Supraventricular Tachycardia Initiated by Sinus Beats

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SUMMARY
In the present report, A-V dissociation with concealed retrograde conduction into the A-V node, caused SINUS beats to exhibit antegrade A-V nodal conduction delay sufficient to result in episodes of supraventricular tachycardia (SVT). In this patient, atrial ectopic premature depolarizations were never observed. The primary role of A-V nodal conduction delay in the genesis of paroxysmal SVT is substantiated by this unusual electrophysiological observation.

Additional Indexing Words:
Paroxysmal atrial tachycardia  Atrial reentry  Atroventricular conduction
A-V dissociation  Ventricular reciprocal rhythm  A-V refractoriness
Atrial echoes  Ventricular echoes  Reentry phenomenon

Occasionally, a rare physiological event is of significance not because of its rarity, but because it illuminates a frequently occurring but poorly understood phenomenon. Such an event occurred in 1956, when during studies of A-V conduction, Moe et al.,1 found a single dog in whom paroxysms of supraventricular tachycardia could be initiated by appropriately timed single stimulated atrial premature depolarizations. This extremely unusual individual animal demonstrated that supraventricular tachycardia (SVT) could be initiated by reentry with the A-V-conduction system. The mechanism responsible for paroxysms of SVT in man was again brought into question. Recent studies have demonstrated that Moe’s unique observation was extremely relevant.

In patients with paroxysmal SVT, episodes of this arrhythmia are almost invariably initiated by atrial premature beats and sustained by reentry via the A-V node.2-6 In all of the previously reported and studied patients, however, SVT was always initiated by some ectopic atrial depolarization. In the subject of this report, spontaneous episodes of paroxysmal SVT were uniformly initiated by sinus beats during which A-V-nodal conduction was dramatically prolonged by concealed retrograde conduction occurring during periods of isorhythmic A-V dissociation. Undoubtedly an extremely unusual mechanism for the initiation of paroxysmal SVT, this chance observation nonetheless provides strong reinforcement for the conclusion that SVT is initiated and sustained by A-V-nodal reentry rather than any ectopic atrial focus.

Methods
The patient (E.B.) was an 18-year-old girl referred because of frequent episodic palpitations. She had had only the usual childhood diseases, had normal exercise tolerance, and had sustained an uneventful pregnancy the year prior to referral. Beginning at age 12, the patient recalled frequent (1–2/day) episodes of rapid regular palpitations always beginning and ending suddenly. Episodes lasted from minutes to hours, and

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although occasionally accompanied by light-headedness, they never resulted in syncope. She was unaware of any precipitating cause and had never attempted vagal maneuvers to terminate episodes of tachycardia. Physical examination, chest X-ray, and electrocardiogram were all normal.

Electrocardiographic monitoring demonstrated episodes of supraventricular tachycardia that developed during a period of isorhythmic A-V dissociation. Since the precise mechanism of this arrhythmia was difficult to ascertain from the ECG alone, the patient consented to electrophysiological studies during right heart catheterization. Atrial electrogram from the high right atrium and His-bundle electrogram were obtained and recorded by methods previously described in detail.4-6,7 The A-H interval was measured from the initiation of atrial depolarization in the high right atrium to the onset of His-bundle depolarization in the His-bundle electrogram (HBE). The A-H interval could not be measured from the HBE alone, since during A-V dissociation atrial and ventricular depolarizations occurred simultaneously in the HBE. The H-V interval was measured from the initiation of His-bundle depolarization to the depolarization of the interventricular septum as reflected in the HBE. The functional capacity of the A-V-conduction system was measured by the extrastimulus method.2,8

Results

During sinus rhythm recordings from the high right atrium, low interatrial septum, and His bundle demonstrated a normal sequence of antegrade atrial depolarization resulting in upright P waves in standard ECG leads I, II, and III (fig. 1). The normal A-H (120 msec) and H-V (40 msec) intervals resulted in a normal P-R interval of 0.16 sec. The A-V effective refractory period measured by the extrastimulus method during atrial pacing at a cycle length of 630 msec was 300 msec and within normal limits.8 Occasional VPCs evoked during positioning of the His-bundle catheter demonstrated 1:1 ventriculoatrial conduction; VA refractory periods were not determined.

More than 40 spontaneous episodes of A-V dissociation were observed (fig. 2). These invariably resulted when the sinus-node pacemaker slowed so that its rate became less than that of a subsidiary pacemaker. During A-V dissociation, the H-V interval and the sequence of ventricular depolarization as reflected in the standard ECG leads were identical to that observed during sinus rhythm. Because of this, during the A-V junctional rhythm the subsidiary pacemaker was judged to reside within the His bundle or lowest (N-H) region of the A-V node. Episodes of A-V dissociation were uniformly terminated by one of two mechanisms. Generally, the sinus-node pacemaker increased its rate and the normal sequence of antegrade A-V conduction was
Figure 2

A-V dissociation. ECG leads 1, 2, 3, atrial electrogram (A), two His-bundle electrograms (H), and time marks (T) at 10 and 500 msec are shown. The atrium is depolarized by the sinus pacemaker (A), and P waves are upright in the surface ECG leads. Intervals between successive atrial and His-bundle depolarizations are given in msec above the respective electrograms. The first sinus beat is conducted to the His bundle and ventricles, but thereafter the ventricles are controlled by a subsidiary pacemaker located within the His bundle (H). The two pacemakers are firing at similar rates and antegrade and retrograde impulses collide within the A-V node. Events within the A-V node are diagrammatically represented by diagonal lines.

Figure 3

The initiation of SVT. ECG leads 1, 2, and 3, atrial electrogram (A), two His-bundle electrograms (HB), and time marks (T) at 10 and 500 msec are shown. The atrial depolarizations of sinus origin are labeled A, and the first retrograde atrial depolarization (A'). His-bundle depolarizations are labeled H. Diagonal lines represent the events postulated within the A-V node. Intervals between successive atrial and His-bundle depolarizations are given in msec above the appropriate electrogram.

The first two beats recorded occur during a period of A-V dissociation. During the second beat retrograde concealed conduction results in markedly prolonged antegrade A-V nodal conduction of the atrial depolarization of sinus origin and an atrial reentrant beat 630 msec later. Following this beat, A-V nodal reentry is sustained as an episode of SVT. The arrows indicate that the initiating beat of SVT is of sinus origin (upright P waves in the inferior ECG leads), whereas subsequent beats of SVT have a reversed sequence of atrial depolarization (low interatrial septum depolarizes before the high right atrium, and P waves are inverted in the inferior ECG leads). Note that the H-H intervals during the initiation of SVT do not show progressive shortening such as would be expected of the warm-up behavior of an ectopic pacemaker.
reestablished. During catheterization, however, 25 episodes of A-V dissociation were terminated by a regular supraventricular tachycardia with a cycle length between 480 and 520 msec (fig. 3).

Episodes of SVT were always and only initiated by atrial depolarizations of sinus origin during periods of A-V dissociation. As can be seen in figure 3, a sinus beat (P wave and intraatrial conduction identical to other sinus beats) follows His-bundle depolarization by 110 msec, and an episode of SVT is initiated. During the initiation of 25 spontaneous episodes of SVT the sinus beat followed His-bundle depolarization by an interval ranging between 60 and 160 msec. All sinus beats that followed His-bundle depolarization by this interval resulted in episodes of SVT. Sinus beats initiating SVT demonstrated dramatically prolonged A-V-nodal conduction (395 to 500 msec) and resulted in atrial reentry 600 to 690 msec later. The initial reentrant beat was characterized by a reversed sequence of atrial depolarization; that is, depolarization of the low interatrial septum preceded depolarization of the high right atrium, and P waves were deeply inverted in standard ECG leads II and III. Reentry was then sustained for a variable number of beats (4 to 100) and was manifested by a regular tachycardia with normal QRS configuration at cycle lengths of 490 to 530 msec. Spontaneous atrial premature beats were never seen on routine electrocardiograms or during catheterization. SVT was not initiated during measurement of the A-V refractory periods, because following even very premature stimulated atrial depolarizations the A-H interval did not become sufficiently prolonged (the longest A-H interval was 380 msec, 10 to 20 msec shorter than the shortest A-H interval of sinus beats initiating SVT).

His-bundle premature beats were similarly never observed. Although enhanced automaticity of a spontaneous His-bundle pacemaker was manifested during periods of A-V dissociation, its firing cycle length varied between 690 and 900 msec. Shortening of the H-H interval to cycle lengths between 450 and 530 msec occurred only when a sinus beat depolarized the atrium 60 to 160 msec after a His-bundle depolarization. After a series of His-bundle depolarizations at short cycle lengths (SVT) the subsequent H-H interval abruptly assumed the value it had previously been.

Discussion

Three hypotheses might be advanced to explain the mechanism responsible for this patient's paroxysmal tachycardia: (1) atrial reentry via the A-V node, (2) ventricular reciprocal beating, and (3) His-bundle tachycardia. Each hypothesis will be considered.

Atrial reentry has been demonstrated to depend upon prolonged A-V conduction in isolated tissues,9 animal preparations,10 and patients with paroxysmal SVT.2, 4-6 In patients with paroxysmal SVT the requisite prolongation of A-V conduction occurs when spontaneous atrial extrasystoles arise within the relative refractory period of the A-V node.4, 5 In these same patients, paroxysmal SVT can be precipitated by prolonged A-V conduction subsequent to single stimulated APDs evoked when the A-V node is relatively refractory4, 5 or by prolonged A-V conduction produced by pacing the atria at rates sufficient to result in A-V-nodal Wenckebach cycles.6

Premature depolarization of the His bundle and concealed retrograde conduction has been demonstrated to result in various degrees of A-V conduction delay.11 Damato and co-workers12 have demonstrated in animal studies that the interval between premature His-bundle depolarizations and the subsequent atrial beat of sinus origin will determine the antegrade conduction time of that sinus beat. Since the initiation of SVT in patients with that arrhythmia depends primarily upon sufficient A-V-nodal conduction delay, retrograde concealed conduction could provide the conditions necessary for the initiation of SVT.6 In this patient there is considerable evidence to demonstrate that A-V-nodal conduction delay sufficient to allow atrial reentry and episodes of SVT results from concealed retrograde conduction.
Concealed retrograde conduction and atrial reentry. The antegrade A-V nodal conduction time in msec of the sinus beat initiating SVT ($A_s-H'$) is plotted as a function of the interval in msec between the preceding His-bundle depolarization and that sinus beat ($H-A_s$) for nine episodes of SVT occurring in rapid succession. A reverse relationship can be seen in which the more closely coupled is the initiating sinus beat, the longer is its subsequent A-V-nodal conduction time. This suggests that $H'$ is conducted from $A_s$ and the episode of SVT results from reentry initiated by the markedly prolonged antegrade conduction time across the A-V node.

(1) SVT occurred only during periods of A-V dissociation; and during A-V dissociation SVT resulted only when the atrial depolarization of sinus origin followed His-bundle depolarization by 60 to 160 msec. At shorter intervals, antegrade and retrograde impulses collided within the A-V node. (2) The capacity for retrograde conduction via the A-V node was demonstrated when catheter-induced ventricular premature depolarizations resulted in ventriculoatrial conduction. Ventricular extrasystoles never resulted in tachycardia. Despite our interest in the effects of stimulated VPCs during sinus rhythm, in the patient's interest this was not undertaken because of the very frequent episodes of SVT, the variable R-R interval, and the fear of engendering a sustained ventricular arrhythmia. (3) During nine episodes of SVT occurring in rapid succession, the antegrade conduction time of the sinus beat initiating SVT was inversely related to the interval between the dissociated His-bundle beats and atrial depolarizations of sinus origin (fig. 4). Thus, the prolongation of antegrade conduction was demonstrated to be related to the degree of retrograde concealment.

There are other similarities between the arrhythmia documented in this patient and patients with more classically initiated paroxysmal SVT. (4) SVT only followed atrial depolarizations that had significantly prolonged A-V-nodal conduction. (5) During SVT, the sequence of atrial depolarization was from low interatrial septum to high right atrium—that is, consistent with depolarization expected of atrial beats reentering via the A-V node. (6) SVT terminated when A-V-nodal conduction was not sufficiently prolonged, or became too prolonged, to sustain reentry. (7) The relation between P waves and QRS complexes in the surface ECG was not a clue as to the mechanism of this tachycardia.

The possibility that this tachycardia resulted from ventricular reciprocal rhythm may be excluded. In both animal and human studies,13-15 ventricular echoes (reciprocal beating) can be shown to depend upon markedly prolonged ventriculoatrial conduction of the ventricular beat initiating the echoing response. If one assumed that the His-bundle depolarization during A-V dissociation immediately preceded each episode of tachycardia conducted retrograde to the atrium, retrograde conduction times would vary from 60 to 160 msec. These ventriculoatrial conduction times are so short that it is difficult to conceive of how reentry could occur. In addition, the P wave of the beat initiating the tachycardia was upright in every instance, whereas all subsequent P waves during the tachycardia were inverted (fig. 3).

The possibility that this arrhythmia results from an ectopic rapid pacemaker in the His bundle (A-V junction) must be considered in careful detail. Evidence of augmented automaticity within the His bundle exists in this patient. During episodes of A-V dissociation, the rate of the subsidiary (His bundle) pace-
SUPRAVENTRICULAR TACHYCARDIA

maker is relatively rapid (cycle length 680 to 900 msec). In addition, this pacemaker emerges promptly when the sinus rate slows. But, is this subsidiary pacemaker responsible for the episodes of tachycardia? Several considerations exclude this possibility.

(1) H-H intervals dramatically shorten only after a sinus beat has achieved a specific relation to His-bundle depolarization during A-V dissociation. There is no reason why an ectopic pacemaker should fire (or speed) only when this condition obtains. (2) The H-H interval during tachycardia is not half that during A-V dissociation: the His-bundle rate during A-V dissociation does not represent 2:1 exit block from a more rapidly firing His-bundle pacemaker. (3) The H-H interval during the first few beats of tachycardia does not show progressive shortening such as would be expected from the warm-up of an ectopic pacemaker. (4) At the termination of tachycardia, the H-H interval suddenly assumes the value it had immediately prior to the episode of tachycardia.

All these suggest that the tachycardia is an independent rhythm superimposed upon a stable degree of His-bundle automaticity.

References
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