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Letter to the Editor

Repetitive, incessant supraventricular tachycardia: Noninvasive determination of the electrophysiologic mechanism

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In patients with supraventricular tachycardia (SVT), clinical electrophysiologic studies utilize programmed atrial and ventricular stimulation and electrical activation mapping to decipher the mechanism of the tachycardia. In cases of repetitive SVT, electrocardiographic (ECG) monitoring provides a unique opportunity to noninvasively investigate the effects of spontaneous premature atrial and ventricular complexes (PACs and PVCs) on the initiation and termination of the SVT [1,2]. In addition, analyzing the QRS and P-wave morphologies during ventricular preexcitation and during SVT, respectively, can be viewed as rudimentary forms of electrical activation mapping [3]. The following report describes a patient with repetitive SVT where careful analysis of telemetry recordings led to an accurate diagnosis of the tachycardia mechanism. It also offered a logical explanation for paradoxical responses of the SVT to conventional antiarrhythmic medications. The patient underwent successful catheter ablation.

A 58-year-old woman with chronic left bundle branch block (LBBB) was hospitalized for acute respiratory failure. During her hospital course, she had several episodes of SVT. On each occasion, intravenous adenosine transiently terminated the tachycardia but frequently, the SVT quickly returned. The patient was then treated with repeat intravenous boluses of metoprolol and diltiazem after which the tachycardia became repetitive and almost incessant. When all cardioactive medications were turned off, the SVT paradoxically subsided. Fig. 1 illustrates the 12-lead ECG during sinus rhythm (panel A) and during SVT (panel B). Fig. 2 shows the heart rate trend curves with (panel A) and in the absence of repeat administration of IV metoprolol and diltiazem (panel B). Over a 3-day observation period, more than 30 spontaneous initiations and terminations of the SVT were documented in telemetry. Fig. 3, panels A–D are representative examples of initiation of the SVT. Fig. 3, panels E–H are examples of spontaneous terminations. Based on analysis of the 12-lead ECGs and telemetry recordings, we concluded that the patient had repetitive orthodromic atrioventricular macroreentrant SVT utilizing a concealed left-sided accessory pathway. She underwent an electrophysiologic study which confirmed this ECG diagnosis (Fig. 4). Successful radiofrequency ablation was performed.

The following simple observations helped establish the tachycardia mechanism. First, each episode of the SVT was triggered by a PAC with a relatively long P-R interval (Fig. 3, panels A–D). The P-wave morphology during SVT was different from the morphology of the triggering PAC (Fig. 3D). These observations were consistent with a reentrant mechanism [4]. Second, both the administration of adenosine and spontaneous PVCs was able to terminate the tachycardia (Fig. 3, panels E–H). These findings suggested that the tachycardia circuit involved both anterograde and retrograde A-V conduction. Third, spontaneous PVCs that occurred at a time when the His bundle was refractory consistently and markedly preexcited the atria. Note that in Fig. 3H, a PVC that was inscribed about 40 ms before the expected onset of the next QRS complex of the SVT resulted in a P-P interval of 380 ms, 110 ms shorter than the other P-P intervals during the tachycardia. This finding can only be explained by the presence of an accessory pathway capable of retrograde conduction [4,5]. Finally, during SVT the retrograde P waves were negative in the left-sided leads and were upright in lead V1, indicating a left-to-right atrial activation sequence and thus, retrograde conduction over a left free wall accessory pathway (Fig. 1B) [6].

Uncovering the electrophysiologic mechanism of the SVT also helped explain the paradoxical response of the SVT to conventional antiarrhythmic medications. In patients with concealed Wolff–Parkinson–White syndrome two important elements of reentry, namely two conduction pathways and unidirectional block in one of the pathways, are always present. Under these circumstances, initiation and maintenance of reentry depends on a delicate balance between conduction time

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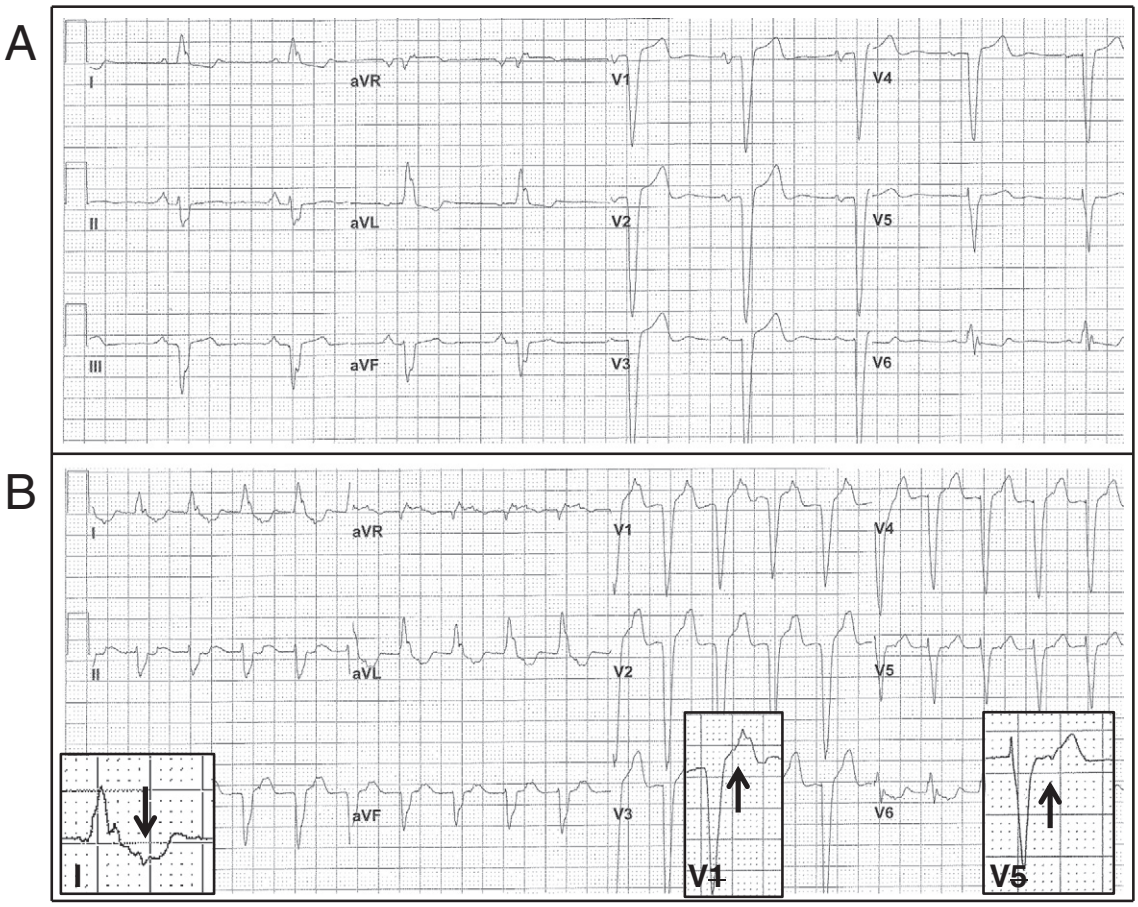

Fig. 1. A: Twelve-lead electrocardiogram (ECG) during sinus rhythm. B: Twelve-lead ECG during supraventricular tachycardia. Note the retrograde P waves that follow the onset of the QRS complexes by about 220 ms. The enlarged inserts demonstrate that the retrograde P waves are negative in the left-sided leads and are upright in V1.

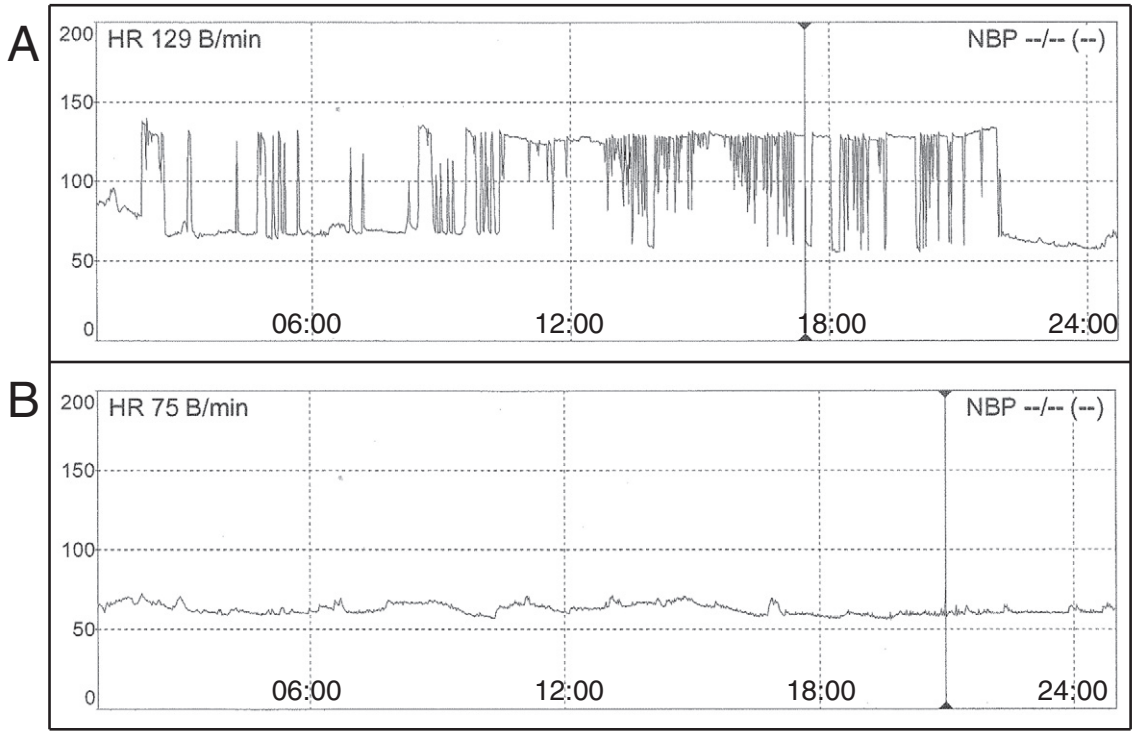

Fig. 2. A: 24-hour heart rate trend curve demonstrates repetitive, incessant supraventricular tachycardia at a time when the patient was receiving repeat intravenous boluses of metoprolol and diltiazem. B: 24-hour heart rate trend curve after all cardioactive medications were discontinued.