

## Image in cardiology

## Antidromic Tachycardia Secondary to Accessory Pathway Ablation

## Taquicardia antidrómica secundaria a ablación de vía accesoria

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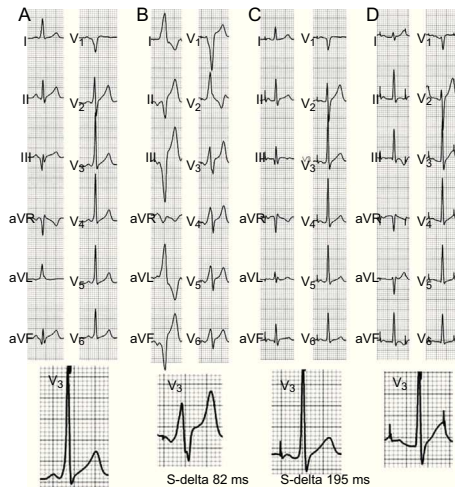


Figure 1.

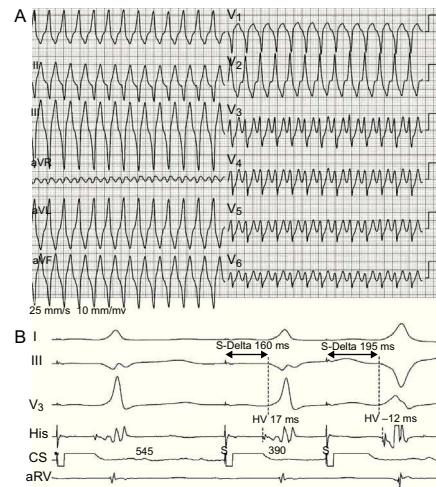


Figure 2.

A 23-year-old man with Wolff-Parkinson-White pattern was referred for evaluation and radiofrequency ablation treatment. The baseline electrocardiogram showed sinus rhythm at 67 bpm and delta wave consistent with the presence of a right posteroseptal accessory pathway (Fig. 1A). The accessory pathway had a refractory period of 450/290 ms. Tachycardia was not induced. Ventricular mapping was performed during atrial pacing at 600 ms (Fig. 1B), and 6 radiofrequency pulses with temperature control at 60° were applied to the region posterior to the tricuspid annulus, resulting in an increase in the local atrioventricular conduction time, a decrease in the degree of preexcitation (Fig. 1C, atrial pacing at 600 ms), and temporary success (Fig. 1D). Atrial pacing led to induction of antidromic tachycardia (Fig. 2A), using the accessory pathway as the anterograde arm and the specific conduction system as the retrograde arm. After radiofrequency treatment, the pathway acquired a Mahaim physiology (Fig. 2B; CS, coronary sinus; aRV, right ventricle), characterized by lengthening of the interval between atrial stimulation and the delta wave onset, associated with progressively greater preexcitation, which occurred reproducibly and with a wide range of extrastimuli coupling intervals, as well as with continuous atrial runs at gradually increasing frequencies. In addition, the pathway showed a conduction block following intravenous administration of a 12-mg bolus of adenosine. With an open irrigated catheter, a 60-s radiofrequency pulse (50 °C, 50 W) was applied at the same position, and conduction through the pathway terminated at 2 s (Fig. 1D). Conduction did not reappear after a 40-min wait, and posterior tachycardia induction was not performed.

Radiofrequency treatment on tissues of the accessory pathways and surroundings results in cellular electrophysiological changes that can lead to decremental conduction in pathways with a degree of persistent conduction. This may lead to sustained arrhythmias that were not inducible before treatment and that might appear during subsequent follow-up. Complete elimination of accessory pathway conduction should be the final aim in these patients.

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