INTRODUCTION

Ectopic foci that induce paroxysmal atrial fibrillation (PAF) are generally located in the pulmonary veins (PVs). Nevertheless, several studies have described foci elsewhere, mainly in the posterior wall of the left atrium (LA) and in the superior vena cava (SVC). These sites, although less common, can be clinically relevant in specific cases. We describe our experience in three patients who showed focal activity in the superior vena cava in patients with symptomatic paroxysmal atrial fibrillation.

Key words: Atrial fibrillation. Atrial focus ablation. Superior vena cava.

PATIENTS AND RESULTS

Case 1

A 61-year-old man, who had been paraplegic since his youth due to an accident, had experienced two prior episodes of heart failure associated with sustained atrial tachyarrhythmia (atrial fibrillation and atrial flutter). The cardiologic study was normal, with the exception of mild left ventricular systolic dysfunction. Thyroid function was normal. He came to the emergency room with pulmonary edema, atrial fibrillation (AF), and a rapid ventricular rate. Monitoring during hospitalization showed sinus rhythm with runs of self-limited atrial tachyarrhythmia and episodes of sustained AF. Transesophageal echocardiography confirmed mild left ventricular systolic dysfunction, slight dilation of the LA, and an image consistent with thrombus in the left atrial appendage.

Following 6 weeks of anticoagulation therapy and after confirming resolution of the LA thrombus, an electrophysiological study was performed. A 24-pole catheter was advanced through the femoral vein to...
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simultaneously record the septal and anterolateral aspects of the right atrium (RA), and a deflectable catheter was inserted in the pulmonary artery (PA) for indirect mapping of the LA roof. A multipolar catheter was advanced through the right basilic vein toward the coronary sinus (CS) (Figure 1). Although no ectopic activity was present at baseline, infusion of high doses of isoproterenol induced atrial extrasystoles (AEs), with early activation in the posterior RA roof and late activation in the PA. Insertion of an explorer catheter in the SVC triggered frequent AEs and numerous runs of nonsustained atrial tachycardia. Recordings from the explorer catheter in the SVC at 2 cm above the junction with the RA showed earliest activation during the extrasystoles (Figure 2). After ruling out phrenic nerve stimulation, three applications of radiofrequency energy to the anterior aspect of the SVC were carried out, and the patient had no further episodes of spontaneous arrhythmia or inducible arrhythmia with stimulation maneuvers. Contrast venography showed no significant alterations. Echocardiography performed at one year showed normal left ventricular systolic function. After four years of follow-up, the patient has had no clinical arrhythmias.

Case 2

A 40 year-old woman, smoker with no other factors of interest, had experienced frequent, self-limited palpitations of short duration over the previous year; on two occasions PAF had been documented in the emergency room. The physical examination and echocardiography findings were normal. Holter monitoring with propafenone administration showed high-density AEs with numerous runs of self-limited AF.

The electrophysiological study was aimed at mapping possible focal activity. A 24-pole catheter was inserted to simultaneously record the anterior and septal RA activity, a decapolar catheter was directed toward the CS, and a deflectable explorer catheter was inserted in...
the right PA. Atrial stimulation triggered highly reproducible extrasystoles, with early recording in the high septal RA and late electrograms in the LA roof and the CS. During SVC mapping, simple pressure induced an automatic rhythm and extrasystoles, with premature beats occurring 50-60 ms early in the SVC recordings during the AEs. After ruling out phrenic nerve stimulation, two applications of radiofrequency energy were carried out, guided by the premature activation in the anterior area of the SVC at 2 cm above the atrio caval junction, thereby suppressing the automatic activity. After a follow-up of 17 months without antiarrhythmia medication, the patient has had no other episodes of arrhythmia, although she sometimes experiences sporadic precordial “jumps” and Holter monitoring shows an occasional AE.

**Case 3**
A 49-year-old woman with a history of hyperthyroidism-associated PAF in 1996. In 1999, her thyroid function was normal, but the symptoms persisted, with multiple episodes of PAF while she was receiving propafenone treatment. In 2002, ablation of the vena cava-tricuspid isthmus was performed for flutter occurring with the same treatment, but the PAF attacks continued, as well as numerous episodes of self-limited AF and frequent AEs on the Holter recordings.

The electrophysiological study was aimed at locating arrhythmogenic foci. A 24-pole catheter was advanced toward the RA for simultaneous recording of the anterior wall and septal wall, a decapolar catheter was advanced to the CS and an explorer catheter to the PA. Following administration of isoproterenol, AE was induced with early activation in the high RA septum, which triggered nonsustained AF. The earliest activation during extrasystole was located in the SVC; an origin in the PV was ruled out by mapping through the patent foramen ovale. Guided by a Lasso catheter (Cordis Webster, Diamond Bord, CA) placed in the SVC, three applications of radiofrequency energy were performed in the anterior and medial area, thereby electrically excluding the vein (Figure 3). Persistent blockage of the isthmus was confirmed.

At 2 months, PAF recurred. A new study confirmed isolation of the SVC and disclosed pronounced ectopic activity originating in the left superior PV that triggered nonsustained AF. The vein was electrically isolated using a Lasso catheter as a guide, and radiofrequency energy was applied with an irrigated catheter. The patient did not wish to discontinue propafenone treatment, and, after 22 months of follow-up, showed no clinical recurrence or alterations on Holter monitoring.

**DISCUSSION**
The role of the SVC in the genesis of AF was demonstrated in the two cases in which direct ablation of the arrhythmogenic foci completely resolved the arrhythmia. The third patient appeared to have multifocal ectopy because electrical exclusion of a PV in a second procedure was required to resolve the arrhythmia, despite the fact that PVs were initially ruled out as the origin of the ectopy by direct recording through the patent foramen ovale in the first study. This incidence accounts for 14% of all patients undergoing mapping of arrhythmogenic foci prior to ablation in our department and is close to reported values in the literature, which have described foci exclusively in the SVC in 6% and simultaneously in the SVC and other locations in up to 11%. Myocardial extensions into the SVC are the probable source of such ectopic
activity, which contributes to the initiation and maintenance of AF in all likelihood by a mechanism of abnormal automatic activity or a focal triggering activity, without ruling out possible reentry mechanisms within the SVC.

This incidence, although small, indicates the importance of mapping the origin of AEs whether they occur spontaneously or are induced during the study, particularly in recurrent cases following PV ablation. When the P-wave is visible, the ECG can help locate the site of origin. However, when the ectopic P-wave is superimposed on the T-wave and AF is induced with the first AE, study of the P-wave becomes more difficult. The location in the SVC has the added importance of easy access, making trans-septal catheterization unnecessary. In this context, a catheter in the CS and another in the PA to record activity in the LA roof are helpful for ruling out ectopic foci in the PV or the LA when ECG recordings in these locations are delayed with respect to those of the RA.

Induction of repetitive extrasystoles and runs of AF when the explorer catheter was inserted in the SVC was a noteworthy finding, particularly in case 1, in which stimulation and isoproterenol did not induce arrhythmia. It is difficult to separate this finding from a mere artifact in each individual case, but perhaps it should be regarded as important when no spontaneous or inducible ectopic activity from other origins is detected.

REFERENCES