue in the PLA. In clinical EPS, an ablation line (3.32 cm long) that connected right and left PV isolation lesions terminated the arrhythmia (Figure 8B, ii).

In model 3, the simulation ablation target was a segment of atrial tissue (3.22 cm in length) that connected the mitral valve to fibrotic tissue inferior to the LIPV (Figure 8C, i). A region of tissue joining the mitral valve to the LIPV (lesions dimensions 3.84 cm $\times$ 1.06 cm) was ablated in clinical EPS to terminate LAFL (Figure 8C, ii).

The simulation ablation target in model 4 was a segment (2.63 cm in length) of atrial tissue from the mitral valve to fibrotic tissue inferolateral from the right inferior PV (Figure 8D, i). In EPS, an ablation line (2.98 cm in length) from the mitral valve to the right PV isolation lesions was executed to terminate LAFL. This ablation line converted the arrhythmia to AF (Figure 8D, ii), and cardioversion was applied to terminate AF. After cardioversion, LAFL was noninducible after application of further programmed electrical stimulation.

Finally, in model 5, the simulation ablation target was a segment of atrial tissue (2.98 cm in length) connecting the mitral valve to fibrotic tissue in the PLA (Figure 8E, i). In EPS, ablation lesions (2.85 cm) extending from the mitral valve to atrial tissue in the PLA terminated LAFL (Figure 8E, ii). Similar information regarding models 6 and 7 can be found in the Online Supplementary Results and Online Supplemental Figure 5.

Discussion

The purpose of this study was to test the feasibility of using personalized atrial models to identify ablation targets for LAFL. To achieve this goal, we simulated LAFL in 10 patient-specific models with individualized atrial geometry and fibrotic tissue distribution derived from clinical LGE-CMR scans. We developed a novel graph-cutting algorithm to identify the MC in the flow network representing reentrant wave propagation, and we demonstrated that targeting MCs for ablation in each atrial model successfully terminated LAFL. The in silico ablation targets were compared to those executed in the clinic to terminate LAFL in patients. The main contributions of our study can be summarized as follows:

1. We present the first proof-of-concept study demonstrating that patient-specific models can be used to accurately simulate LAFL and predict the optimal ablation targets.
2. Graph theory tools identified the optimal ablation targets (the MC in reentrant wave propagation).
3. Ablation of MCs terminated LAFL or uncovered additional LAFL morphologies with slower tachycardia cycle lengths.

4. Ablated MCs that terminated LAFL in simulation were smaller in length and similar in location to the ablation lesions that, in addition to PV isolation lines, terminated LAFL in EPS.

Characteristics of in silico LAFL

The LAFLs observed in our image-based simulations included sustained reentry involving the mitral valve, LIPV, or large regions of fibrotic tissue in the PLA. These reentrant morphologies are consistent with LAFLs observed in clinical studies.^{7,17} For instance, Jaïs et al⁷ showed that in 22 patients with LAFL, 7 patients had reentry around the mitral valve, 1 had reentry around the left PVs, and 4 had reentry around electrically silent areas in the PLA. Similarly, Ouyang et al¹⁷ showed that in 28 patients with LAFL, 9 had reentry around the mitral valve, 13 had reentry around the left PVs, and 3 had reentry around electrically silent areas in the PLA. The electrically silent areas, defined in electroanatomic mapping as atrial tissue in which electrical potential recordings are indistinguishable from noise, can be viewed as analogous to regions with dense fibrosis in our models, which exhibit significantly inhibited conduction because of decreases in tissue conductivities and in the upstroke velocity in myocyte action potentials. Experimental studies in both animals and isolated human tissue have shown that electrically silent areas occur in dense fibrotic tissue regions.¹⁸

Furthermore, the critical isthmus locations of LAFL reentrant circuits found in simulations and clinical EPS matched in 6 of 7 patients. In the unsuccessful case, LAFL could not be entrained during clinical EPS. This finding of successful correspondence between simulation and clinical EPS in a few patients suggests that it may be possible for patient-specific models to noninvasively locate reentrant circuits in LAFL, which will benefit preprocedural treatment planning for this arrhythmia. However, a larger prospective clinical study comparing activation maps of LAFL found in simulation and EPS is still needed to validate the application of patient-specific models for LAFL management.

Identification of ablation targets using network flows analysis

In order to terminate LAFLs in simulation, we developed an algorithm based on network flow analysis to identify the minimum amount of tissue needed to interrupt electrical wave propagation during reentry.¹⁶ Network flows analysis has been routinely applied in many other nonmedical fields, such as image segmentation, railway transportation, and telecommunications.¹⁹ In this article, we present the first application of network flows analysis in cardiac electrophysiology to identify optimal ablation targets (the MCs). A major advantage of the new approach is that finding the optimal ablation targets in each model was done automatically, without manual intervention. The development of an automatic predictor of optimal ablation targets is an important advancement toward the use of patient-specific atrial models to noninvasively guide clinical ablation. Additionally, the MC approach identified the optimal ablation targets based on information about the 3-dimensional reentrant circuits in each 3-dimensional fibrotic substrate.

The MC in each LAFL was located in short segments (lengths ranging from 1.40–3.85 cm) of atrial tissue involved in the reentrant circuit that connected nonconductive anatomic

barriers such as the mitral valve or LIPV to fibrotic tissue regions. Ablation of the MC prevented the reentrant wave from circumnavigating the ablation lesion and propagating along the original reentrant circuit pathway. This strategy is analogous to strategies espoused in clinical studies of interatrial reentrant tachycardia after congenital heart surgery,²⁰ which show that extending ablation lesions from fibrotic scar caused by surgical incisions to natural atrial boundaries such as the inferior vena cava, superior vena cava, or tricuspid annulus is more effective in terminating arrhythmia than executing focal ablation lesions in the critical isthmus of the reentrant circuit.

Postablation LAFL

Ablation of the MC successfully interrupted wave propagation in the original reentrant circuit that produced LAFL in all 7 models. This led to LAFL termination in 4 of the models and in the emergence of new LAFL morphologies in the remaining 3 cases. In the original LAFL for the latter 3 cases, 2 wavefronts (1 clockwise and 1 counterclockwise) split from the reentrant circuit at different time points and collided with each other. This collision extinguished both wavefronts but did not affect wave propagation of the reentrant circuit that sustained LAFL. Ablation of the MC eliminated the formation of the counterclockwise wavefronts in both models, so the clockwise wavefront propagated along excitable tissue to cause the formation of a new and slower LAFL. These findings demonstrate the importance of understanding and appreciating the specific pathway of wave propagation in the entire atria during LAFL as opposed to just identifying the reentrant circuit critical isthmus, because ablation can uncover additional reentrant morphologies. The transformation of reentrant morphologies that sustain LAFL by catheter ablation has also been noted in clinical studies. For example, Ouyang et al¹⁷ described a transformation of LAFL, reporting an absence of electrical activity in the ablated region determined to be the critical isthmus of the reentrant circuit. Remapping the transformed tachycardia revealed that another region of the atria, which was a bystander in the original reentry, was now the critical isthmus of the new reentrant circuit. Similarly, Rostock et al²¹ noted the transformation of reentrant morphologies in atrial tachycardia after ablation in 45 patients and reported that the mean cycle length of the postablation tachycardia was longer than the cycle length of the original tachycardia (320 ± 88 ms vs 274 ± 40 ms).

Comparison of simulation and EPS ablation lesions

Finally, we compared the ablated MCs in simulations to ablation targets that successfully terminated LAFL in EPS. In the 7 LAFL-inducible models, the ablation lines were shorter (2.8 ± 1.5 cm in simulation vs 4.8 ± 1.7 cm in EPS) but in locations fairly similar to the ablation lesions, outside of PV isolation lines, that terminated LAFL in EPS. The strategies of determining ablation lesions in EPS varied from patient to patient but generally involved connecting nonconductive barriers to each other (ie, connected mitral valve to LIPV in patient 3, and right and left PV isolation lesions in patient 2). This is similar to the ablation strategy via MCs in simulations, where nonconductive anatomic barriers were connected to regions of fibrotic tissue with reduced capacity for excitation and conduction. Previous image-based simulation studies from our team have also demonstrated a successful correspondence between in silico and EPS ablation targets for ventricular tachycardia.²² These studies highlight the potential of patient-specific models to noninvasively determine

the optimal ablation targets for complex cardiac arrhythmias due to a reentrant mechanism. This study is a retrospective proof of concept, and prospective studies are required to further support the approach.

Study limitations

In 3 of the models, LAFL was not inducible in simulations despite the presence of arrhythmia in clinical EPS. This may be due to the low amount of fibrotic tissue in these patients identified by LGE-CMR. In this study, voxels with an image intensity ratio (IIR) >0.97 in LGE-CMR were segmented to correspond to fibrotic tissue.⁸ This threshold overcomes the limitation that LGE-CMR voxel intensities vary between patient scans because of differences in surface coil proximity, contrast dose, and body mass index.²³ However, the identification of a threshold in LGE-CMR to represent fibrotic tissue is still controversial, and whether the threshold chosen in our study leads to the best correlation between simulation and clinical reentrant morphologies is unknown.

Another limitation of this study was that activation mapping was not performed in these patients because ablation targets were primarily determined from entrainment mapping during clinical EPS. Therefore, we could not compare the exact reentrant patterns in LAFL found in simulation to LAFL observed in clinic. A future prospective study with detailed comparisons of dense activation maps, combined with entrainment mapping at putative circuit sites and larger study population size, is needed to validate image-based simulation for LAFL treatment planning.

Finally, functional changes in electrophysiology might be contributing to LAFL in patients, which our models, designed to represent the contribution of the structurally remodeled substrate only, cannot capture.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.hrthm.2016.04.009>.

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Figure 1.

Model generation and identification of minimum cuts in reentrant flow propagation. **I:** Pipeline to construct magnetic resonance image–based models of fibrotic human atria. **A:** Representative late gadolinium-enhanced cardiac magnetic resonance (LGE-CMR) slice of human atria. **B:** Atrial tissue segmentation into fibrotic (*green*) and nonfibrotic (*gray*) regions. **C:** Three-dimensional reconstruction of atrial geometry. **D:** Fiber orientation in atrial model. **E:** Atrial action potentials in fibrotic (*green*) and nonfibrotic (*gray*) tissue. **II:** Schematic for identifying minimum cuts in reentrant flow propagation, represented as a flow network. The minimum cut of the flow network is indicated with the *dashed line*.

Figure 2.

Three-dimensional (3D) patient-specific atrial models. Personalized 3D models of all 10 patient atria reconstructed from late gadolinium-enhanced cardiac magnetic resonance scans.

Figure 3.

Examples of left atrial flutter sustained by peripulmonary vein reentry. Transmembrane maps showing reentry around the left inferior pulmonary vein in model 1 (A) and figure-of-eight reentry in model 2 (B). The counterclockwise circuit in the figure-of-eight reentry is around the left inferior pulmonary vein and the clockwise circuit is in the posterior left atrium. LAA = left atrial appendage; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; MV = mitral valve; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

Figure 4.

Examples of left atrial flutter sustained by perimitral reentry. Transmembrane voltage maps showing figure-of-eight reentry in model 3 (A) and model 4 (B). In both cases, the clockwise circuit in the figure-of-eight reentry is around the mitral valve and the counterclockwise circuit is in the posterior left atrium. LAA = left atrial appendage; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; MV = mitral valve; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

Figure 5.

Example of left atrial flutter sustained by reentry around fibrotic tissue in the posterior left atrium. Transmembrane voltage maps showing reentry around fibrotic tissue in the posterior left atrium for model 5. Fibrotic tissue is highlighted in *green*. LAA = left atrial appendage; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; MV = mitral valve; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

Figure 6.

Location of minimum cuts in reentrant flow propagation and termination of left atrial flutter (LAFL) by ablation. Location of minimum cuts (cyan) of reentrant flow propagation for model 2 (A), model 3 (B), and model 4 (C) overlaid on activation maps of LAFL episodes. Transmembrane voltage maps showing LAFL termination in model 2 (D), model 3 (E), and model 4 (F) after in silico ablation was applied (red). LAA = left atrial appendage; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; MV = mitral valve; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

Figure 7.

Emergence of postablation left atrial flutter (LAFL). Location of minimum cuts (cyan) of reentrant flow propagation for model 1 (A) and model 5 (B) overlaid on activation maps of LAFL episodes. Transmembrane voltage maps showing emergence of new LAFL in model 1 (C) and model 5 (D) after in silico ablation was applied (red). LAA = left atrial appendage; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; MV = mitral valve; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.

Figure 8.

Comparison of in silico and clinical ablation targets (those outside of pulmonary vein isolation lines). Ablated minimum cut in models 1 (A, i), 2 (B, i), 3 (C, i), 4 (D, i), and 5 (E, i). Ablated tissue to terminate left atrial flutter in clinical electrophysiologic study for patients 1 (A, ii), 2 (B, ii), 3 (C, ii), 4 (D, ii), and 5 (E, ii).