Simultaneous, But Dissociated Left Atrial Fibrillation and Pulmonary Vein Tachycardia
A Case of Occult Pulmonary Vein Isolation

Marc A. Miller, MD; Sheldon M. Singh, MD; Andre d’Avila, MD, PhD; Vivek Y. Reddy, MD

A 74-year-old man with symptomatic drug-refractory persistent atrial fibrillation (AF) was referred for a catheter ablation procedure. The patient was in AF at the beginning of the procedure. As shown in Figure 1A, a decapolar catheter in the coronary sinus and a spiral mapping catheter within the ostium of the left common pulmonary vein (LCPV) demonstrated the presence of AF within both the left atrium (LA) and the LCPV, respectively. During circumferential ablation of the LCPV, the electrograms (EGMs) in the LCPV changed to an organized rhythm (mean cycle length, 178 ms) consistent with an ongoing PV tachycardia (Figure 1B). Suspecting that the LCPV was in fact isolated, the patient underwent electrical cardioversion, which terminated both the LA AF and the LCPV.

Figure 1. Surface ECG leads (I, aVF, and V₁) and intracardiac EGMs recorded from a circular multielectrode catheter placed inside the LCPV (SP ds to SP px) and the coronary sinus from proximal to distal (CS px to CS ds). A, Baseline EGMs before the onset of ablation. B, EGMs following circumferential ablation of the LCPV, demonstrating simultaneous, but dissociated LA fibrillation and PV tachycardia. Notice the regular cycle length of the PV tachycardia compared with the irregular activity within the LA. CS indicates coronary sinus; ds, distal; px, proximal; SP, spiral catheter.

Received July 11, 2010; accepted September 20, 2010.
From the Cardiac Arrhythmia Service, The Mount Sinai School of Medicine, New York, NY.
Correspondence to Vivek Y. Reddy, MD, Helmsley Electrophysiology Center, Mount Sinai School of Medicine, Zena and Michael A. Weiner Cardiovascular Institute, 1 Gustave L. Levy Pl, Box 1030, New York, NY. E-mail vivek.reddy@mounsinai.org
(Circ Arrhythm Electrophysiol. 2010;3:668-670.)
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Circ Arrhythm Electrophysiol is available at http://circep.ahajournals.org
DOI: 10.1161/CIRCEP.110.958645
tachycardia (Figure 2A). PV entrance and exit block was confirmed by (1) spontaneous isolated PV potentials (Figure 2A, arrows), (2) catheter-induced AF confined solely to the LA (Figure 2B), and (3) pacing within the PV (Figure 2C). Following a second cardioversion, programmed stimulation with burst atrial pacing (180 ms) from within the LCPV induced tachycardia confined exclusively to the vein (Figure 2D). Ablation of a single fractionated EGM (duration 82 ms) occupying 46% of the tachycardia cycle length within the LCPV terminated the tachycardia within 2 s (Figure 3). Thereafter, repetitive burst pacing failed to reinduce the PV tachycardia. Following electric isolation of the right PVs (not shown), there was no acute PV reconnection during a waiting period of 45 minutes or with the administration of isoproterenol (20 μg/kg per minute) or adenosine (12 mg).

PV isolation, the cornerstone of AF ablation, can be effectively performed not only in sinus rhythm, but also during AF. Yet in unusual situations, such as our case of simultaneous LA fibrillation and dissociated PV tachycardia, recognizing isolation is not always straightforward. The electrophysiological clue suggestive of this "occult" PV isolation was the organization of EGMs within the PV, despite ongoing fibrillatory activity within the LA. Although there certainly were periods of transient organization of the LCPV at baseline, PV electric organization only stabilized during circumferential PV ablation; this was important to recognize to avoid additional and unnecessary ablation of an already-isolated PV. Finally, although in theory the PV tachycardia should not clinically manifest from the isolated PV, it was nonetheless ablated because of (1) the known high rates of chronic PV reconnection; (2) the spontaneous and easily inducible nature of this PV tachycardia, suggesting a highly arrhythmogenic focus; and (3) the identification of a highly fractionated EGM consistent with a zone of slow conduction.

Although spontaneous sustained PV tachycardias following electric isolation only occur in a small percentage (1%) of PVs, canine models suggest that PVs provide a favorable substrate for reentry.1,2 Intra-PV decremental conduction, refractory period heterogeneity, and PV myofiber anisotropy have all been postulated to facilitate local reentry.3 However, whether PV tachycardia is due to focal ectopy or reentry still remains unclear. In a group of patients who underwent intra-PV burst pacing following electric isolation, a sustained PV tachycardia could be induced in 2.6% of veins, of which a majority demonstrated properties consistent with reentry.4 In our patient, the mechanism of induced PV tachycardia remains unknown. Nevertheless, identification of a critical zone of (presumed) slow conduction was identified and proved to require a minimal amount of ablation to achieve tachycardia termination.

Disclosures
Drs Reddy and d’Avila have received consulting fees from St Jude Medical, Inc, the manufacturer of the electroanatomical mapping system used in this case.
References


Key Words: atrial fibrillation • pulmonary veins • tachycardia
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Circ Arrhythm Electrophysiol. 2010;3:668-670
doi: 10.1161/CIRCEP.110.958645
Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/3/6/668

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