Inadvertent electrical isolation of the left atrial appendage during catheter ablation of persistent atrial fibrillation

Chin Pang Chan, MRCP, Wai Shun Wong, MD, Satchana Pumprueg, MD, Srikar Veerareddy, MD, Sreedhar Billakanty, MD, Christopher Ellis, MD, Sanders Chae, MD, Daniel Buerkel, MD, Johan Aasbo, DO, Thomas Crawford, MD, Eric Good, DO, Krit Jongnarangsin, MD, Matthew Ebinger, DO, Frank Bogun, MD, Frank Pelosi, MD, Hakan Oral, MD, Fred Morady, MD, Aman Chugh, MD

From the Division of Cardiovascular Medicine, Cardiovascular Center, University of Michigan, Ann Arbor, Michigan.

BACKGROUND Left atrial appendage (LAA) isolation is rare and may be associated with impaired transport function and thromboembolism.

OBJECTIVE The purpose of this study was to determine the mechanisms of inadvertent isolation of the LAA during atrial fibrillation (AF) ablation.

METHODS This study consisted of 11 patients (ejection fraction 0.43 ± 0.18, left atrial diameter 51 ± 8 mm) with persistent AF who had LAA conduction block during a procedure for AF (n = 8) or atrial tachycardia (AT) (n = 3).

RESULTS LAA conduction block occurred during ablation at the Bachmann bundle region in 6 patients, mitral isthmus in 3, LAA base in 2, and coronary sinus in 1. The mean distance from the ablation site to the LAA base was 5.0 ± 1.9 cm. LAA isolation was transient in all 6 patients in whom LAA conduction was monitored and was permanent in the 4 patients in whom conduction was *not* monitored during energy delivery. The remaining patient was noted to have LAA isolation during a redo procedure before any ablation. Nine of (82%) the 11 patients have remained arrhythmia-free without antiarrhythmic drugs at mean follow-up of 6 ± 7 months, and all have continued taking warfarin.

CONCLUSION Electrical isolation of the LAA may occur during ablation of persistent AF and AT even when the ablation site is remote from the LAA. This likely is due to disruption of the Bachmann bundle and its leftward extension, which courses along the anterior left atrium and bifurcates to surround the LAA. Monitoring of LAA conduction during ablation of persistent AF or AT is important in avoiding permanent LAA isolation.

KEYWORDS Atrial fibrillation; Bachmann bundle; Catheter ablation; Left atrial appendage isolation

ABBREVIATIONS AF = atrial fibrillation; **AT** = atrial tachycardia; **CS** = coronary sinus; **LA** = left atrium; **LAA** = left atrial appendage; **PV** = pulmonary vein; **RF** = radiofrequency

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Patients with persistent atrial fibrillation (AF) frequently require ablation of the left atrial (LA) substrate, consisting of ablation of complex atrial electrograms and/or linear ablation. Although this approach has been shown to be highly effective,[1] there is a concern that such extensive ablation may be associated with electrical and mechanical impairment of the LA in some patients. Indeed, a prior case report showed that the left atrial appendage (LAA) may be electrically isolated during extensive LA ablation for persistent AF.[2] This has important implications for LA transport function and the potential for thromboembolic complications despite maintenance of sinus rhythm. The goal of this study was to determine the mechanisms of LAA isolation and to discuss strategies to prevent this complication in patients undergoing catheter ablation of persistent AF.

Methods

The subjects of this study were 11 patients who experienced LAA conduction block/isolation as a result of catheter ablation of persistent AF or postablation atrial tachycardia (AT). All antiarrhythmic medications except for amiodarone were discontinued at least five half-lives prior to ablation. Amiodarone was discontinued at least 8 weeks prior to the ablation procedure. All patients were anticoagulated with warfarin for at least 1 month before the procedure and were bridged with low-molecular-weight heparin following warfarin discontinuation 3 days prior to the procedure. Transesophageal echocardiography was performed within 24 hours of the procedure to exclude LA thrombus. The clinical characteristics of the patients are listed in Table 1. Nine (82%) of the 11 patients had evidence of structural heart disease (congestive heart failure, ischemic and nonischemic cardiomyopathy, coronary disease, atrial septal

Drs. Oral and Morady are co-founders of Ablation Frontiers. **Address reprint requests and correspondence:** Dr. Aman Chugh, Division of Cardiovascular Medicine, University of Michigan Hospital, Cardiovascular Center, SPC 5853, 1500 East Medical Center Drive, Ann Arbor, Michigan 48109-5853. E-mail address: achugh@umich.edu. (Received August 10, 2009; accepted October 26, 2009.)

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Table 1 Clinical characteristics of the study patients

No. of patients	11
Age (years)	57 ± 11
Gender (M/F)	7/4
AF duration (years)	7 ± 3
Ejection fraction	0.43 ± 0.18 (0.10–0.65)
Left atrial diameter (mm)	51 ± 8 (41–65)
Long-lasting persistent AF	6/11
Structural heart disease	9/11
Prior procedures per patient	1.5 ± 1.1 (0–3)
Procedure indication (AF/AT)	8/3
Prior thromboembolic event	3/11
Prior device implantation	5/11

AF = atrial fibrillation; AT = atrial tachycardia.

defect, left ventricular hypertrophy), and all had evidence of LA enlargement. The indication for the procedure during which LAA conduction block was observed was AF in 8 patients and AT in 3 patients. Eight patients had previously undergone a procedure for persistent AF; the other 3 presented for their first ablation procedure for persistent AF.

A decapolar catheter (EZ Steer, Biosense-Webster, Diamond Bar, CA, USA) was positioned within the coronary sinus (CS) or recording electrograms and atrial pacing. Intracardiac echocardiography was used to guide a double transseptal puncture. Heparin was infused to maintain an activated clotting time of 300 to 350 seconds. An open-irrigation, 3.5-mm-tip deflectable-tip ablation catheter (ThermoCool, Biosense-Webster) was used for mapping and ablation. Bipolar electrograms were recorded at a bandpass of 30 to 500 Hz (EPMedSystems, West Berlin, NJ, USA). A three-dimensional replica of the LA was created with an electroanatomic mapping system (CARTO, Biosense-Webster). To avoid collateral injury to the esophagus, the esophagus was visualized by a radiopaque probe or barium administration.[3] Radiofrequency (RF) energy was delivered at a maximum power output of 35 W at a flow rate of 30 mL/min and maximum temperature of 45°C. Power was reduced to 20 to 25 W at a flow rate of 17 mL/min during applications of energy near the pulmonary vein (PV) ostia, in the posterior LA, and in the CS.

In 9 of the 11 patients, the procedural end-point was conversion of AF to sinus rhythm during RF ablation. The ablation strategy included antral PV isolation followed by catheter ablation of sites exhibiting complex electrograms. The acute end-point during the latter step was organization of the local atrial electrogram. The next step was linear ablation at the roof and mitral isthmus, with demonstration of conduction block as previously described.[4,5] If AF did not terminate after these steps, complex electrograms in the right atrium were targeted. Two patients underwent the ablation procedure using a combination of these steps without the end-point of acute termination of AF. After we noted that the LAA may be isolated even when ablation was performed several centimeters away from the structure,[2] we routinely monitored conduction by placing a ring catheter (Lasso, Biosense-Webster) in the LAA.

LAA isolation was defined as the complete elimination or dissociation of LAA potentials. If LAA conduction was being monitored during ongoing AF, LA conduction impairment was defined as *reproducible* slowing of the LAA cycle length by at least 50% while documenting no change in the global AF cycle length. If conduction slowing or block into the LAA was observed during energy delivery, RF current was immediately discontinued.

Mapping and ablation of AT

In patients who underwent a procedure for postablation AT, the arrhythmia was mapped by a combination of activation and entrainment mapping.[6] In patients who presented in sinus rhythm, isoproterenol (5–20 μg/min) was infused to facilitate arrhythmia induction. During ablation of AT, the ring catheter was placed in the LAA to monitor conduction into the appendage. RF current was discontinued if energy delivery resulted in LAA conduction slowing or block. After termination of AT, rapid atrial pacing and programmed atrial stimulation were performed with and without isoproterenol infusion.

Postablation care

Patients were monitored overnight and discharged the next day. They were prescribed warfarin and low-molecular-weight heparin until the international normalized ratio was >2.0. Patients were also treated with rate-control medications, but none received rhythm-control medications. Patients were seen in an outpatient clinic at 3 and 6–9 months after the procedure. Rhythm status was assessed with an auto-triggered monitor worn for 30 days or by interrogation of the existing pacemaker/defibrillator.

Statistical analysis

Continuous variables are given as mean ± SD.

Results

Electrophysiologic findings

The presenting rhythm was AF in 6 patients, AT in 2, and sinus in 3 (Table 2). Among the 3 patients who presented in sinus rhythm, isoproterenol was required to induce AF in 1 patient and AT in another. In the third patient (no. 9), no arrhythmias could be induced (see later). In patients no. 3 and 5, who presented for their first ablation procedure and had no evidence of structural heart disease, mapping revealed confluent areas of very low voltage (<0.5 mV), consistent with an atrial myopathic process.

Among the 7 patients with AF, including 1 patient in whom AF was induced, RF ablation resulted in AF termination in 6 patients. AF converted to AT in all 6 patients. The mechanisms of AT in these patients were macroreentry involving the left and right atrial septum in 2 patients; small reentrant circuit near the right upper PV, typical flutter, and peri-mitral reentry in 2 patients each; focal AT from the base of the LAA in one patient; and small reentrant circuit at the anterior base of the LAA in 1 patient. All of these ATs were successfully eliminated during RF energy delivery.