Paroxysmal Supraventricular Tachycardia With Wenckebach Block: Evidence for Reentry Within the Upper Portion of the Atrioventricular Node

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Reentry utilizing dual pathways within the atrioventricular (AV) node and AV reentry involving retrograde conduction over an accessory extranodal pathway are the two mechanisms most commonly thought to be responsible for clinical episodes of recurrent paroxysmal supraventricular tachycardia (1). In these two forms of paroxysmal supraventricular tachycardia, anterograde conduction occurs through the AV node, and the occurrence of spontaneous, vagally mediated or drug-induced AV nodal block would terminate the arrhythmia. In a number of reports (2-6), however, cases of paroxysmal supraventricular tachycardia have been described in which anterograde Wenckebach type second degree AV block has occurred without tachycardia termination. Although this phenomenon would be expected to occur in patients with either sinus node reentry or intraatrial reentry, there have been cases in which the earliest atrial activation during tachycardia documented at electrophysiologic study was recorded by the catheter in the His bundle position.

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regular, narrow complex tachycardia with a rate between 150 and 170 beats/min. On three occasions, the tachycardia was terminated by administering intravenous verapamil. Continuous electrocardiographic tracings from these visits are not available. On occasion, the patient did not respond to 10 mg of verapamil and the tachycardia was terminated with direct current cardioversion. Chronic prophylactic therapy with digoxin, propranolol, quinidine, disopyramide or verapamil alone and in various combinations had been unsuccessful in preventing recurrent episodes or had resulted in side effects.

Physical examination revealed a grade 2/6 systolic ejection murmur most audible at the right upper sternal border and a 1/6 decrescendo diastolic murmur along the left sternal border. No opening snap or diastolic rumble was heard. The surface electrocardiogram showed normal sinus rhythm with nonspecific ST-T wave abnormalities. Cardiac catheterization performed to evaluate chest pain 1 year previously had demonstrated normal coronary arteries, trace aortic regurgitation and no mitral valve gradient. There was no calcification in either the aortic or mitral valve on fluoroscopy.

Methods

Electrophysiologic study. This was performed with the patient in a fasting nonsedated state after she had given written informed consent. All antiarrhythmic drugs were discontinued 48 hours before the study. Four quadripolar electrode catheters (USCI) with a 1 cm interelectrode distance were introduced through the right and left femoral veins and a left antecubital vein and positioned in the high right atrium, the coronary sinus, the region of the bundle of His and the right ventricular apex. Intracardiac electrograms were filtered below 30 and above 500 Hz and displayed with surface electrocardiographic leads I, II and V1 on a multichannel oscilloscope (Electronics for Medicine, VR-16) for subsequent retrieval and analysis. Stimulation was performed using a programmable stimulator (Medtronic VR-16) for subsequent retrieval and analysis. Stimulation was performed using a programmable stimulator (Medtronic VR-16) for subsequent retrieval and analysis. Stimulation was performed using a programmable stimulator (Medtronic VR-16) for subsequent retrieval and analysis. Stimulation was performed using a programmable stimulator (Medtronic VR-16) for subsequent retrieval and analysis. Stimulation was performed using a programmable stimulator (Medtronic VR-16) for subsequent retrieval and analysis. Stimulation was performed using a programmable stimulator (Medtronic VR-16) for subsequent retrieval and analysis. Stimulation was performed using a programmable stimulator (Medtronic VR-16) for subsequent retrieval and analysis. Stimulation was performed using a programmable stimulator (Medtronic VR-16) for subsequent retrieval and analysis.

Drug administration. Crystalline adenosine (Sigma Chemical Company) was dissolved in normal saline solution to a final concentration of 10 mg/ml. The electrophysiologic effects of single intravenous bolus doses of adenosine were determined during both sinus rhythm and supraventricular tachycardia. In both instances, an initial dose of 37.5 μg/kg was injected and, if no effect was observed, the dose was increased in 37.5 μg/kg increments either: 1) until sinus bradycardia or second degree AV block, or both, was observed when the patient had sinus rhythm, or 2) until the tachycardia was terminated when supraventricular tachycardia was present. Multiple episodes of supraventricular tachycardia were terminated with adenosine; 1 episode at a dose of 75 μg/kg and another 11 with a dose of 112.5 μg/kg. The administration of adenosine was undertaken in accordance with a protocol approved by the Human Investigations Committee of the University of Virginia.

After the initial assessment of the action of adenosine, the following drugs—atropine (0.03 mg/kg), propranolol (0.15 mg/kg) and verapamil (10 mg)—were injected in that sequence. Paroxysmal supraventricular tachycardia was reinitiated immediately after atropine and propranolol administration and the effects of adenosine on the tachycardia reassessed. The tachycardia could not be reinitiated after verapamil administration.

Results

Observations during sinus rhythm. During sinus rhythm, the PR (190 ms), AH (110 ms) and HV (50 ms) intervals were within normal limits. Only a single discrete His bundle potential could be recorded despite careful catheter manipulations in search of a split His potential. Intra-ventricular conduction during sinus rhythm was normal. During programmed atrial stimulation with incremental extrastimuli, a continuous AV nodal conduction curve was demonstrated. There was no evidence for pre-excitation. No ventriculoatrial conduction was observed during ventricular pacing at any cycle length below the sinus cycle length of 800 ms or after any ventricular extrastimulus introduced throughout diastole during sinus rhythm.

Properties of the tachycardia. Paroxysmal supraventricular tachycardia could be reproducibly initiated with a single right or left atrial extrastimulus during sinus rhythm and during atrial pacing with A1A2 intervals of 280 to 310 ms at drive cycle lengths between 500 and 700 ms. A critical A2H2 interval of greater than 210 ms was always associated with initiation of supraventricular tachycardia. The A1A2 intervals that initiated tachycardia were outside the atrial relative refractory period (260 ms). During tachycardia, there was a constant atrial cycle length of 340 ms, but the ventricular rate was occasionally irregular (Fig. 1). Analysis of AV conduction demonstrated variable Wenckebach cycles (10:9 to 2:1) that alternated with periods of 1:1 AV conduction. The P wave configuration in lead II was biphasic and of lower amplitude than that recorded during sinus rhythm. During atrial mapping, the earliest atrial activity recorded was the septal atrial deflection on the His bundle electrogram.

The tachycardia could be readily terminated with a single appropriately timed right or left atrial extrastimulus or with rapid atrial pacing, but was not affected by ventricular extrastimuli. When premature atrial stimuli were introduced during paroxysmal supraventricular tachycardia, atrial capture that did not terminate the tachycardia was followed by a fully compensatory pause in the atrial cycle but the ventricular cycle length was unchanged. After the administra-
Figure 1. Control study. In the top panel are shown surface electrocardiographic lead II and intracardiac recordings from the high right atrium (HRA), the coronary sinus (CS) and the bundle of His (HB). A ladder diagram based solely on these initial tracings is shown in the bottom panel. The atrial cycle length is regular at 270 ms but there is 4:3 anterograde AV nodal block with a progressive lengthening of the AH interval. Note that the earliest atrial activity is recorded on the His bundle electrogram. This ladder diagram, although useful in explaining the electrocardiographic pattern, does not fully account for the observations after drug administration.

In each instance, there was an increase of 25 to 30 ms in both atrial and ventricular cycle lengths before the tachycardia was interrupted. The last atrial impulse was always conducted to the ventricle. During sinus rhythm (cycle length 800 ms) an increase in the AH interval after intravenous adenosine did not occur until a dose of 187.5 μg/kg was reached.

Pharmacologic responses. Adenosine. Intravenous adenosine (75 or 112.5 μg/kg) reproducibly terminated the tachycardia within 20 seconds after peripheral injection (Fig. 2).

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Atropine with and without propranolol. After atropine administration (0.03 mg/kg), the tachycardia cycle length decreased to 310 ms and there was 1:1 AV conduction. The infusion of intravenous propranolol (0.1 mg/kg) slowed the tachycardia to a cycle length of 420 ms. Atioventricular conduction was variable with periods of both 1:1 conduction and Wenckebach block observed. Intravenous adenosine (112.5 μg/kg) continued to terminate episodes of parox-
Figure 3. Effect of verapamil. The tracings are labeled as in Figure 2. Verapamil (10 mg intravenously) was injected 3 minutes earlier. On the last 5 beats of tachycardia, the atrial cycle length oscillates from 390 to 470 ms with only a minor change in AH interval or ventricular cycle length. The oscillation in atrial cycle length would be consistent with variable retrograde exit delay from a nonatrial reentrant site with a basic cycle length of about 420 ms that continued to manifest anterograde conduction to the ventricle. Termination of the tachycardia was associated with anterograde block. Abbreviations as in Figure 2.

Discussion

Possible mechanisms for the arrhythmia. The electrophysiologic findings described may be used to define the mechanism responsible for recurrent paroxysmal supraventricular tachycardia in our patient. The lack of ventriculoatrial (VA) conduction eliminates reentry using an accessory atrioventricular (AV) nodal bypass tract as a mechanism for the observed tachycardia. Sinus node reentry can be excluded in light of the low to high atrial activation sequence and the altered P wave configuration during tachycardia. The reliable initiation and termination of the arrhythmia with a single atrial extrastimulus excludes an automatic focus. Four other possibilities must be considered: 1) intraatrial reentry, 2) triggered automaticity from a low atrial or AV junctional focus, 3) reentry located within the AV node with the site of Wenckebach block either within the node itself, or 4) within the bundle of His.

Intraatrial reentry. Josephson and Seides (8) suggested criteria for the diagnosis of supraventricular tachycardia due to intraatrial reentry. These criteria are: 1) the arrhythmia is initiated by atrial premature beats during the atrial relative refractory period; 2) the atrial activation sequence is different from that observed in normal sinus rhythm; 3) the PR interval varies inversely with the tachycardia cycle length; 4) AV nodal block may exist without affecting the tachycardia; and 5) vagal maneuvers usually do not terminate the tachycardia.

By these criteria, intraatrial reentry may be excluded. The tachycardia could be initiated by atrial extrastimuli beyond the atrial relative refractory period and latency was not associated with the extrastimulus that initiated the tachycardia. In the past, vagal maneuvers had been successful in terminating some episodes of tachycardia. Adenosine effectively terminated multiple episodes and we have previously shown (9) that adenosine has no effect on the atrial cycle length in either intraatrial reentry or atrial flutter even at doses that produce high degree AV block. In these studies (9), we also demonstrated that adenosine does not terminate these arrhythmias. The termination of the tachycardia after verapamil is also more likely to occur if the reentrant circuit involved the AV node, even though verapamil may increase the atrial refractory period in many patients (10). After verapamil and when atrial extrastimuli were introduced, atrial activation could vary without a concomitant change in ventricular cycle length, again suggesting the atrium was not an integral part of the reentrant circuit (11).

Triggered automaticity. It is difficult to distinguish triggered automaticity from reentry because the responses to programmed stimulation may be identical. Adenosine blocks catecholamine-induced triggered automaticity in vitro (12). However, the continued ability of adenosine to initiate and
terminate the tachycardia after beta-adrenergic blockade and the transient AV dissociation after verapamil strongly suggest that triggered automaticity was not the mechanism responsible for supraventricular tachycardia in this patient.

Wenckebach block within AV node or bundle of His. Wenckebach type second degree AV block is usually thought to result from decremental conduction within the midportion of the AV node (13,14), but intra-Hisian Wenckebach block has occasionally been observed (15–17). In these reports, however, the increments in intra-Hisian conduction have usually been minor (10 to 15 ms) in comparison with the marked increments (to 150 ms) in AH interval that could occur during the Wenckebach cycles in our patient. Despite persistent attempts to demonstrate intra-Hisian block, two distinct His potentials could not be recorded during the study. We also noted that after atropine, 1:1 conduction at a shorter cycle length of 310 ms could occur, a finding that is at least more consistent with the AV node being the prior site of block. Thus, although intra-Hisian Wenckebach block cannot be totally excluded, it seems an unlikely explanation for the phenomena we observed.

For these reasons we believe that the reentrant circuit was most probably located within the upper portion of the AV node. Specifically we made the following observations: 1) persistence of tachycardia despite Wenckebach type, second degree AV block; 2) termination of the tachycardia by adenosine, an agent that preferentially affects the upper AV node (18) but does not influence intraatrial conduction or terminate intraatrial reentry (9); 3) a tachycardia cycle length sensitive to autonomic influences; 4) earliest atrial activation in the region adjacent to the AV node, and 5) dissociation of atrial and ventricular cycle lengths after verapamil and atrial extrastimuli. The coexistence of two functionally separate AV nodal regions that may both exhibit cycle length-dependent decremental conduction may also partially explain the oscillation in atrial activity with a constant ventricular cycle length we observed after administration of verapamil.

We are unable to exclude participation of a small portion of atrial tissue in the immediate perinodal region, as has been suggested by Inunna et al. (19), but this tissue would have to be isolated during tachycardia from the other atrial sites where we were recording during the study.

Clinical significance. Reentrant tachycardias arising within the AV node have been attributed to longitudinal dissociation of conduction. Classic dual AV nodal pathways may be demonstrated in many patients with such tachycardias, but if the change in conduction between the pathways is minor, continuous conduction curves may be recorded (20). The site within the node at which the dissociation occurs has been studied less frequently. Several authors (3,4,21,22) postulated that some episodes of paroxysmal supraventricular tachycardia may involve reentry within the upper portion of the AV node, but clinical demonstration of this has proven difficult. We believe the electrophysiological data obtained in our patient represent the clearest available clinical evidence in support of this hypothesis.

References