ARRHYTHMIA OF THE MONTH

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A Narrow QRS Complex Tachycardia: What is the Mechanism of Tachycardia?

BRADLEY P. KNIGHT, M.D.

From the Division of Cardiology, Department of Internal Medicine, University of Michigan Health System, Ann Arbor, Michigan

Case Presentation

An 80-year-old man underwent an electrophysiologic procedure because of recurrent episodes of supraventricular tachycardia. Episodes of nonsustained narrow QRS complex tachycardia occurred spontaneously after catheter placement. Examples of the onset and termination of an episode of tachycardia are shown in Figures 1 and 2, respectively. What is the mechanism of the tachycardia? What is the level of AV block?

Commentary

Figure 1 shows a sinus beat followed by two atrial premature depolarizations. Each premature beat has a "high-low" atrial activation sequence and conducts to the ventricle. A tachycardia with an average cycle length of 360 msec and a "low-high" atrial activation sequence then is initiated. The septal ventriculoatrial conduction time is 120 msec with the first tachycardia beat, but progressively shortens with the next two beats. The third tachycardia beat does not conduct to the ventricle and is followed by transient 2:1 AV block. The tachycardia persists with 1:1 AV conduction (not shown) and then spontaneously terminates (Fig. 2).

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Address for correspondence: Bradley P. Knight, M.D., University of Michigan Health System, 1500 East Medical Center Drive, B1F245, Ann Arbor, MI 48109-0022. Fax: 734-936-7026; E-mail: bpk@umich.edu

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This case highlights the diagnostic value of careful analysis of the onset and termination of spontaneous tachycardias. The development of apparent Wenckebach AV block initially suggests that the tachycardia might be atrial in origin and definitively excludes orthodromic AV reentry as the mechanism of tachycardia. However, an atypical "slow-slow" form of AV nodal reentry or junctional ectopic tachycardia remains in the differential diagnosis. Of note is that the tachycardia terminates with an atrial electrogram at a time when there is 1:1 conduction during the tachycardia. The coincidental termination of an atrial tachycardia and the development of AV block would be very unlikely. That this observation was reproducible excludes atrial tachycardia as the tachycardia mechanism. Although junctional ectopic tachycardia cannot be excluded, the presence of AV nodal block during tachycardia makes AV nodal reentry much more likely than junctional ectopic tachycardia. Therefore, the tachycardia diagnosis is most likely AV nodal reentry.

Sustained 2:1 AV block occurs in approximately 10% of cases of AV nodal reentry induced in the electrophysiology laboratory.[1-3] There are two possible mechanisms of 2:1 AV block: (1) intranodal, lower common pathway block or (2) infranodal block. The absence of a His-bundle potential in the blocked beats has been considered evidence of intranodal block.[1,4] However, there is evidence that 2:1 AV block usually is due to functional block below the AV node, set up by a "long-short" initiation sequence. Block has been shown to persist despite the administration of atropine and to resolve after

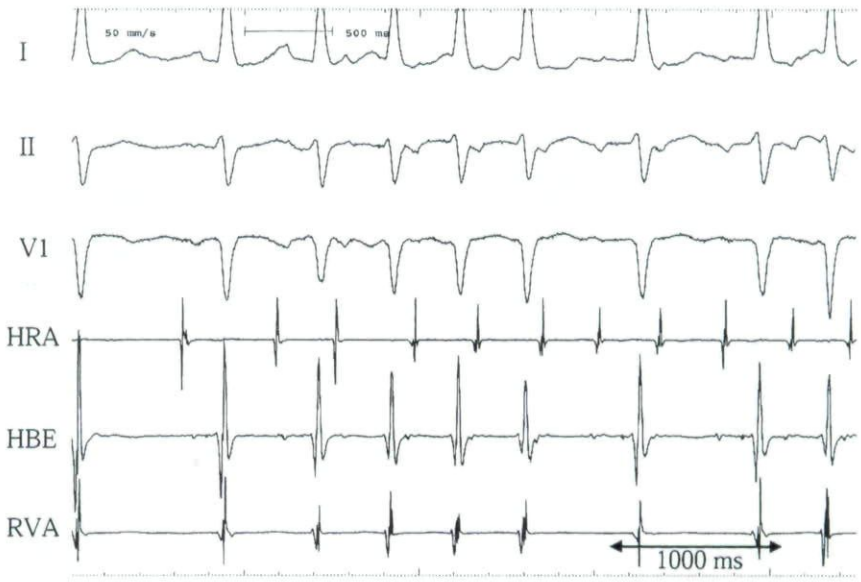

Figure 1. *Spontaneous onset of tachycardia. Shown are leads I, II, and V_1, and electrograms from the high right atrium (HRA), His-bundle (HBE), and right ventricular apex (RVA).*

a single ventricular extrastimulus, even in the absence of a His-bundle potential with the blocked beats.[2]

In the present case, the AH intervals of the conducted beats during 2:1 AV block are considerably shorter than the AH interval during tachycardia when there is 1:1 conduction. This observation indicates that the site of block in this case probably is intranodal, in a lower common pathway. There have been previous reports of lower common pathway Wenckebach block during AV node reentry.[5,6]

The tachycardia was eliminated successfully by the delivery of radiofrequency current to the low right atrial septum near the coronary sinus os at the same location where slow pathway ablation is performed for typical AV nodal reentry.

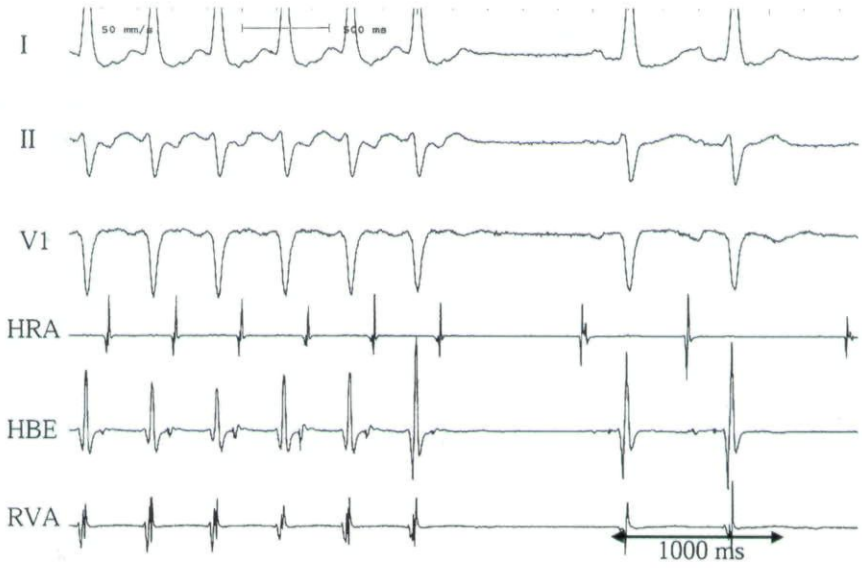

Figure 2. *Spontaneous termination of tachycardia. Shown are leads I, II, and V_1, and electrograms from the high right atrium (HRA), His-bundle (HBE), and right ventricular apex (RVA).*

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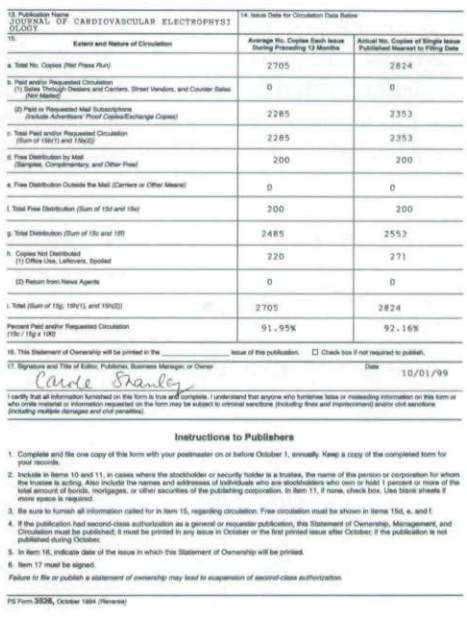

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