ARRHYTHMIA OF THE MONTH

Section Editor: Fred Morady, M.D.

Spontaneous Termination of a Tachycardia: What Is the Tachycardia Mechanism?—VI

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Case Presentation

A 55-year-old man underwent an electrophysiologic procedure for evaluation and treatment of recurrent episodes of paroxysmal tachycardia that had been occurring sporadically for 10 years. After placement of electrode catheters in the high right atrium and right ventricular apex, but before catheter placement in the His-bundle position or coronary sinus, premature ventricular depolarizations triggered an episode of tachycardia that terminated spontaneously in 10 seconds. The tachycardia cycle length varied between 390 and 410 msec. The last 3 seconds of the tachycardia are shown in Figure 1. What is the tachycardia mechanism?

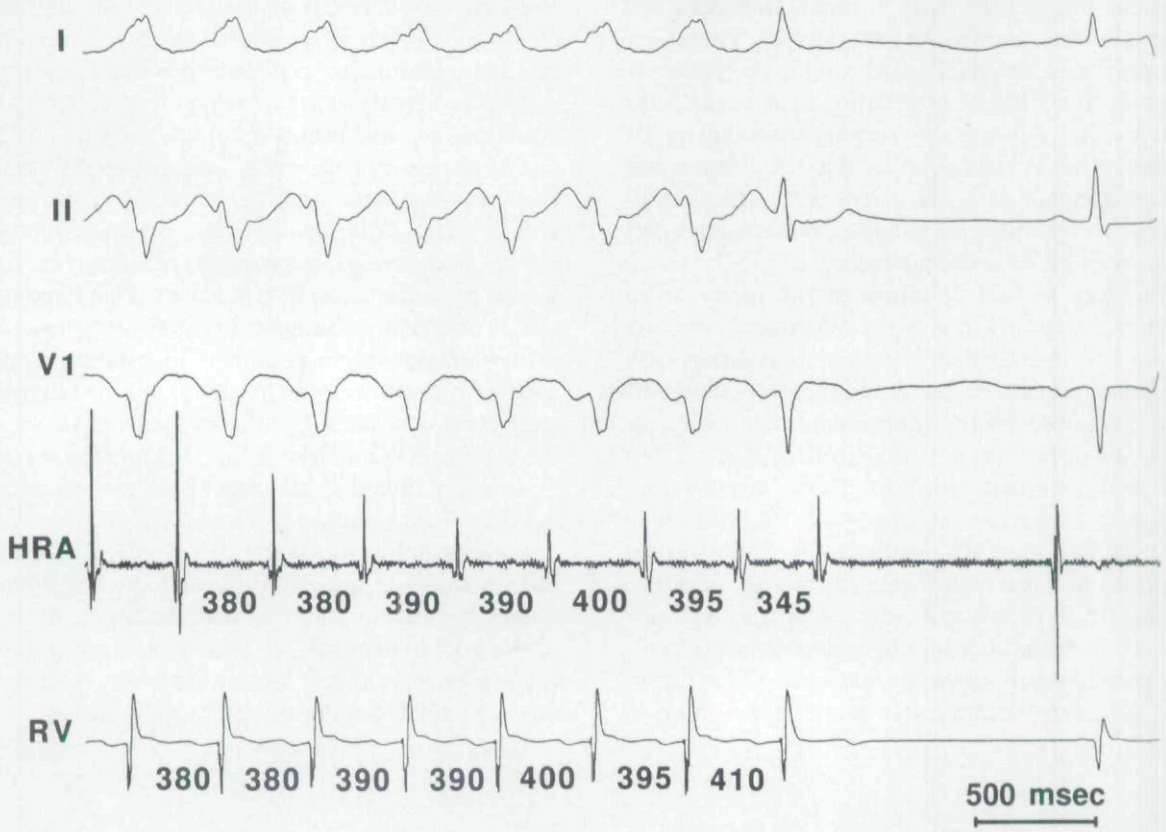

Figure 1. *Spontaneous termination of a tachycardia that occurred in the early phase of an electrophysiologic procedure, during catheter placement. Shown are leads I, II, and Vl, the high right atrial electrogram (HRA), and the right ventricular apex electrogram (RV). The atrial and ventricular cycle lengths are indicated in the HRA and RV recordings in milliseconds.*

J Cardiovasc Electrophysiol, Vol. 7, pp. 905-906, September 1996.

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Manuscript received 22 May 1996; Accepted for publication 22 May 1996.

Commentary

Because this episode of tachycardia occurred before the His-bundle or coronary sinus electrograms were recorded, a paucity of data is available from analysis of the intracardiac electrograms. The ECG leads demonstrate that the tachycardia consists of wide QRS complexes that have a left bundle branch block configuration, except for the last QRS complex, which is narrow. Narrowing of the QRS complex occurs in association with a slight prolongation of the tachycardia cycle length to 410 msec. There is a 1:1 relationship between the ventricular and atrial electrograms, and the ventriculoatrial (VA) interval is constant at 210 msec, except for the last VA interval of the tachycardia, which is 160 msec.

Normalization of the QRS complex in association with prolongation of the tachycardia cycle length strongly suggests that there was a rate-dependent left bundle branch block that resolved when the rate became slightly slower. Therefore, a supraventricular tachycardia with aberration is a much more likely possibility than ventricular tachycardia. Among the various mechanisms of supraventricular tachycardia, the VA relationship in this tachycardia is consistent with either orthodromic tachycardia, atypical atrioventricular (AV) nodal reentry, or atrial tachycardia.

The key to identification of the tachycardia mechanism in this case is the 50-msec shortening of the VA interval that occurs in association with resolution of the left bundle branch block aberration. This observation indicates that the tachycardia is an orthodromic tachycardia utilizing a left free-wall accessory pathway. There would be no reason to expect shortening of the VA interval in association with QRS normalization in AV nodal reentrant or atrial tachycardia. However, in orthodromic tachycardia utilizing a left free-wall accessory pathway, left bundle branch block aberration necessitates transseptal conduction of the tachycardia wavefront to the left ventricle, resulting in prolongation of the VA interval by at least 30 msec. Therefore, shortening of the VA interval in this case is explained by resolution of the left bundle branch block and elimination of transseptal conduction during the tachycardia.

Why does the tachycardia terminate when the VA interval shortens? The most likely explanation is that the abrupt shortening of the atrial cycle length from 395 to 345 msec, which occurs because of the 50-msec decrement in the VA interval, results in AV nodal block.

As is often the case when analyzing electrophysiologic phenomena, alternative explanations are possible. One possible alternative explanation is that the last atrial complex of the tachycardia simply represents a spontaneous atrial premature depolarization, in which case any type of supraventricular tachycardia could have been terminated. However, this explanation requires the simultaneous occurrence, by coincidence, of two unrelated phenomena—slowing of the tachycardia and the occurrence of an atrial premature depolarization. A second alternative explanation is that the tachycardia is ventricular in origin, that retrograde conduction to the atrium occurs over either a concealed accessory pathway or one of two AV nodal pathways, and that upon termination of the ventricular tachycardia, anterograde conduction through the AV node results in a narrow QRS complex followed by a single atrial echo beat. This explanation is unattractive because it requires the presence of two unrelated abnormalities. In contrast, in the case of orthodromic tachycardia and a left free-wall accessory pathway, the events that occur at the time of termination of the tachycardia all can be causally linked to slowing of the rate, and it is not necessary to invoke a coincidence or more than one abnormality to explain the findings. During the remainder of the electrophysiologic procedure, diagnostic maneuvers confirmed that the tachycardia was orthodromic, and mapping demonstrated the presence of a left lateral accessory pathway that was ablated with radiofrequency energy.