A case is reported of recurrence of paroxysmal atrial fibrillation after pulmonary vein ablation. A second procedure achieved isolation of three pulmonary veins and showed persistence of pulmonary vein tachycardia in one with implications concerning the electrophysiology of atrial fibrillation.

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Figure 1  ECG and intracardiac electrograms immediately after isolation of the RSPV. Patient is in sinus rhythm, as can be seen on the surface leads and the proximal and distal coronary sinus recordings (CSp and CSd, respectively). However, the 10-polar registrations from the Lasso catheter just inside the RSPV (L1-2–L10-1) show a very rapid, irregular PV rhythm inside the vein.

Figure 2  After 15 min of electrical silence inside the RSPV induction of PV tachycardia was attempted by pacing at basic CL of 400 ms, followed by a progressively shortened extrastimulus. Bipolar stimuli were given between Lasso electrodes 6 and 7. Capture was still achieved at a coupling interval of 80 ms (arrow), but no tachycardia was induced. Note that pacing in the PV does not capture the atria, as sinus rhythm persists. Abbreviations as in Fig. 1.
the atria were not captured and remained in sinus rhythm. Initially, no PV tachycardia could be induced, although the PV could be captured at very short coupling intervals of 80 ms (Fig. 2). To mimic the effects of atrial fibrillation in the PV, burst pacing was performed inside the PV at CL 100 ms, for 3 min. After this episode of burst pacing, sustained PV tachycardia could be induced very easily, even by one or two stimuli of the basic train at 400 ms (Fig. 3).

After its initial description in 1997 [1], catheter ablation in and around the PVs has become a promising technique for treatment of atrial fibrillation. Complete electrical isolation of the arrhythmic pulmonary veins has proven to be successful.[2] Recently, Oral et al. described the occurrence of rapid PV tachycardias during atrial fibrillation [3]. They showed that the occurrence of these PV tachycardias was related to input of impulses from the left atrium. During atrial fibrillation, there might be interaction between the posterior wall of the left atrium and the PVs, with PV tachycardias maintaining fibrillation in the atrium and input from the left atrium reinducing PV tachycardia. In dogs, both microreentry [4] and triggered activity [5] have been observed as possible mechanisms for tachycardias inside the pulmonary veins. Rapid activity from the atria may facilitate both triggered activity and reentry. Electrophysiological properties of human pulmonary veins, including very short refractory periods and slow conduction, may create a substrate for local reentry [6].

In our patient, spontaneous activity in the RSPV stopped shortly after its electrical isolation from the left atrium and initially no PV tachycardia could be induced by single extrastimuli. However, after a period of rapid burst pacing, mimicking an input from the left atrium during AF, PV tachycardia could easily be induced. We cannot exclude the possibility that scar formation after the initial ablation had created a substrate for PV reentry. Furthermore, the possible role of autonomic nervous system has not been explored. Our observations support the hypothesis of an interaction between the left atrium and PVs, contributing to perpetuation of AF.

References


