

Electrophysiological Breakthroughs From the Left Atrium to the Pulmonary Veins

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Background—The extent of ostial ablation necessary to electrically disconnect the pulmonary vein (PV) myocardial extensions that initiate atrial fibrillation from the left atrium has not been determined.

Methods and Results—Seventy patients underwent PV mapping with a circumferential 10-electrode catheter during sinus rhythm or left atrial pacing. After assessment of perimetric distribution and activation sequence of PV potentials, ostial ablation was performed at segments showing earliest activation, with the end point of PV disconnection. A total of 162 PVs (excluding right inferior PVs) were ablated. PV potentials were present at 60% to 88% of their perimeter, but PV muscle activation was always sequential from a segment with earliest activation (breakthrough). Radiofrequency (RF) application at this breakthrough eliminated all PV potentials in 34 PVs, whereas a secondary breakthrough required RF applications at separate segments in 77; in others, >2 segments were ablated. A median of 5, 6, and 4 bipoles from the circular catheter were targeted in the right superior, left superior, and inferior PVs, respectively, to achieve PV disconnection. Early recurrence of arrhythmia was observed in 31 patients as a result of new venous or atrial foci or recovery of previously targeted PVs, most related to a single recovered breakthrough that was reablated with local RF application.

Conclusions—Although PV muscle covers a large extent of the PV perimeter, there are specific breakthroughs from the left atrium that allow ostial PV disconnection by use of partial perimetric ablation. (*Circulation*. 2000;102:2463–2465.)

Key Words: fibrillation ■ veins ■ lung ■ ablation

The pulmonary veins (PVs) have been shown to trigger paroxysms of atrial fibrillation (AF). Radiofrequency (RF) energy ablation can be delivered distally into the PV, or preferably at the ostium, with the end point of distal PV disconnection.^{1–3} Because the topography and activation of myocardial extensions to the PV are unknown, the extent of ostial ablation necessary to disconnect the distal PV muscle has not been determined. The present study was prospectively performed to assess this issue.

Methods

Patient Characteristics

The study population consisted of 70 consecutive patients (13 female, 53±13 years old) with multidrug-resistant paroxysmal AF.

Electrophysiological Study

The study was performed as described previously.³ The left atrium and PVs were explored through either a patent foramen ovale (16 patients) or transseptal catheterization with 2 catheters: 1 for circumferential PV mapping, and a quadripolar mapping/ablation catheter. Selective PV angiography was performed by hand injection of 5 to 10 mL of contrast medium and was displayed during the procedure. Heparin was titrated to maintain a partial thromboplastin time of 60 to 90 seconds (control=30 seconds).

An arrhythmogenic PV was defined on the basis of documented ectopy: single or multiple, isolated or initiating AF, and with or without

conduction to the left atrium, observed spontaneously or after provocative maneuvers.

Perimetric Distribution and Activation of PV Muscle

PV mapping was performed with a steerable circular catheter 15 or 20 mm in diameter (choice based on PV angiography) equipped with ten 1-mm electrodes in a loop made of shape-retaining material (Lasso, Biosense Webster) orthogonal to the shaft. It was uncoiled to allow introduction into the 8F transseptal sheath and deployed into the body of the left atrium (after resuming its shape), then pushed, like any steerable catheter, into the desired PV, easily into both superior PVs but requiring some manipulation into the left inferior PV. Sequential recordings of transverse slices of PV activity were performed at 5, 10, and 15 mm from the angiographically defined left atrial–PV junction. PV muscle potentials (PVPs) were defined as described previously and recorded in bipolar mode at 10 bipoles (1 to 2, 2 to 3, . . . , up to 10 to 1) with bandpass filters of 30 to 500 Hz and amplification of 1 to 2 cm/mV with a polygraph (Midas PPG or Labsystem Bard).

The number of bipoles showing local PVP deflection defined the extent of PV perimeter covered by myocardial extensions (Figures 1 and 2). Activation was assessed in the proximal PV (where ablation was performed) on the basis of the timing of the maximum peak of electrograms at the 10 bipoles from the Lasso. The circumferential conduction time was calculated from the earliest to latest PVPs.

Ablation Procedure

RF ablation of arrhythmogenic PVs was performed as proximally as possible, the exact location depending on catheter stability. Segments

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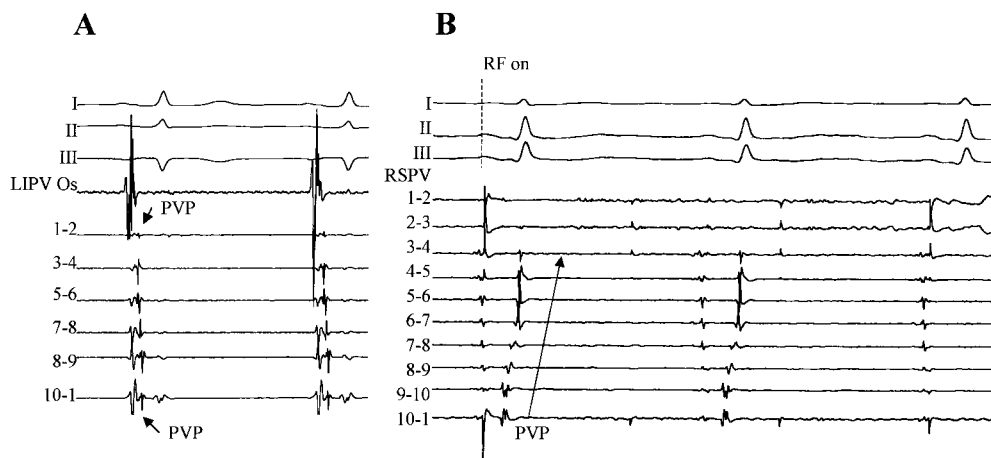


Figure 1. A, Sequential activation in a cascade from site of earliest breakthrough (bipole 1 to 2) at top of left inferior PV (LIPV). PVP present in all bipoles indicates circumferential muscle coverage. Local ablation at LIPV ostium corresponding to bipole 1 to 2 eliminated all PVPs. B, PV mapping for recurrent AF: focal ablation at right superior PV (RSPV) bottom eliminated all sequentially activated PVPs. Note noise at 4 contiguous bipoles 10 to 4 due to nearby RF delivery.

of the PV perimeter were targeted on the basis of the bipole(s) from the circular catheter showing the earliest activation during sinus rhythm or pacing of the distal coronary sinus (or left atrial appendage). The ablation catheter was positioned correspondingly on fluoroscopy, and the local largest-amplitude PVP was first targeted for ablation. Subsequent RF applications were performed if needed at contiguous sites showing synchronous PVP. If PV activation changed as a result of RF ablation, the ostial sector now showing the earliest PVP was targeted. The end point was elimination of PV muscle conduction distal to the ablation site(s) based on either abolition or dissociation of distal PVPs and elimination of ectopic beats, spontaneous or induced by provocative maneuvers (isoproterenol and burst pacing). RF ablation of atrial foci, if present, was performed at the site of earliest activation.

RF energy was delivered at the distal electrode (Celsius, Biosense Webster), of the thermocouple-equipped catheter (target: 50°C) with a power limit of 25 to 30 W for 30 to 60 seconds at each site. If this power could not be reached (presumably because of reduced local blood flow), an irrigated-tip catheter (17 mL/min saline flow) was used with the same target temperature and power. Arrhythmogenic PVs were sequentially

ablated, provided that the PV diameter was unchanged on angiography. PV angiography was repeated after 20 minutes of surveillance, with PV "stenosis" defined as a diameter reduction of >50% and a CT scan performed >3 months later in 36 patients to exclude late PV compromise.³⁻⁵

Patients were discharged after day 3 under oral anticoagulant. Success was defined as elimination of AF without antiarrhythmic drug. Anticoagulants were interrupted 3 months after successful elimination of AF, unless there were other risk factors.

Statistical Analysis

Continuous variables are expressed as group mean value \pm SD or median value (nongaussian distribution). Statistical significance was selected at a value of $P < 0.05$ with a Kruskal-Wallis or χ^2 test.

Results

A total of 162 PVs were mapped, including 61 left superior, 55 right superior, and 46 left inferior. Eleven arrhythmogenic right inferior PVs could not be mapped but were ablated anatomically. Procedure duration and fluoroscopy times were 206 ± 49 and 65 ± 18 minutes, respectively.

Perimetric Distribution

At the atrial margin of the ostia, PVPs were present circumferentially (all bipoles displayed local PVPs), whereas inside the PVs, PVPs covered only various parts of the perimeter (from 3 to 10 bipoles), with a consistent reduction from proximal to distal (Table). The percentage of perimetric PV muscle coverage was higher ($P < 0.05$) for both superior PVs than for the left inferior PV.

Activation of PV Muscle in the Proximal PV

Muscle activation was never circumferentially synchronous in the PVs, during either sinus rhythm or atrial pacing, indicating preferential breakthrough(s) into the vein. The earliest PVPs were localized to a segment of the perimeter (median of 3 contiguous bipoles, range 1 to 4), whereas the remaining perimeter was activated sequentially later. The circumferential conduction time was 33 ± 15 ms (range 10 to 85 ms) during sinus rhythm.

RF Ablation

RF delivery was begun at the earliest activated ostial segment, with the circumferential catheter in a distal monitoring position

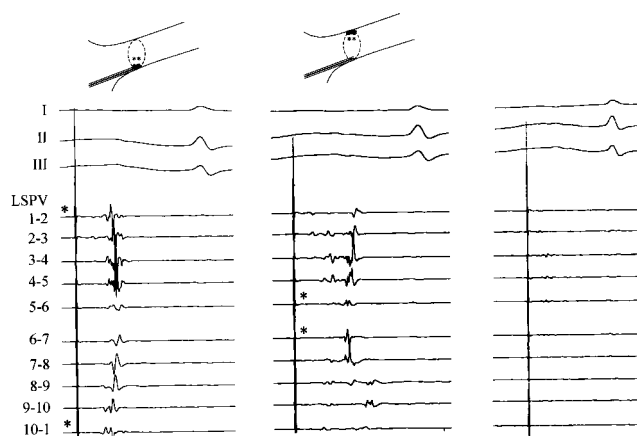


Figure 2. Ostial ablation of left superior PV (LSPV) performed during distal coronary sinus pacing to highlight PVPs. Left, RF applications at 1 segment (affecting bipoles 1 to 2 and 10 to 1) changed activation pattern by eliminating breakthrough at this site, as indicated by 90-ms prolongation of local conduction, and revealed a secondary breakthrough (center). Further delivery at this site (*) of secondary breakthrough abruptly abolished all PVPs (right).

Mapping and Ablation Data

	RSPV (n=55)	LSPV (n=61)	LIPV (n=46)
PV muscle coverage (n=162), %			
At 5 mm	84±15	88±16	60±19
At 10 mm	64±22	67±20	25±28
At 15 mm	45±30	58±31	
Ablation data in successfully ablated PV (n=157)	(n=53)	(n=59)	(n=45)
Extent of perimetric ablation			
1 segment, n=34	8	7	19
2 segments, n=77	28	30	19
>2 segments, n=46	17	22	7
Increment in PV conduction time before block, ms	42±29	42±26	34±30
RF delivery duration, min	8±3	9±3	6±3
Requirement for irrigated-tip catheter	6	4	1

RS indicates right superior; LS, left superior; and LI, left inferior.

(Figure 1A). Ablation restricted to this segment totally eliminated PVPs in 34 PVs. In others, RF delivery eliminated local PVPs only or segmentally delayed them by 38 ± 30 ms, resulting in a change in activation sequence (Figure 2). This secondary breakthrough was usually located at the opposite segment of the circumference. Additional RF delivery eliminated all distal PVPs in 77 PVs, whereas RF applications to other parts of the PV perimeter (including to the full circumference) were delivered in the remaining PVs (Table). The final successful RF application always corresponded to the bipole showing the earliest PVP, with centrifugal activation to other sites. Thus, in 45% of targeted PVs, all PVPs disappeared abruptly at the same time ("all or none" phenomenon), whereas they were abolished in ≥ 2 steps in 55%.

PV disconnection was achieved in all but 5 PVs (97% of targeted PVs) with a mean RF duration of 8 ± 3 minutes per PV (Table). A median of 4, 5, and 6 bipoles was targeted in left inferior, right superior, and left superior PVs, respectively. A single breakthrough (and ablated segment) was associated with a similar PVP sequence during both sinus rhythm and pacing in 95% of cases, whereas multiple breakthroughs had a similar versus dissimilar sequence in 35% and 65% of cases.

Two patients had a pericardial effusion and 2 a femoral aneurysm, but no PV stenoses were observed.

Reablation and Final Outcome

Thirty-one patients (44%) had recurrence of AF, and a reablation session was performed in 29. The ectopy was related to a previously ablated PV in 18 patients (27 PVs), with recovery of all distal PVPs. A single breakthrough had recovered in 21 PVs at the same ostial site as in the index procedure and was ablated by RF delivery limited to this site (Figure 1B). Other ectopic beats or AF initiations were mapped to multiple sources, including previously untargeted PVs in 11 (9 right inferior PVs), the PV ostia proximal to previous ablation in 9, and the atrial tissue in 10, requiring additional RF applications. Such foci

could be difficult to localize precisely because they immediately induced sustained AF.

With a mean follow-up of 4 ± 5 months after discharge, AF was completely eliminated in 51 patients (73%) without antiarrhythmic drug. No PV stenosis was noted during follow-up.

Discussion

This study describes the results of PV ostial ablation guided by circumferential mapping data. It indicates that although PV muscle covers a large extent of PV perimeter, there are specific breakthrough(s) from the left atrium that allow ostial PV disconnection with minimal ablation.

The left atrial-PV breakthroughs were inferred from the mapping data showing sequential activation of the PV perimeter. Differing sequences during right (sinus rhythm) or left (pacing) atrial activation indicated differing breakthroughs, whereas an unchanged sequence did not necessarily indicate a single breakthrough, perhaps because of 1 input having a shorter conduction time and/or a nonoptimal pacing site. Mapping data were confirmed by results of ablation producing local elimination or prolongation of conduction and shifting of the breakthrough. The extent of perimetric ablation was thus less than the actual muscle coverage. During RF ostial ablation, monitoring of distal PVPs provided online demonstration of ablation effects, showing abrupt abolition of PVPs in 1 step in nearly half of the veins, indicating a distally interconnected PV network, whereas abolition in >1 step suggested distally separated PV fascicles. Recovery of all PV conduction after ablation was linked to a single recovered input in most, which was focally reablated.

The question of anatomic inputs (or their embryological development) to the PV has not been specifically addressed in the literature; however, a single breakthrough may be related to a myocardial band with oblique or circular course ending in a cul-de-sac, whereas either a dual band or fascicles described as "looping back in the left atrium" may be the substrate for dual-input muscle.

The findings of this study have practical implications. In addition to providing an immediate obvious end point, circumferential mapping optimizes RF ablation at the PV ostia by directing energy at specific segments and avoiding unnecessary applications at others, thus minimizing the risk of PV stenosis. However, this technique may not be applicable to RF ablation outside the PV ostia, which may require complete circumferential lesions to produce distal disconnection. Other limitations include the continued high recurrence rate of AF due to unmasked foci from the ostial edge or atrial tissue characterized by difficulty in precise mapping and absence of a similar end point.

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