Left Atrial Appendage Activity Masquerading as Pulmonary Vein Potentials

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Background—Despite extensive proximal ablation, all potentials frequently cannot be eliminated from the left pulmonary veins (PV).

Methods and Results—PV electrograms were analyzed during sinus rhythm, coronary sinus, and left atrial appendage (LAA) pacing, and PV and LAA angiography performed. During pacing, an initial low-amplitude slow potential was recorded on the anterior aspect of the left superior PV and anticipated with shortest activation time by LAA pacing. Its timing coincided with posterior LAA activation, shown to be immediately adjacent to the left superior PV by angiography. In the left inferior PV, the first potential was smaller and less sharp, coinciding with adjacent low LA activation. Angiographically, the LAA was at least 15 mm from the left inferior PV. The second sharper potential in both left PVs was eliminated by proximal ablation.

Conclusion—Far field LAA activity consistently adds to PV myocardial electrograms in the left superior PV whereas lower, less sharp extra venous potentials in the left inferior PV originate from the inferior LA. They can be identified by LAA and coronary sinus pacing. (Circulation. 2002;105:2821-2825.)

Key Words: ablation ■ fibrillation ■ pulmonary veins

Electrical disconnection of pulmonary vein (PV) myocardium from the left atrium (LA), performed for the treatment of atrial fibrillation (AF), is indicated by elimination or dissociation of all PV electrograms.1 However, elimination of all left PV potentials is often impossible even after extensive circumferential ablation and disappearance of all arrhythmias originating from the veins. This study analyzed the mechanism of extra venous electrograms recorded within the left PVs.

Methods

Thirty-five patients (29 males, 51 ± 12 years) undergoing PV ablation for paroxysmal atrial fibrillation were studied. Three had structural heart disease.

After obtaining informed consent, a 7F 4-mm tip quadripolar ablation catheter and a 15- or 20-mm diameter 7F decapolar-preformed circular catheter (Celsius and Lasso, respectively, Biosense Webster) were placed transeptally as required in the LA body, the left atrial appendage (LAA), and the PVs under cover of 50 U/kg of IV Heparin. A PV position was recognized by a catheter beyond the cardiac silhouette, lack of movement during atrial contraction, and correlation with selective venography. A LAA position was verified by a characteristic swinging and twisting movement of the catheter tip during each atrial contraction, an anterior, superior, and leftward position within the cardiac silhouette and correlation with selective angiography. A 6F quadripolar catheter was placed distally in the coronary sinus (CS) between 1 to 3 o’clock in the LAO 45° projection at the level of the LAA base, and withdrawn 2 cm proximally when required.

Selective pulmonary venography through a 7F NIH catheter allowed placement of an appropriate-sized decapolar catheter 5 to 10 mm within the PV. Proximal ablation was performed (temperature and power limit 50°C and 30W) at the earliest point of circumferential activation.1 Disconnection from the LA was indicated by elimination of spontaneous and provoked arrhythmias from the vein and elimination or dissociation of all PV potentials distal to ablation.1,2 PV potentials were defined as the sharpest potentials with a proximal to distal activation sequence during longitudinal mapping of the vein and culminating distally as a dead-end during sinus rhythm or atrial pacing. During ectopy from that vein, their activation sequence was reversed preceding all atrial and other PV activation. Bipolar electrograms (bandpass 50 to 500 Hz) were recorded on a multichannel polygraph (LabSystem Duo, Bard Electrophysiology, USA). Bipolar stimulation (cycle length 500 to 700 ms) was performed at twice diastolic threshold with a pulse width of 2 ms after verifying that activation patterns did not change at low output (near threshold).

Decapolar catheter recordings were obtained from the left superior (n=35) or both left PVs (n=20) before and after ablation during sinus rhythm, CS pacing, and LAA pacing. LA body and appendage mapping was performed during sinus rhythm and CS pacing to look for electrograms with high amplitude and slopes coinciding in timing with PV electrograms. A double potential electrogram was defined by 2 deflections separated by an isoelectric or very low voltage (<0.1 mV) interval of more than 20 ms. The maximum amplitude and slope of the major deflection of each potential, shortest

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2821
activation timing, and maximum separation were compared during pacing and sinus rhythm.

Selective opacification of the LAA was performed (with 5-mL contrast) with the decapolar catheter successively within the left superior PV (LSPV) and left inferior PV (LIPV) and recorded in at least 2 different views (anteroposterior, left anterior oblique 45°, and/or right anterior oblique 30°) to demonstrate anatomic interrelationships and the electrodes within the vein closest to the LAA.

Data are presented as mean±SD, and comparisons assessed by the Student’s t test. A 2-tailed value of P<0.05 was considered statistically significant.

Results
PVs (138 of 139) were successfully ablated in the 35 patients. There was one common left common ostium and one LIPV did not require ablation because no PV potentials were recorded.

Sinus Rhythm
Before ablation, single potentials were recorded in the LSPV in 63% (22/35) and a double potential (separated by 31±11 ms) in at least one bipolar in the remaining (Figure 1A). In the LIPV, single potentials were recorded in 70% (14/20) and a double potential separated by 29±8 ms in 6. Pacing maneuvers described in the following sections exaggerated this separation.

CS Pacing
In the LSPV, double potentials were recorded in all, composed of a low amplitude (0.4±0.3 mV), slow (slope 0.039±0.03 mV/ms) initial potential, and a higher amplitude (0.9±0.5 mV; P<0.05), sharper (0.17±0.08 mV/ms; P<0.05) later potential (Figure 1A); however, there was a large overlap in amplitude and less so in slope (Figure 2). The 2 potentials exhibited different activation patterns. The first was recorded on 4.4±1.1 bipoles (range 2 to 7) restricted to the anterior wall of the vein (or with the largest amplitude on anterior bipoles of the decapolar catheter) and activated from below upwards, whereas the second showed activation radiating circumferentially (on 8.6±1.6 bipoles) and longitudinally (from proximal to distal in the vein) from the LA PV inputs, where the 2 potentials fused into a continuum. Distal compared with proximal CS pacing activated the first potential earlier (35±7 versus 48±13 ms; P<0.05) and separated the 2 potentials more (83±30 versus 55±30 ms; P<0.05) (Figure 1A).

In the LIPV, double potentials were recorded in 80%, with either the first or second potential missing in the remaining. The first potential was small and slow (0.15±0.13 mV and 0.015±0.014 mV/ms; P<0.05 compared with LSPV). It was recorded on 5.6±1.2 bipoles, only along the anterior wall in 4 patients (Figure 1B), and variably including posteriorly, at the top and bottom in the remaining. Inferior bipoles were activated before superior ones.

The second potential was larger and sharper (0.8±0.5 mV and 0.16±0.09 mV/ms; P=NS compared with LSPV) but limited to 5±3 bipoles; its activation radiated longitudinally and circumferentially from the site of input into the vein where it fused with the first potential. Amplitudes and slopes of the 2 potentials also overlapped but less than for the LSPV (Figure 2). The activation time of the first potential (27±11 versus 21±10 ms) and separation from the second (76±28 versus 52±33 ms; P=0.11) during proximal compared with distal CS pacing respectively did not differ significantly.

LAA Pacing
Double potentials similar to CS pacing were recorded. In the LSPV, the first potential was activated from above downwards and fused within the stimulus artifact at the top of the vein or adjacent to the site of stimulation (Figure 1A), whereas the second was sharp (0.2±0.1 mV/ms) and larger (1.1±0.7 mV). The timing of the first potential was earlier (21±11 ms; P<0.05), but the separation between the 2 potentials (98±29 ms) was greater than during distal CS pacing (P<0.05).

In the LIPV, LAA pacing resulted in a longer activation timing of the first potential compared with CS pacing (47±22 ms; P<0.05) with activation in superior bipoles preceding inferior ones. The separation between the 2 potentials was not significantly less than during distal CS pacing (58±23 ms; P=0.14).

Recordings from the common ostium were like a LSPV, and the catheter when pushed distally into the respective branches recorded electrogams like the superior and inferior pulmonary veins.

LA Mapping
Only electrograms from the posterior wall of the LAA coincided with the first potential in the LSPV during sinus rhythm as well as both CS pacing sites (Figure 1A). The LAA electrograms were larger (1.6±0.6 mV; P<0.05) and sharper (0.2±0.07 mV/ms; P<0.05) than the first LSPV potential.

In the LIPV, activation of the anterior LA below the LAA or close to the bottom of the ostium of the LIPV similarly coincided with the first potential during sinus rhythm and CS pacing (Figure 1B). LA electrograms were also larger (1.5±0.5 mV; P<0.05) and sharper (0.2±0.05 mV/ms; P<0.05) than the first LIPV potential.

No site with activation coinciding with the second PV potential was found.

Effect of Ablation
The second potential was eliminated, leaving the first unchanged (Figure 1).

LAA Angiography
The LAA was at the level of the LSPV in 33/35, overlapping the circular catheter within the LSPV in the anteroposterior view. In oblique views, the LAA posterior wall was in close apposition to the anterior aspect of the LSPV, overlapping the anterior electrodes of the catheter (which recorded the first electrogram potential) or separated from them by 4±2.5 mm (Figure 1A). The appendage was below the level of this vein in the remaining 2 patients who exhibited the greatest amplitude of the first potential at the anteroinferior segment of the vein ostium.

The appendage was above the LIPV in the anteroposterior view in 90% but at least 15 mm away in all patients in the oblique views (Figure 1B); as a result, the low LA below the
Figure 1. A, LSPV electrograms (top) and LAA angiograms (bottom) with decapolar catheter in LSPV. Electrodes 3 to 8 anterior. B, LIPV electrograms (left) and LAA angiograms (right) with decapolar catheter in LSPV. Panel III shows catheter position recording far field low LA potential. Far field potentials (arrows) persist after ablation. Details in text.
appendage was closest to the superior electrodes of the decapolar catheter in the vein.

Discussion

This study shows that electrogram components from different sources can be recorded within the left PVs and their origins identified. Sinus rhythm electrograms can be separated into two potentials by pacing the LAA or the distal CS. The PV myocardial origin of the second potential is indicated by near field characteristics of higher voltage and slope, proximal to distal activation within the vein and elimination by proximal ostial ablation. However, there is significant overlap of amplitudes and slopes (although less in the LIPV than the LSPV); therefore, these parameters do not reliably distinguish between the two potentials.

The two potentials clearly differ in their distribution and activation characteristics during pacing. In the LSPV, during both CS and LAA pacing, the characteristic anterior distribution of the first potential, vertical activation sequence (unlike proximal to distal longitudinal activation of the second potential) coinciding with higher amplitude and sharper electrograms recorded from the immediately adjacent LAA (only 4 mm away by angiography), indicates that the first potential is far field activity originating from the LAA and recorded within the vein. This is also supported by the ability to capture the LAA by high-output stimulation from the LSPV.

Because the LAA and the LSPV are very close to each other, summation of synchronous activation from the two structures can produce a single potential, or in case of slow conduction in the PV, closely separated double potentials. Stimulation capturing the LAA should activate this appendage potential with a short activation time and split the electrogram into two potentials. LAA and distal CS pacing should result in shorter activation times compared with proximal CS pacing, the latter being further from the LAA. The results of this study are consistent with the above hypothesis.

Compared with the LSPV, the LAA is above and removed from the LIPV. Accordingly, low anterior LA activation (but not LAA activation) is coincidental with the first potential at the top and along the anterior wall, whereas the timing of the first potential at the bottom and posteriorly corresponds to the inferior LA just below the vein ostium. Distal CS pacing
maximally anticipates the bottom and posterior potential, whereas LAA pacing anticipates the top and anterior potential.

**Clinical Implications**
By establishing the nonvenous origin of the first potential, this study explains the inability to eliminate all potentials recorded in the left-sided PVs by proximal ablation and confirms selective elimination of the second potential as an indicator of electrical disconnection from the LA. This may reduce unnecessary RF delivery and the incidence of PV stenosis. Doubt about the left PV origin of a given signal can be clarified by CS pacing supplemented by LAA pacing, particularly if atrial capture in a distal position within the CS cannot be achieved. During LAA pacing, potential anticipation indicates LAA origin, whereas delay suggests PV potentials. Very short timing during atrial pacing resulting in one potential selectively fusing with the stimulus artifact indicates nonvenous origin as long as far field capture can be excluded (by threshold stimulation). Identical atrial electrogram timing should be verified in at least two different forms of activation, such as LAA and CS pacing, or distal and proximal CS pacing, or during sinus rhythm if a double potential is evident.

**Limitations**
This analysis depends on the circular catheter being within the PV so that the most near field structure remains the vein. Variations in catheter contact and position were minimized by choosing a slightly oversized circular catheter (confirmed by an increase in catheter loop diameter on withdrawal from the vein) and correlation with venography.

**Conclusion**
In addition to PV myocardium, the LAA provides a consistent and significant contribution to electrograms recorded in the LSPV. Lower amplitude, less-sharp extravenous potentials in the LIPV originate from the inferior LA. Nonvenous potentials can be reliably distinguished from left PV potentials by analysis of LAA and CS pacing.

**References**
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