Techniques, Evaluation, and Consequences of Linear Block at the Left Atrial Roof in Paroxysmal Atrial Fibrillation
A Prospective Randomized Study

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Background—There are no reports describing the technique, electrophysiological evaluation, and clinical consequences of complete linear block at roofline joining the superior pulmonary veins (PVs) in patients with paroxysmal atrial fibrillation (AF).

Methods and Results—Ninety patients with drug-refractory paroxysmal AF undergoing radiofrequency ablation were prospectively randomized into 2 ablation strategies: (1) PV isolation (n=45) or (2) PV isolation in combination with linear ablation joining the 2 superior PVs (roofline; n=45). In both groups, the cavotricuspid isthmus, fragmented peri-PV-ostial electrograms, and spontaneous non-PV foci were ablated. Roofline ablation was performed at the most cranial part of the left atrium (LA) with complete conduction block demonstrated during LA appendage pacing by the online mapping of continuous double potential and an activation detour propagating around the PVs to activate caudocranially the posterior wall of the LA. The effect of ablation at the LA roof was evaluated by the change in fibrillatory cycle length, termination and noninducibility of AF, and clinical outcome. PV isolation was achieved in all patients with no significant differences in the radiofrequency duration, fluoroscopy, or procedural time between the groups. Roofline ablation required 12±6 (median 11, range 3 to 25) minutes of radiofrequency energy delivery with a fluoroscopic duration of 7±2 minutes and was performed in 19±7 minutes. Complete block was confirmed in 43 patients (96%) and resulted in an activation delay that was shorter circumventing the left than the right PVs during LA appendage pacing (138±15 versus 146±25 ms, respectively; P=0.01). Roofline ablation resulted in a significant increase in the fibrillatory cycle length (198±38 to 217±44 ms; P=0.0005), termination of arrhythmia in 47% (8/17), and subsequent noninducibility of AF in 59% (10/17) of the patients inducible after PV isolation. However, LA flutter, predominantly perimitral, could be induced in 10 patients (22%) after roofline ablation. At 15±4 months, 87% of the roofline group and 69% with PV isolation alone are arrhythmia free without antiarrhythmics (P=0.04).

Conclusions—This prospective randomized study demonstrates the feasibility of achieving complete linear block at the LA roof. Such ablation resulted in the prolongation of the fibrillatory cycle, termination of AF, and subsequent noninducibility and is associated with an improved clinical outcome compared with PV isolation alone. (Circulation. 2005;112:3688-3696.)

Key Words: ablation ■ arrhythmia ■ atrium ■ fibrillation ■ lesion

Catheter-induced ablation of paroxysmal atrial fibrillation (AF) is centered on isolation of the pulmonary veins (PVs), which results in a success of between 56% and 85%.1–3 To improve the clinical outcome, additional linear ablation to modify the substrate for AF has been proposed.4–10 However, this has been technically difficult, associated with altered atrial conduction and with incomplete or recovered gaps being proarrhythmic. Although linear ablation at the roof of the left atrium (LA) joining the 2 superior veins (roofline) is incorporated in most lesion sets, no reports have described the technique and consequences of achieving complete linear block. This prospective study evaluates the feasibility of performing roofline ablation, electrophysiological consequences of complete block, and outcome of roofline ablation in combination with pulmonary vein isolation (PVI) in patients with paroxysmal AF.
of any atrial arrhythmia (clinical and asymptomatic) without the use was performed. The primary end point of the study was the absence initiating AF during the index procedure, mapping guided ablation immediately before the procedure, and, in all cases, this was ceased during periods. Fifteen patients were being treated with amiodarone immediately before the procedure, and, in all cases, this was ceased during the postabsorptive state under light sedation. All antiarrhythmics, with the exception of amiodarone, had been discontinued having failed 3.6 ± 1.3 antiarrhythmic drugs, including amiodarone in 15 patients. Twenty-five patients had structural heart disease. Baseline characteristics of these patients are presented in the Table. All patients provided written informed consent for the study protocol.

### Methods

#### Study Population

The study population consisted of 90 patients referred for ablation of paroxysmal AF at a single institution over a 12-month period from January 2003 (71 men; mean age, 55 ± 9 years). These patients had symptomatic AF for 63 ± 54 months and presented for ablation after having failed 3.6 ± 1.3 antiarrhythmic drugs, including amiodarone in 15 patients. Twenty-five patients had structural heart disease. Baseline characteristics of these patients are presented in the Table. All patients provided written informed consent for the study protocol.

#### Electrophysiological Study

Electrophysiological study was performed with patients in the postabsorptive state under light sedation. All antiarrhythmics, with the exception of amiodarone, had been discontinued having failed 3.6 ± 1.3 antiarrhythmic drugs, including amiodarone in 15 patients. Twenty-five patients had structural heart disease. Baseline characteristics of these patients are presented in the Table. All patients provided written informed consent for the study protocol.

#### Pulmonary Vein Isolation

The techniques used for PVI have been previously described. In brief, the following catheters were introduced via the right femoral vein for electrophysiological study: (1) A steerable quadripolar catheter (Xirem; Ela Medical) was positioned in the coronary sinus (CS); (2) a circumferential mapping catheter (Lasso; Biosense-Webster) was introduced after transseptal access with the use of a long sheath (Preface multipurpose, Biosense-Webster) that was continuously perfused with heparinized glucose; and (3) a 4-mm irrigated-tip ablation catheter (Celsius Thermocool, Biosense-Webster) was used. Ablation was performed at 1 cm from the ostium of both right PVs as well as the posterior and superior aspects of the left PVs to minimize the risk of PV stenosis and continued circumferentially by gravitating around the PV during ongoing AF to achieve electrical isolation of the PVs. When ablation was required at the posterior portions of the left PVs, energy had to be delivered within the first millimeters of the vein (rather than the posterior wall of appendage) to achieve effective disconnection (Figure 1). Radiofrequency (RF) energy was delivered for 30 to 60 seconds at each point and was prolonged for 1 to 2 minutes when a change occurred in activation/ morphology of the PV potentials. PVI was defined by complete elimination or dissociation of PV potentials determined with the Lasso catheter. After PVI, a careful search was performed of the entire circumferential perimeter of the PV, and ablation was performed at sites demonstrating residual fragmentation (≥ 2 positive or negative deflections), long-duration (≥ 50 ms) potentials. In addition, spontaneously occurring (without isoproterenol or pacing) triggers of AF or isolated premature beats were mapped and ablated.

RF energy was delivered with power of 30 to 35 W with irradiation rates of 5 to 20 mL/min to achieve the desired power delivery. When RF energy was needed to infringe the left veins (at their anterior aspect), the power was reduced to 25 W. Temperature was limited to 50 °C.

#### Roofline Ablation: Techniques and Evaluation

Ablation of the LA roof was performed by creation of a contiguous line of ablation lesions joining the superior PVs. RF ablation was performed in all cases with the use of the externally irrigated ablation catheter in a temperature-controlled mode with temperature limited to 50 °C. When the catheter was positioned perpendicular to the tissue interface, a delivered power of 30 W was used, and when parallel, a delivered power of 30 to 35 W was used. Irradiation rates varied from 20 to 50 mL/min to deliver the desired power.

To perform this ablation, the ablation catheter was introduced through a long sheath to achieve stability and allow orientation of the catheter tip toward the LA roof. Commencing at the incising lesion at the left superior PV, the sheath and catheter assembly were rotated clockwise posteriorly and dragged toward the right superior PV (Figure 2). To avoid the risk of atrioesophageal fistula, as recently described, ablation was performed cranially at the LA roof. To achieve stability along the cranial LA roof, the catheter may be directed toward the left superior PV and the sheath rotated to face the right PVs or vice versa. Two alternative methods could also be used to reach the LA roof for ablation: (1) The catheter can be looped around the lateral, inferior, septal, and then cranial walls, laying the catheter down along the cranial wall of the LA to allow dragging of the catheter by withdrawal from the left to the right superior PV ostia; or (2) the catheter can be maximally deflected to form a tight loop near the left superior PV with the tip facing the right PVs. Releasing the curve positions the catheter tip adjacent to the right superior PV ostia and allows dragging back to the left PV. Regardless of the technique utilized, ablation was preferably performed cranially rather than posteriorly. RF energy was delivered for 60 to 120 seconds at each point while the online potentials were monitored. Local potential elimination or formation of double potentials during pacing or AF signifies effectiveness of ablation locally. The stability of the catheter was monitored during RF applications with the use of the proximal electrogrograms, intermittent fluoroscopy, and/or a navigation system to recognize inadvertent displacement of the catheter. Electroanatomic mapping (CARTO, Biosense Webster) or NavX navigation (Endocardial Solutions Inc) was used for real-time monitoring and to tag the ablation sequence.

### Table: Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>PVI (n=45)</th>
<th>PVI + Roofline (n=45)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>55 ± 8</td>
<td>54 ± 10</td>
<td>0.5</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>34 (76)</td>
<td>37 (82)</td>
<td>0.4</td>
</tr>
<tr>
<td>Duration of AF, mo</td>
<td>56 ± 44</td>
<td>70 ± 61</td>
<td>0.4</td>
</tr>
<tr>
<td>Antiarrhythmic drugs, n</td>
<td>3.3 ± 1.0</td>
<td>3.8 ± 1.5</td>
<td>0.2</td>
</tr>
<tr>
<td>Structural heart disease, n (%)</td>
<td>15 (33)</td>
<td>10 (22)</td>
<td>0.2</td>
</tr>
<tr>
<td>LV end-diastolic diameter, mm</td>
<td>51 ± 5</td>
<td>50 ± 9</td>
<td>0.8</td>
</tr>
<tr>
<td>LV end-systolic diameter, mm</td>
<td>31 ± 6</td>
<td>32 ± 5</td>
<td>0.8</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>67 ± 11</td>
<td>67 ± 8</td>
<td>0.96</td>
</tr>
<tr>
<td>LA size, mm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parasternal</td>
<td>41 ± 6</td>
<td>41 ± 6</td>
<td>0.8</td>
</tr>
<tr>
<td>Longitudinal</td>
<td>54 ± 8</td>
<td>51 ± 8</td>
<td>0.4</td>
</tr>
<tr>
<td>Transverse</td>
<td>38 ± 6</td>
<td>37 ± 7</td>
<td>0.4</td>
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ration of sinus rhythm to allow pacing of the anterior LA adjacent to the line. Anterior LA pacing could be achieved by pacing the LA appendage or the distal CS to capture the anterior wall by advancing the catheter to the anterior aspect of the CS. Complete linear block was defined by the following criteria: (1) Demonstration by point-by-point mapping of an online corridor of double potentials along the entire length of the roof during pacing of the anterior LA (Figure 3); and (2) demonstration of an activation detour circumventing the right and left PVs to activate caudocranially the posterior wall with no conduction through the LA roof (Figure 4); the latter was demonstrated by point-by-point sequential mapping either conventionally or by electroanatomic mapping. In some cases, it was difficult to record both double potentials along the ablation line; thus, the second potential was measured at the posterior LA close to the ablation line (Figure 1). When residual conduction was demonstrated, detailed online mapping was performed to identify and ablate gaps in the linear lesion.

Monitoring the Effect of Ablation

In all patients the inducibility of AF was evaluated before and after PVI and, in those randomized to linear ablation, after the roofline. Inducibility was performed with the use of a previously described standardized protocol. In brief, atrial burst pacing was performed (10-second bursts) in decrements from a cycle length (CL) of 250 mA down to refractoriness at maximum output (20 mA) from the distal CS and the right and LA appendage. Inducible AF was defined as sustained AF for ≥10 minutes. In the event that AF terminated in <10 minutes, induction was attempted 3 times from each of these sites. If a regular arrhythmia was induced, activation and entrainment mapping was performed to characterize the arrhythmia, and no attempt was made to ablate these arrhythmias. AF was defined by the beat-to-beat variability in CL and morphology, and atrial flutter was defined as a rapid regular atrial rhythm with stable CL, morphology, and activation sequence.

The effect of ablation at each site was evaluated by determining the fibrillatory CL within the CS by averaging the interval of 30 consecutive cycles before and after ablation of each PV and after the roofline with the use of automated CL monitoring software (Bard Electrophysiology). Interelectrogram intervals of <100 ms and continuous electric activity were counted as a single interval. At each time point, the automated annotation was manually verified and corrected with the use of online calipers at a paper speed of 100 mm/s. To avoid transitional CLs, these parameters were determined at least 10 minutes after the onset of AF and 10 cycles before the termination of AF. Termination of AF was defined as previously described, as direct transition to sinus rhythm or conversion to atrial tachycardia. No additional ablation other than that specified above was performed even in the presence of inducible arrhythmia during the index procedure.

Follow-up

All patients were monitored in the hospital for at least 5 days after their procedure. After ablation, all antiarrhythmic drugs were discontinued in the absence of concurrent indications. Patients were periodically reevaluated at 1, 3, 6, and 12 months, after which, in the absence of AF or symptoms, the referring physician provided the follow-up data. At each review, exercise testing and ambulatory 48-hour monitoring were performed to evaluate the presence of asymptomatic arrhythmias. In the event of recurrent symptomatic or asymptomatic arrhythmia, patients were offered an additional ablation after a trial of drug therapy. At repeated procedures, the prior ablation lesions were evaluated and consolidated if required. If
additional linear ablation was required (in addition to the randomized protocol), these patients were considered ablation failures for the analysis. All patients underwent CT angiography to evaluate the presence of PV stenosis at 12 months. Cessation of anticoagulant therapy was considered after 3 months if patients remained in sinus rhythm without antiarrhythmic drugs.

Statistical Analysis
All variables are reported as mean±SD. Comparison between groups was performed with either the Student t test or, when data were not normally distributed, the Wilcoxon rank sum or signed rank test. Proportions were compared with the χ² test. A Kaplan-Meier analysis with log-rank test was used to determine the probability of freedom from recurrent AF. Statistical significance was established at P<0.05.

Results
Patients were prospectively randomized into 2 groups: Half were assigned PVI alone, and the other half were assigned roofline ablation in addition to PVI. There were no differences in baseline characteristics between these groups (Table).

PVI was achieved in all patients with no differences in the RF duration (33±11 versus 35±9 minutes; P=0.1), fluoroscopy (15±7 versus 19±10 minutes; P=0.3), and procedural time (56±20 versus 59±23 minutes; P=0.6) to perform ablation in patients randomized to roofline ablation compared with those with PVI alone, respectively. Bidirectional cavotricuspid isthmus block was performed with no differences in the RF duration in patients randomized to roofline ablation compared with those with PVI alone (8±6 versus 7±4 minutes; P=0.5). In addition, non-PV triggers of AF were also ablated in 5 patients (2 in those undergoing roof ablation and 3 in those with PVI alone) with a mean of 7±2 minutes of RF energy. These were localized to the superior vena cava, low anterior right atrium, anterior LA, inferior LA, and left septum.
Electrophysiological Validation of Complete Block at the LA Roof

Ablation of the LA roof was performed with a median of 11 minutes (range, 3 to 25 minutes) of RF energy delivery (median with a fluoroscopic duration of 7±2 minutes and performed in 19±7 minutes). The length of the ablation lesions at the LA roof was 46±11 mm as determined by 3D mapping, and complete block could be confirmed with the use of the predefined criteria in 43 patients (96%).

Activation mapping in the presence of conduction block during LA appendage pacing demonstrated more rapid propagation of the activation front around the left PVs than around the septum, resulting in a shorter conduction time at the left than the right extremity of the line. This resulted in activation delay shorter to the second potential at the left PV (138±15 ms) compared with the right PV (146±25 ms; P=0.01). This activation delay corresponded to the detour time required to activate the posterior flank of the line. Figure 4A demonstrates the activation map after roofline ablation during LA appendage pacing, showing the detour of activation around the left and right PVs to activate caudocranially the posterior LA with fusion of the activation front in the mid to septal posterior wall. The propagation map and activation schematic of the same patient are shown in Figure 4B, demonstrating early activation of the posterior LA on the left compared with the right, giving rise to the difference in delay along the line near the right and left PVs, respectively.

Figure 4. A, Electroanatomic activation map during LA appendage pacing to demonstrate the activation detour to the posterior LA wall. The PVs are marked in this case with orange tags, and the ablation line is seen as areas of electric silence marked in gray as scar. Abbreviations are as defined in Figure 1 legend. B, Propagation map of the same patient. The wave front commences at the LA appendage (LAA) and propagates rapidly around the left PV and later around the right PVs to collide in the posterior LA near the septum.

Effect of Ablation on CL, AF Termination, and Inducibility

Spontaneous or induced sustained AF was present in 64 patients (71%), and in the remainder AF lasted <10 minutes. With PVI, a progressive increase in fibrillatory CL was observed within the CS, increasing from 183±30 to 208±41 ms (all patients, P<0.0001), culminating in the termination of AF in 45 patients (70%), directly to sinus rhythm in 43 and to cavotricuspid isthmus–dependent flutter in 2. After PVI, arrhythmia was persistent or could be induced in 31 (48%).

In the 45 patients randomized to roofline ablation, 17 patients (38%) had persistent or inducible sustained arrhythmia after PVI: 14 with AF and 3 with LA flutter. Activation and entrainment mapping in these latter patients demonstrated macroreentry around the right PVs. In patients with sustained AF, roofline ablation resulted in a significant increase in the fibrillatory CL within the CS, increasing from 198±38 to 217±44 ms (roofline group only; P=0.0005). In
5 of these 14 patients (36%), AF terminated: 4 to sinus rhythm and 1 to cavotricuspid isthmus–dependent flutter, with no inducible AF in 4 after roofline ablation (Figure 5). In all but 2 patients in whom AF terminated during roofline ablation, further ablation was performed during LA appendage pacing to achieve complete block. In all 3 patients with LA flutter rotating around the right PVs after PVI, ablation of the roofline terminated flutter. The remaining 9 patients required electric cardioversion to restore sinus rhythm. After completion of ablation at the LA roof in sinus rhythm, 5 of these 9 patients could no longer sustain AF.

**LA Flutters During Ablation of AF**

After ablation of the PVs and before linear ablation, LA flutter was induced in 4 patients, with mapping demonstrating macroreentry around the right PVs in 3 (Figure 6) and perimital in 1; the latter was not ablated as predefined by the protocol. The 3 patients with macroreentry around the right PVs had been randomized at the commencement of the procedure to roofline ablation, which resulted in arrhythmia termination in all.

After roofline ablation in the subset randomized to this arm of the study, LA flutter could be induced by burst atrial pacing in 10 patients (22%). None of these patients had such arrhythmia observed before. Activation and entrainment mapping demonstrated perimital macroreentry in 9 patients and reentry localized to the anterior LA wall in 1 patient. No attempt was made to ablate these induced flutters, and they were pace terminated.

During the follow-up period, 3 of these 10 patients developed spontaneous clinical flutter. Mapping was performed in all 3, demonstrating perimital reentry, which was ablated by linear ablation joining the lateral mitral annulus to the left inferior PV as previously described. It is noteworthy that, after ablation of the mitral isthmus, the confirmation of roofline conduction block demonstrated a variation of the aforementioned description (Figure 7). In the presence of complete block at the mitral isthmus, pacing from the LA appendage resulted in conduction block around the left PVs with the posterior LA being activated by wave fronts that wrap around the septum and right PVs and inferiorly around the mitral annulus. Thus, the activation delay determined on the line is reversed, being shorter at the right PV than the left but with caudocranial maintained in the posterior LA.

**Clinical Outcome**

At 15±4 months (14±5 and 15±4 months in the roofline and PV groups, respectively), 39 patients (87%) who had roofline ablation in addition to PVI and 31 patients (69%) with PVI.
alone remained free of all arrhythmia (flutter or AF) without the use of antiarrhythmic drugs (Figure 8; \( P = 0.04 \)). All other patients were being treated with an antiarrhythmic agent.

**Complications**

The procedure was complicated in 1 patient by pericardial tamponade during cavotricuspid isthmus ablation. It was preceded by an audible pop associated with a power of 38 W. Percutaneous drainage was performed with no long-term sequelae. One patient developed right phrenic nerve injury during ablation of the right superior PV, with complete recovery 4 months after the procedure. During routine follow-up, 1 asymptomatic patient was observed to have stenosis of the left superior PV (70%) by CT angiography at 12 months.

**Discussion**

This prospective randomized study provides new information on the feasibility of performing roofline ablation, electrophysiological evaluation of complete conduction block, and clinical consequences of LA roof ablation in addition to PVI.

First, this study demonstrates the feasibility of performing roofline ablation. With a mean of 12\(\pm\)6 minutes of RF energy, complete block at the LA roof could be achieved in 96%, demonstrated by a continuous line of double potentials and an activation detour associated with a delay to the second potential of 138\(\pm\)15 and 146\(\pm\)25 ms at the left and right extremes of the line, respectively, during anterior LA pacing.

Second, ablation at the LA roof had a direct effect on the AF process, prolonging the fibrillatory CL and terminating arrhythmia in 47%. In addition, 59% of patients with inducible or sustained arrhythmia after PVI were rendered noninducible after roofline ablation, implicating the LA roof in the substrate maintaining AF after PVI in humans.

Finally, in this randomized comparison, additional roofline ablation with complete conduction block was associated with 87% of patients being arrhythmia free without antiarrhythmics compared with 69% undergoing PVI alone.

**LA Linear Ablation to Modify the Substrate for AF**

Studies on the extensive biatrial linear ablation to compartmentalize the atria and prevent multiple wavelet reentry have demonstrated the difficulty of creating complete linear lesions, which have been associated with prolonged procedural and fluoroscopic durations, development of atypical flutters, and significant procedural complications.4,5,14–16 With the recognition of the PVs as a dominant source of triggers initiating AF, most ablation strategies have focused on disconnection of the PVs at the LA-PV junction, but to improve the outcome of ablation, additional substrate modification with the use of limited linear ablation may be necessary.6,9,10,17 A common theme that has evolved in this experience has been the importance of complete linear lesions to prevent the resultant proarhythmic risk.

To achieve complete conduction block, left linear ablation needs to be anchored on both ends to anatomic barriers available within the LA. Transection of the anterior LA results in significantly delayed activation of the lateral LA during sinus rhythm, which has potentially deleterious hemodynamic consequences.10 Ablation connecting the PV to the mitral annulus allows favorable catheter positioning to evaluate and confirm bidirectional conduction block. However, although short in length, it has required long RF energy.
delivery and ablation distally within the CS. Different groups have performed ablation posteriorly between the 2 superior PVs using high power (up to 100 W) but without confirmation of conduction block resulting in better clinical outcome than PVI alone. However, a significant incidence of LA flutter has been recognized due to persistent gaps.

In the present study, ablation joining the 2 superior PVs was performed with power limited to 30 to 35 W. This line suspended between the 2 PVs resulted in a conduction detour to the posterior wall, with caudocranial activation during anterior LA pacing, and with an activation delay of 138±15 and 146±25 ms at the left and right extremes of the line, respectively, when complete block was achieved. Complete block at this site was achieved in a similar percentage of patients (96% for the roofline and 92% for the mitral isthmus line) and resulted in the same clinical outcome (87%) as mitral isthmus ablation but was achieved with much shorter RF application (12±6 versus 24±14 minutes, respectively). This difference in the duration of RF required is possibly due to the differences in thickness of the atrial myocardium at these regions.

Interestingly, whereas previous studies on linear ablation have highlighted the proarrhythmic effect of incomplete and recovered gaps, the present study shows that ablation of the roof with complete block produced a detour of conduction that favored perimital macroreentry. However, only 3 of these 9 patients with inducible perimital macroreentry developed this clinical arrhythmia during 15±4 months of follow-up.

Implications for the Substrate Maintaining AF

The PVs are a dominant source of triggers initiating AF and recently have been implicated in the substrate maintaining paroxysmal AF. Several investigators have observed paroxysmal short CL activity within the PV during AF with a distal-to-proximal activation sequence and have suggested that this may represent a continual feeding of the fibrillatory process by the PVs. PVI in patients with paroxysmal AF results in a slowing of the fibrillatory process, culminating in its termination in 75% of patients, providing direct evidence for the role of the PVs in the perpetuation of AF. However, the remaining patients could sustain AF, suggesting residual substrate capable of maintaining AF after exclusion of the PV and the PV-LA junction.

Several potential mechanisms could coexist to form the substrate for AF after PVI; these include localized high-frequency activity (focal or reentrant), meandering multiple wavelet reentry, and macroreentry. Markides et al observed a line of conduction block associated with abrupt change in subendocardial muscle fiber orientation that extended from the LA roof along the posterior wall between the ostia of the PVs. Betts et al demonstrated that as a result of this line of functional or fixed conduction block, activation in sinus rhythm resulted in depolarization spreading rapidly across the LA roof, turning around the right PVs to activate the posterior LA. Furthermore, areas of conduction abnormalities were observed inferiorly down the interatrial septum. In concert, these studies implicate the potential for supporting macroreentry around the PVs utilizing the LA roof. Such a mechanism was observed in our cohort during follow-up, with 3 patients developing macroreentry around the right PV after ablation of the PVs.

Nademanee et al also reported that the LA roof represents a region demonstrating highly fragmented electrograms, perhaps indicating the presence of substrate capable of sustaining localized reentry or focal activity that may maintain fibrillation. Fibrillatory sources with epochs of centrifugal activation have been described in the LA roof during organized AF after prior ablation. In the present study, ablation along the LA roof resulted in a slowing of the AF process and termination of AF in 47% of patients undergoing roofline ablation during AF. Although we are unable to determine the exact mechanism by which the LA roof supports the fibrillatory process, our data clearly implicate this region in the substrate for AF.

Clinical Implications

Ablation of the LA roof in addition to PVI provides a clinical benefit similar to other described lines but can be achieved with shorter RF application and procedural durations. The present randomized study demonstrates that this lesion set with complete block significantly results in improved clinical outcome compared with PVI alone in patients with paroxysmal AF.

Limitations

Although this study demonstrates greater efficacy for the suppression of atrial arrhythmia with the addition of a complete line of block at the roof compared with PVI alone, this required additional ablation and may be associated with a proarrhythmic risk. Whether such linear ablation should be empirically performed in all patients with paroxysmal AF or applied selectively on the basis of clinical or procedural variables, notably persistent inducibility after PVI, was not evaluated in the present study and remains to be determined prospectively. Finally, this study did not evaluate the efficacy or usefulness of incomplete linear ablation at the LA roof.

Conclusion

The present study demonstrates that complete linear ablation at the LA roof can be achieved safely and can be electrophysiologically evaluated for completeness. Ablation at the LA roof after PVI was associated with slowing, termination and the subsequent noninducibility of AF, and better clinical outcome, implicating this region in the substrate maintaining AF in some patients.

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Disclosure

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received lecture fees from Endocardial Solutions. The other authors report no conflicts.

References


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