Mechanisms of Organized Left Atrial Tachycardias Occurring After Pulmonary Vein Isolation

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Background—A proarrhythmic consequence of pulmonary vein (PV) isolation can be a recurrent organized left atrial (LA) tachycardia after ablation. This arrhythmia is frequently referred to as “left atrial flutter,” but the mechanism and best ablation strategy have not been determined.

Methods and Results—Isolation of arrhythmogenic PVs was initially performed by segmental ostial PV ablation guided by a circular mapping catheter in 341 patients. Patients whose predominant recurrent arrhythmia was a persistent organized tachycardia returned for mapping and ablation. Recurrent organized LA tachycardias (cycle length 253±33 ms, range 213 to 328 ms) occurred in 10 (2.9%) of 341 patients (age 59±9 years, 1 woman). Mapping was consistent with a focal origin in 8 patients and with macroreentry in 1 patient and was unclear in 1 patient owing to degeneration to atrial fibrillation. Focal tachycardias originated from reconnected segments of prior isolated PVs (6 patients), the posterior LA (1 patient), or the superior septum (1 patient). Focal atrial tachycardias were ablated with point lesions that targeted the earliest activation. All reconnected PVs were also reisolated. Reentrant LA flutter occurred around the left PVs in 1 patient. After 6.7±2.3 months of follow-up, 9 (90%) of 10 patients were arrhythmia free (4 of whom were taking antiarrhythmic drug therapy), and one was having recurrent atrial fibrillation.

Conclusions—Recurrent organized LA tachycardia after PV isolation is uncommon and typically has a focal origin from reconnected PV ostia. Reisolation of the PV and ablation of non-PV foci are sufficient to treat this proarrhythmia. Linear lesions are only required when a macroreentrant mechanism is present. (Circulation. 2004;110:1351-1357.)

Key Words: fibrillation ■ atrium ■ catheter ablation

Electrical isolation of the pulmonary veins (PVs) to treat drug refractory atrial fibrillation is becoming more common. Early attempts to ablate triggers focally inside the PVs have been abandoned because of high recurrence rates and a high incidence of PV stenosis.1,2 The current approach involves isolating the PV by ablating on the left atrial side of the left atrial/PV junction, either circumferentially3,4 or segmentally,5–8 to electrically isolate the arrhythmogenic PVs. This results in a much lower incidence of PV stenosis and prevents other triggers within the PV from initiating AF. However, a proarrhythmic side effect of extensive left atrial ablation can be the development of an organized left atrial tachycardia after ablation.6–12 These tachycardias have frequently been termed left atrial “flutter”; however, the mechanism and best treatment strategy have not been determined. We describe the incidence, mechanism, and outcome of a series of patients who developed persistent organized left atrial tachycardias after PV isolation and underwent intracardiac mapping and ablation.

Methods
Patients with drug-refractory paroxysmal or persistent AF underwent electrophysiological mapping and ablation at the University of Pennsylvania Health System. All patients signed informed consent paperwork. Our methods have been described previously.13 AF triggers were provoked with isoproterenol infusion (up to 20 μg/min) and cardioversion of induced AF if necessary in all patients. Only PVs that initiated atrial premature beats or AF were isolated. Electrical isolation was performed with a 4-mm-tip ablation catheter (Navistar, Biosense Webster, Inc). Ablation was guided by a circular mapping catheter (Lasso 10-pole 15 to 20 mm, Biosense Webster, Inc) located at the PV ostium. Positioning of the Lasso catheter was confirmed by intracardiac echocardiography (Acunav, Acuson Inc). Ablation was performed segmentally, with isolation of those segments of the PV that demonstrated entry or exit of PV signals. Isolation was confirmed by loss of all high-frequency electrical signals on the circular mapping catheter and loss of atrial capture pacing from all overlapping bipole tips of the circular mapping catheter at 10-mA, 2-ms pulse width (exit block4). All PVs were revisited after at least a 20-minute waiting period, and PVs with evidence of recurrent conduction were reisolated. Patients were given antiarrhythmic medications for 6 weeks (paroxysmal AF) to 6 months (persistent AF). Follow-up consisted of transtelephonic monitoring with twice-daily transmissions for 2 weeks before and after ablation, after discontinuation of drug therapy, and at 6 months; outpatient follow-up occurred at 6 weeks, 3 months, 6 months, and every 6 months thereafter.

Patients with persistent organized left atrial tachycardias after isolation initially underwent electrical cardioversion and frequently
discontinuation or change in antiarrhythmic therapy. Those with persistent organized tachycardias more than 6 weeks after ablation that continued despite discontinuation of antiarrhythmic therapy underwent repeat mapping and ablation. All antiarrhythmic agents were discontinued at least 4 days before the procedure. During the repeat ablation session, entrainment was initially performed from the tricuspid valve/inferior vena cava isthmus to exclude a right atrial isthmus dependent atrial flutter. Two transseptal punctures were then performed for placement of a mapping/ablation catheter and circular mapping catheter. A detailed left atrial electroanatomic map was acquired, and entrainment mapping was performed from multiple left and right atrial sites whenever feasible during a stable tachycardia. Ablation was performed with a 4- or 8-mm-tip ablation catheter (Navistar, Biosense Webster, Inc). All patients were prescribed antiarrhythmic therapy for 6 weeks after ablation and followed up in the same manner as after the first procedure.

Results

Isolation of 955 arrhythmogenic PVs was performed in 341 patients with drug-refractory paroxysmal (n = 293, 86%) or persistent (n = 48, 14%) AF. Overall, AF control with or without antiarrhythmic drugs was achieved in 89% of patients. Recurrent AF refractory to antiarrhythmic therapy occurred in 39 patients (11%), and an additional 29 patients (9%) were still having occasional AF episodes despite taking antiarrhythmic drugs, but with significant (>90%) improvement in AF burden. Of these 68 patients with recurrent AF after ablation, 10 (15%) of 68 developed a persistent left atrial tachycardia. These patients returned for electrophysiological mapping and ablation after 5.7 ± 2.8 months. None of these patients had organized atrial tachycardias identified before ablation. Patient characteristics and tachycardia features are shown in the Table.

Five (1.5%) of the 341 patients developed typical isthmus-dependent right atrial flutter after ablation. Although a right atrial flutter line was not performed as part of the PV isolation procedure, 24% (82 of 341) of patients had undergone right atrial flutter ablations before presenting for PV isolation.

Surface ECG Characteristics

Surface ECG features of the tachycardias were consistent with origin from the superoposterior left atrium (Figures 1 through 4). The ECG revealed regular atrial activation that was positive in the inferior leads in 8 of 10 patients, although 2 patients had negative components in the inferior leads (Figure 4). The atrial depolarization was always predominately positive in V1 and positive across the precordium in 8 of 10 patients, with 2 patients having late transitions to a negative atrial depolarization by leads V4 to V6. These features are not consistent with typical isthmus-dependent clockwise or counterclockwise atrial flutter. Because the tachycardia was regular, one could typically visualize the atrial depolarization unobstructed from the prior T wave. Surface ECG features for focal tachycardias were often typical for the PV of origin,15 ie, flat in lead I, lead II ≅ III, negative in aVL, and M-shaped in V1 for left superior PV origin (Figure 1), and positive in lead I, lead II ≅ III, and flat to biphasic in lead aVL, with late-peaking positive wave in lead V1 for right superior PV origin (Figure 2).

Intracardiac Features

Activation and/or detailed electroanatomic mapping of the tachycardia revealed a focal origin with concentric spread of activation in 8 patients, of which 6 originated from partially reconnected PV ostia (Figures 1 and 2), 1 from the posterior LA just outside the left superior PV (Figure 3), and 1 from the superior septum near the limbus of the fossa ovalis. In the 6 patients with earliest activation at the PV ostium, the location of earliest activation was typically at or just inside (<0.5 cm) the PV ostium. This was confirmed by the ablation catheter location relative to the circular Lasso catheter, which was positioned at the PV ostium with intracardiac echocardiography. The tachycardia cycle length was 253 ± 33 ms. Entrainment mapping during stable tachycardia was feasible in 7 patients. The mechanism in 1 patient could not be determined because of degeneration to AF with pacing. Pacing from the tricuspid valve/inferior vena cava isthmus revealed a fused intracardiac activation sequence with a postpacing interval (PPI) > 30 ms longer than tachycardia cycle length in all patients. Pacing from the tachycardia focus typically led to an identical intracardiac activation sequence with a tachycardia

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LA indicates left atrium; EF, ejection fraction; CL cycle length; RIPV, right inferior pulmonary vein; RS, right superior; RM, right middle; LI, left inferior; LPV, left PVs; LS, left superior; LSPV, left superior PV; RSPV, right superior PV; and sup septum, superior septum near limbus of fossa ovalis.
cycle length equal to the PPI, but often, pacing a short distance away (<1 cm) led to variation in tachycardia cycle length or a short PPI (Figure 5), which made macroreentry around the PVs unlikely.

In 1 patient, a detailed activation map was consistent with a macroreentrant circuit around the left-sided (left upper and lower together) PVs (Figure 4). Entrainment mapping from within the tachycardia circuit revealed a tachycardia cycle length equal to the PPI, whereas PPIs were progressively longer with fusion pacing from the right PV, coronary sinus, and right atrium (Figure 6). In addition, the activation time of the tachycardia circuit on the electroanatomic map was equal to the entire tachycardia cycle length.

**Response to Ablation**

In all 8 cases of focal tachycardias, tachycardias abruptly terminated or slowed and terminated during focal application of radiofrequency energy at the site of earliest activation (Figures 1, 2, 3, and 7). In 1 patient, the PV tachycardia continued despite dissociation from the rest of the left and right atrium, which suggests that the origin of this tachycardia was inside the PV (Figure 1). Further radiofrequency ablation led to termination of this PV tachycardia. After tachycardia termination, the PV of origin typically exhibited evidence of reconnection, with significant delay in left atrium to PV activation time (Figure 3). These PVs were always reisolated, and in addition, all other reconnected PVs were reisolated.
Reisolation of the PVs was also performed in the patient with macroreentrant flutter, to prevent PV triggers from reinitiating tachycardia. In 1 patient early in our experience in whom the mechanism could not be determined because of degeneration to AF with pacing, the PVs were reisolated, and lines were empirically drawn that connected the left inferior PVs to the mitral annulus and connected the superior PVs. No other empiric lines were performed.

In the 1 patient who exhibited reentry around the left-sided PVs, linear lesions were placed between the superior PVs, across an isthmus of preserved voltage, which led to abrupt termination of the tachycardia (Figure 4). The right inferior and both left-sided PVs had evidence of segmental reconnection and were reisolated in this patient.

After 6.7±2.3 months (range 2 to 11 months) of follow-up, 9 (90%) of 10 patients were arrhythmia free. Five patients were free of AF or atrial tachycardia without any antiarrhythmic drugs (including the 1 patient in whom the arrhythmia mechanism could not be determined). Four patients are continuing to undergo antiarrhythmic therapy after early (<6 weeks) AF recurrences. One patient is having recurrent AF despite antiarrhythmic therapy.

Discussion
Since the initial description by Haissaguerre and colleagues,16 segmental PV isolation has been gaining more widespread acceptance as a potentially curative treatment for AF. Recognized complications include cardiac tamponade, stroke, embolic myocardial infarction, and PV stenosis. There have also been reports of organized left atrial “flutter” occurring after PV isolation,4,9–12 presumably due to reentry occurring...
around iatrogenic obstacles (PVs or mitral annulus) created by ablation lesions encircling the PVs. This has led some to advocate empiric linear lesions in patients undergoing PV isolation, either between the superior PVs or along the left atrial “isthmus,” between the left inferior PVs and mitral annulus. Such lesions carry additional risk, particularly because completion of the left atrial isthmus line often requires extensive ablation with an irrigated-tip catheter inside the coronary sinus.

We found that persistent organized left atrial tachycardias are uncommon (2.9%) after segmental PV isolation for paroxysmal or persistent AF. The low incidence of these tachycardias suggests that empiric linear lesions are probably not warranted. When left atrial tachycardias do occur, treatment should be tailored to the arrhythmia mechanism. Our experience demonstrates that these tachycardias are typically focal, originating from ostial segments of reconnected PVs. A focal spread of activation seen with electroanatomic mapping, persistence of the tachycardia after PV isolation, changes in tachycardia rate, and markedly different PPIs at adjacent ostial pacing sites favor triggered activity or enhanced automaticity over macroreentry as the mechanism of the majority of tachycardias. Ablation at the site of earliest activation often results in termination of the tachycardia. If the mechanism cannot be determined, reisolation of all reconnected PVs appears to be sufficient in most cases. Linear lesions between anatomic obstacles are only rarely required when a macroreentrant mechanism is demonstrated and should be tailored to interrupt the path of the reentrant circuit.

Figure 5. Intracardiac recordings from a circular mapping catheter (Lasso) in the right superior PV (RSPV), ablation catheter (Abl), and catheters in the coronary sinus (CS) and posterior right atrium (RA) during atrial tachycardia in patient 8. Entrainment of tachycardia from 2 different bipoles of the Lasso catheter located at the RSPV ostium near the site of early activation is shown in A and B. An electroanatomic activation map acquired during atrial tachycardia is shown in C. Note that pacing from an early site posteriorly (A) yields concealed activation with a PPI=tachycardia cycle length; however, a short distance away (B), the PPI is much shorter. These characteristics suggest that the arrhythmia is not macroreentrant.

Figure 6. Intracardiac recordings from a circular mapping catheter (Lasso) in the left common PV (LPV), ablation catheter (Abl), and catheters in the coronary sinus (CS) and posterior right atrium (RA) during atrial tachycardia in patient 3. Entrainment of tachycardia is performed from the ablation catheter located in the tricuspid valve/inferior vena cava isthmus (A), proximal coronary sinus (B), distal coronary sinus (C), and tachycardia circuit above the left-sided PVs (D). An electroanatomic map acquired during atrial tachycardia is shown in the center. Note that the PPI becomes progressively shorter as one moves closer to the tachycardia origin around the left-sided PVs and is similar to the tachycardia cycle length only from sites along the tachycardia path (D). TCL indicates tachycardia cycle length; LSPV, left superior PV; RSPV, right superior PV; and RIPV, right inferior PV.
Prior Work

Prior case reports from patients without prior ablation have documented focal left atrial tachycardias masquerading as an atypical atrial flutter. Both case reports found that a significant amount of left atrial conduction delay was present, which led to atrial activation time similar to the tachycardia cycle length and therefore surface ECG features similar to a reentrant atypical flutter. Kistler and colleagues reported on 27 patients with focal PV tachycardias and no prior ablation. The surface ECG features and focal ostial PV location of these tachycardias were similar to our findings, although only a minority of these tachycardias originated from the right inferior PV (1 of 28 [3.6%] compared with 3 of 10 [30%] in the present study). Villacastin and colleagues reported 2 patients with electroanatomic maps interpreted as reentrant left atrial flutter after PV isolation. In the first patient, the tachycardia was terminated with a single radiofrequency application at a site where pacing revealed a tachycardia cycle length equal to the PPI. The second patient’s tachycardia terminated after 3 radiofrequency applications in the area of early activation. The electroanatomic maps in these cases were interpreted as demonstrating macroreentry; however, one could also interpret them as having focal activation from a PV ostium. Other laboratories have described that the majority of regular atrial tachycardias that occur after PV isolation are due to reentry around the mitral annulus or pairs of PVs. These studies did not report whether reisolation of reconnected PVs was also performed. The higher incidence of macroreentrant tachycardias in these series may be due to differences in ablation technique, including complete circumferential rather than segmental PV isolation, empiric isolation of all PVs, or use of 8-mm or irrigated-tip catheters.

In the cases of focal left atrial tachycardias in the present study, there was always significant conduction delay into the PV of origin after sinus rhythm was restored. Creation of a segment of tissue at the PV ostium, uncoupled from the surrounding atrial myocardium after ablation, may provide the substrate for an iatrogenic atrial tachycardia. Focal tachycardias with conduction delay from prior ablation and a circuitous course around prior ablation lesions may be difficult to distinguish from a macroreentrant tachycardia without detailed mapping and pacing from multiple atrial sites.

Boecherer and colleagues have previously reported surface ECG characteristics of left atrial tachycardias. They reported that left atrial tachycardias were frequently flat in the inferior leads and positive in V1. In the present study, we found a characteristic ECG pattern of positive “flutter” waves in both the inferior leads, V1, and throughout the precordial leads. This is likely due to the focal origin from the superior-posterior left atrium for the tachycardias in the present study. With previously reported surface ECG criteria derived from atrial pacemapping, the morphology of the tachycardia is also frequently suggestive of the PV of origin. Recognition of these features may facilitate ablation.

Study Limitations

Although we characterized 8 of 9 tachycardias as focal in origin, we cannot determine from the present study the difference between triggered activity, abnormal automaticity, or microreentry within the PV as the mechanism of these tachycardias. More extensive pacing or pharmacological maneuvers were not undertaken because of concern that the tachycardias might degenerate to AF.

We performed segmental isolation of arrhythmogenic PVs guided by a circular mapping catheter. It is possible that complete circumferential isolation of all PVs could result in a higher incidence of macroreentrant tachycardias than found in the present series.

Conclusions

Persistent organized left atrial tachycardias after PV isolation for AF are uncommon, occurring in 2.9% of ablated patients and constituting 15% of AF recurrences. Treatment should be tailored to the arrhythmia mechanism. Focal ablation of the tachycardia at the site of earliest activation and reisolation of all reconnected PVs is sufficient in most cases. Linear lesions between anatomic obstacles are required only when a macroreentrant mechanism is demonstrated.

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References


