The Essential Role of Atrioventricular Conduction Delay in the Initiation of Paroxysmal Supraventricular Tachycardia

By BRUCE N. GOLDBREYER, M.D., AND ANTHONY N. DAMATO, M.D.

SUMMARY
Studies have shown that in patients with paroxysmal supraventricular tachycardia (SVT), spontaneous or stimulated atrial premature depolarizations (APD) falling within a specific portion of the relative refractory period of the atrioventricular (A-V) conduction system initiate SVT. The present study was designed to determine whether the A-V nodal conduction delay these APDs exhibit, or their coupling interval, is essential for the initiation of SVT. Multiple episodes of SVT were initiated and terminated by single APDs in four patients with a history of paroxysmal SVT. The atria were then paced at numerous fixed rates in excess of the spontaneous sinus rate. In each patient, at atrial rates where progressive A-V nodal conduction delay occurred from beat to beat (Wenckebach cycles), a specific degree of A-V nodal conduction delay always resulted in atrial reentry or SVT. Although the atrial coupling interval was considerably longer during Wenckebach cycles initiating SVT than for single APDs resulting in the arrhythmia, the prolongation of A-V nodal conduction was identical. Independent of atrial coupling interval, a requisite degree of A-V nodal conduction delay always resulted in atrial echoes or SVT. This supports the conclusion that SVT results from atrial reentry via the A-V node.

Additional Indexing Words:
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Atrial echoes Atrial reentry His bundle electrogram A-V Wenckebach cycles
Atrial pacing

THE clinical association between atrial premature beats and paroxysmal supraventricular tachycardia (SVT) is well recognized.1, 2 These spontaneous atrial premature beats were initially thought to be evidence of an atrial automatic pacemaker,3-6 but recent clinical studies have suggested they may initiate SVT by a reentrant mechanism.7-12 In a previous study of nine patients with a history of paroxysmal SVT and no evidence of the Wolff-Parkinson-White syndrome, SVT was demonstrated to result from spontaneous or stimulated atrial premature depolarizations (APD) which occur during a specific portion of the atrial cycle—the "echo" zone.13 Atrial reentry via the A-V node was demonstrated to be the mechanism initiating and sustaining SVT in these patients.14

The present study was undertaken to determine the essential condition by which any atrial beat results in atrial reentry or SVT. The prolonged A-V nodal conduction of atrial depolarizations resulting in atrial reentry and SVT, could be analyzed apart from their coupling interval by atrial pacing at rates sufficient to produce A-V nodal Wenckebach cycles. In our patients, sufficient prolongation of A-V nodal conduction resulted in SVT independent of the atrial coupling interval.
The sequence of atrial depolarization during SVT was also analyzed.

Methods

Four patients were referred to the cardiology division because of a history of paroxysmal rapid regular palpitations and electrocardiographic documentation of supraventricular tachycardia. The patients’ ages ranged from 20 to 55 years, and the history of palpitations antedated referral by 2 to 15 years. None of the patients had historical, physical, electrocardiographic, or radiologic evidence of heart disease. None had the Wolff-Parkinson-White syndrome. One patient (age 55) had recently noted anterior chest pain during episodes of tachycardia, but did not have angina on exertion. Routine laboratory studies were within normal limits for all patients.

Patients were brought to the cardiac catheterization laboratory in the post-absorptive, nonsedated state. None was receiving any medication at the time of catheterization. By standard techniques, a quadrupolar catheter was advanced via the right basilic vein into the right atrium. The electrodes were positioned along the lateral border of the atrium, so that the two most proximal electrodes were adjacent to the region of the sinoatrial node. The distal electrodes were used to stimulate the mid-portions of the right atrium. A tripolar catheter was then advanced via the right femoral vein, and a His bundle electrogram obtained.15

The bipolar atrial electrogram was used to trigger an oscilloscope which triggered a single wave form and pulse generator. Atrial premature stimuli of 1.5-msec duration and twice diastolic threshold were introduced during every tenth sinus cycle. A variable delay potentiometer on the pulse generator allowed atrial premature stimuli to be made progressively more premature. A-V refractory periods were then measured using the extra-stimulus method.11 Numerous episodes of SVT were initiated in each patient by single atrial premature stimuli. Episodes of SVT were terminated using the same triggered stimulating apparatus. With the wave form generator operating in the recurrent mode, the atria were paced at various fixed rates in excess of the sinus rate.

Electrocardiographic leads I, II, and III, bipolar atrial electrogram, His bundle electrogram, and time marks at 10 and 100 msec were observed on a separate switched beam oscilloscope and recorded on magnetic tape. The analog tracings were subsequently reproduced on a photographic recorder and electrograms displayed at frequency settings of 40–500 Hz.

The method by which A-V refractory periods were measured and SVT initiated. In each panel are recorded: atrial electrogram (A), His bundle electrogram (H), ECG lead I (I), and time marks at 10 and 100 msec. The heavy vertical bar aligns the tenth atrial beat of sinus origin (A1) prior to the introduction of the stimulated atrial premature beat (A2). The A1A2 interval is given above each atrial electrogram in msec. Stimuli are indicated by arrows. The His bundle depolarizations conducted from A1’s are labeled H2, and the A-H intervals given in msec on the diagonal lines. (Panel A) At an A1A2 interval of 400 msec the A2H2 interval is prolonged to 235 msec. A2 was evoked during the relative refractory period of the A-V node. (Panel B) The A1A2 interval is decreased to 365 msec and A2H2 increases appropriately to 275 msec. (Panel C) The A1A2 interval is further decreased to 330 msec. A2H2 is dramatically prolonged (300 msec) and an episode of SVT is initiated.

Definition of Terms

A1A2 is the interval between an atrial depolarization of sinus origin (A1) and a stimulated atrial premature depolarization (A2).

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$A_2H_2$ is the A-H interval of the stimulated premature depolarization.

$H-V$ is the interval between any His bundle depolarization and the ventricular depolarization conducted from it.

**Effective refractory period of the A-V node** is the shortest $A_1A_2$ interval where $A_2$ propagates to the His bundle.

**Relative refractory period of the A-V node** is the group of $A_1A_2$ intervals where $A_1H_2$ exceeds the A-H interval of the sinus impulse.

**Results**

In all patients the His bundle electrogram was easily recorded. His bundle depolarization was recognized as a rapid deflection during the isoelectric P-R segment of the

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**Figure 2**

The A-V nodal conduction delay of stimulated APDs is plotted as a function of their premature for four patients. In each graph, $A_1A_2$ is the coupling interval of single stimulated APDs introduced during sinus rhythm. $A_2H_2$ is the A-V nodal conduction delay of these stimulated APDs. Numerical values on both ordinate and abscissa are in msec. The small dots (+) indicate single APDs introduced during the relative refractory period of the A-V node; as the coupling interval is decreased, the A-H interval progressively increases. The large dots (*) indicate APDs which resulted in episodes of SVT. The thin vertical line in each graph indicates the atrial coupling interval which almost uniformly resulted in episodes of SVT in that patient (outer limit of the "echo zone"). The thin horizontal lines indicate the A-H interval beyond which A-H prolongation almost uniformly resulted in SVT. Episodes of SVT initiated by single APDs therefore fall within the upper left-hand quadrant formed by these lines.

The large open circles (o) indicate beats during atrial pacing at a fixed rate which resulted in episodes of SVT. The atrial cycle length is plotted on the abscissa, the A-H interval of the beat initiating SVT on the ordinate. In all four graphs the interval between successive atrial beats (coupling interval) leading to Wenckebach cycles and SVT is longer than the longest coupling interval of single APDs resulting in SVT, i.e., falls to the right of the thin vertical lines. On the other hand, the A-V nodal conduction delay of the beat initiating SVT during A-V nodal Wenckebach cycles is uniformly above the thin horizontal lines. There is a minimum A-H interval required for reentry and SVT. The fact that the cycle lengths of atrial depolarizations initiating SVT during Wenckebach cycles (o) fall in the upper right quadrant of each graph demonstrates that the essential condition for the initiation of SVT is a specific degree of A-V nodal conduction delay.

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surface electrocardiogram. During sinus rhythm, A-H and H-V intervals were within normal limits for all patients. Refractory periods of the A-V conduction system were measured during sinus rhythm using a modification of the extra-stimulus method. As the coupling interval of the premature atrial beats (A1A2) was progressively decreased, a progressive prolongation of the A2H2 interval (A-V nodal conduction delay) was encountered. This is illustrated in figure 1. The most refractory component of the entire A-V conduction system was the A-V node in all patients, and blocked atrial premature depolarizations (APD) failed to conduct to the His bundle. In all patients, the effective refractory period of the A-V node was less than 320 msec and within normal limits.16 Conduction delay within the ventricular specialized conduction system was not encountered; the H-V interval remained constant.

APDs introduced during a specific portion of the relative refractory period of the A-V node resulted in reentrant beats and episodes of SVT identical to those observed to arise spontaneously in each patient prior to the time of study (fig. 1). Similarly, appropriately timed single APDs introduced during SVT resulted in termination of the arrhythmia. An initial "instability" of atrial cycle length was observed at the initiation of SVT. The initiating cycles of SVT suggested a reciprocal relationship between the cycle initiating SVT and the first atrial cycle length, but values were insufficient for statistical analysis. Carotid sinus massage terminated SVT by causing progressive increase in A-H interval and, eventually, block within the A-V node.

In our patients, when the A-V nodal conduction delay of stimulated APDs (A2H2 interval) was plotted as a function of the prematurity with which these APDs were evoked (A1A2 interval), we could demonstrate that SVT was uniformly initiated by APDs evoked with a specific degree of prematurity—the "echo" zone. However, within the "echo" zone the A2H2 interval always exceeded a specific value (fig. 2).

After SVT had been initiated and terminated by single APDs several times in each patient, fixed rate atrial pacing was begun at a rate in excess of the spontaneous sinus rate. As the atrial paced rate was increased, progressive but stable prolongation of the A-H

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**Figure 3**

Atrial pacing at a fixed cycle length of 470 msec resulted in SVT. Recorded from top to bottom are ECG leads I, II, and III (1, 2 and 3), atrial electrogram (AE), two His bundle electrograms (HBE), and time marks (T) at 10 and 100 msec. Atrial pacing stimuli are indicated by arrows. His bundle depolarizations are labeled H. Atrial stimulated beats (A1) at CL 470 result in progressive A-H prolongation (150, 180, 195 msec). The third paced beat results in atrial reentry and initiates SVT. The fourth atrial beat is a fusion beat labeled (A2). The next stimulus is ineffective and stable SVT is maintained. Atrial depolarizations during SVT are labeled A1. It can be seen that during SVT in this patient, the low interatrial septum depolarizes significantly ahead of the high right atrium (the thin vertical line in the next to last atrial depolarization).
interval was demonstrated. In each patient a paced atrial cycle length was reached where, at a constant cycle length, the A-H interval showed progressive prolongation from beat to beat. Atrial reentry or SVT uniformly resulted (fig. 3) when the A-H interval reached or exceeded the A-H interval required for reentry of single APDs. During Wenckebach cycles, the atrial cycle length (A-A interval) at which A-V nodal conduction prolonged sufficiently to result in SVT was consistently longer than the longest coupling interval of single APDs resulting in SVT (fig. 2). In one patient, progressive A-H prolongation (A-V nodal Wenckebach) could be achieved over a wide range of paced atrial cycle lengths (450–730 msec). At each rate, independent of the atrial coupling interval, as soon as the A-H interval reached or exceeded 160 msec, reentry or SVT resulted (fig. 2, panel 4).

During sinus rhythm in all patients the high right atrium depolarized 10–30 msec before the low interatrial septum as recorded in the His bundle electrogram. The sequence of atrial depolarization during SVT could be analyzed in two patients. In one, P waves occurred between QRS complexes (i.e., the A-H interval was only moderately prolonged during SVT), and low interatrial septum depolarized significantly before the high right atrium (fig. 3).

In the other three patients, markedly prolonged A-H intervals during SVT resulted in almost simultaneous depolarization of atria and ventricles. As a result, in the His bundle electrogram, atrial depolarization was obscured by ventricular depolarization. The sequence of atrial depolarization during SVT was fortuitously observed in one of these patients during the administration of procainamide. After 500 mg of procainamide had been infused over a period of 25 min during sustained SVT, a brief period of 2:1 block within the ventricular specialized conduction system (below the His bundle) was observed. During this period, the absence of ventricular depolarizations following every other His bundle depolarization showed the low inter-

**Discussion**

**Coupling Interval or Conduction Time?**

Independent of their analysis of the mechanism of SVT, all investigators have recognized the clinical association of atrial extrasystoles (atrial premature depolarizations, APDs) with paroxysmal supraventricular tachycardia. Some have felt these spontaneous APDs indicated atrial automaticity and, therefore, that SVT resulted from a repetitive rapid automatic focus.3–6 Others have felt these APDs might initiate SVT through a reentrant mechanism.7–12 Recent observations in nine

![Figure 4](http://circ.ahajournals.org/content/full/43/3/683/F1.large.jpg)

The sequence of atrial depolarization during SVT is analyzed during a period of 2:1 block below the His bundle. Recorded are: ECG leads, I, II, and III (1, 2, and 3), atrial electrogram (AE), His bundle electrogram (HBE), and time marks at 100 msec. Atrial depolarization in the atrial electrogram is labeled A1; His bundle depolarization, H; and ventricular depolarizations, V. In the first SVT beat, atria and ventricles can be seen to depolarize almost simultaneously, and as a result, atrial depolarization is obscured within the HBE. In the second beat, however, the impulse blocks below the His bundle. The absence of ventricular depolarization in the HBE allows the sequence of atrial depolarization to be analyzed. The arrow indicates when atrial depolarization begins in the low interatrial septum (A). This precedes depolarization of the high right atrium by almost 100 msec. Note that during this beat a P wave can be seen in the surface electrocardiogram. The P wave is biphasic, but predominantly negative in the inferior leads (see discussion).
patients with paroxysmal SVT and no evidence of the Wolff-Parkinson-White syndrome, demonstrated that SVT almost certainly results from atrial reentry via the A-V node.\textsuperscript{13} 14 Observations in our patients conform to those of the previously reported nine cases: (1) SVT was always, and only, initiated by APDs occurring during a specific portion of the relative refractory period of the A-V node; (2) SVT could be terminated by single APDs which blocked within the A-V node and reentrant pathway; and (3) carotid sinus massage terminated SVT by progressive prolongation of A-H interval and eventual failure of conduction within the A-V node.

In several studies, both the coupling interval and P-R interval of APDs initiating reentry have been emphasized.\textsuperscript{12, 17, 18} The “echo” zone was defined as the range of coupling intervals at which APDs uniformly result in either atrial echoes or SVT.\textsuperscript{13} Although it was recognized that the “echo” zone was clearly distinct from the atrial vulnerable period,\textsuperscript{19} there had been no previous attempt to separate the effects of the coupling interval of APDs initiating reentry and SVT, from the prolonged A-V nodal conduction these APDs exhibit.

In the present report, the precise relationship of APDs to A-V nodal conduction was analyzed in patients with SVT. As would be expected from the functional characteristics of the A-V node, the length of the P-R interval of spontaneous or stimulated APDs (fig. 1) is related to their coupling interval.\textsuperscript{20}

The graphs in figure 2 demonstrate that APDs introduced during sinus rhythm, resulting in reentry and SVT, not only have a specific

\textbf{Figure 5}

Three panels demonstrate the relationship between a single APD (A\textsubscript{2}) and fixed rate atrial pacing, both of which result in SVT. In each panel are recorded the atrial electrogram (A), His bundle electrogram (H), ECG lead I (I), and time marks at 10 and 100 msec. (Upper panel) During atrial pacing at a fixed cycle length of 410 msec (A-A is 410 msec), the A-H interval in this patient showed progressive prolongation (not shown in figure). When A-H interval reached 300 msec (the second paced beat in the panel), SVT is initiated and the remainder of the atrial stimuli are ineffective (note atrial depolarizations “marching through” stimulus artifacts). (Lower left) During sinus rhythm a single stimulated APD (A\textsubscript{2}) is introduced with a coupling interval of 410 msec. The A\textsubscript{2}H\textsubscript{2} interval is only 180 msec. Sinus rhythm continues. (Lower right) During sinus rhythm the coupling interval of A\textsubscript{2} is decreased to 330 msec. The A\textsubscript{2}H\textsubscript{2} interval becomes 300 msec and an episode of SVT is initiated.
zone of coupling (A1A2 intervals to the left of the thin vertical lines in these figures), but always demonstrate a specific prolongation of A-H interval (A2H above the thin horizontal lines).

In any patient, pacing the atria at fixed rates in excess of the spontaneous sinus rate prolongs the A-H interval.21 The higher the paced rate, the more prolonged the A-H interval of each paced beat. Atrial pacing at appropriate rates may be used to produce A-V nodal Wenckebach cycles; that is, cycles where the A-H interval shows increasing prolongation from beat to beat until conduction eventually fails within the A-V node.22 In our patients, SVT was initiated in each patient during induced Wenckebach cycles, when the A-H interval exceeded the limit defined by single APDs initiating SVT for that patient (fig. 2). The atrial cycle length (A-A interval) which resulted in SVT during fixed rate atrial pacing was uniformly longer than the longest coupling interval for any single APD initiating SVT (fig. 2), and fell beyond the outer limit of the "echo zone" if defined in terms of coupling interval alone.

For example, in figure 5, atrial pacing at a cycle length of 410 msec resulted in SVT when, during progressive prolongation of A-H interval, the A-H interval reached 300 msec. A single APD introduced with the same coupling interval, 410 msec after a spontaneous sinus beat, had an A-H interval of only 180 msec, and did not result in SVT. In this patient, when the coupling interval of a single APD was decreased to 330 msec, the A-H interval prolonged to 300 msec, and SVT was initiated.

These observations demonstrate that it is the degree of A-V nodal delay (A-H interval) of any atrial beat, and not its coupling interval, which is responsible for the initiation of SVT. The essential condition for the initiation of SVT, therefore, is A-V nodal conduction delay. This strongly supports the conclusion that SVT results from reentry via the A-V node.

A-V nodal Wenckebach periods and atrial reentry are obviously related since both require prolonged A-V nodal conduction for their occurrence. In some patients without a history of SVT, clinical electrocardiograms show that A-V nodal Wenckebach periods may occasionally be terminated by atrial "premature" beats. It has been suggested that this occurrence is more common than would be expected by chance alone and may be due to atrial reentry.2 Our observations support this hypothesis since atrial reentry terminated A-V nodal Wenckebach periods in all our patients.

The Sequence of Atrial Depolarization During SVT

In isolated tissue preparations, single or multiple atrial reentrant beats have been produced by single stimulated atrial premature depolarizations.23 These reentrant beats result from reentry within the A-N region of the A-V node, and result in retrograde atrial depolarization during reentry. In the experimental animal, the sequence of atrial depolarization following atrial premature depolarizations was utilized as evidence that reentry was occurring within the A-V node.24 Clinical electrocardiograms during SVT which demonstrate inverted P waves in the inferiorly directed leads (II and III) have been interpreted as "reentrant" tachycardias solely because the P-wave morphology was consistent with retrograde atrial depolarization.2,12

It has been demonstrated in this and previous studies that paroxysmal SVT in man is the result of atrial reentry via the A-V node. Yet, P waves in the surface electrocardiogram have been inconsistent in their relationship to QRS complexes as well as their morphology.13 In the present study, intra-atrial electrograms were purposely recorded from the high right atrium and low interatrial septum during sinus rhythm and SVT. The expected atrial depolarization sequence (high to low) was observed in all patients during sinus rhythm. Because of the temporal relationship of atrial and ventricular depolarization, the sequence of atrial depolarization during SVT could be analyzed in only two of our patients. In both, during SVT, the low interatrial septum depolarized significantly before the high right atrium. This observation is consistent with the conclusion

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that paroxysmal SVT results from atrial reentry via the A-V node.

**A Diagnostic Test for Paroxysmal SVT**

Our patients all had a history of paroxysmal rapid regular palpitations lasting from minutes to hours. During an episode of typical palpitations, SVT was documented on standard electrocardiograms prior to admission. It is of interest that the frequency of SVT in our patients was such that we did not observe spontaneous SVT during the in-hospital period. Nevertheless, appropriately timed stimulated APDs resulted in SVT in every patient during catheterization. The induced SVT was identical to the previously documented spontaneous arrhythmia.

In our combined experience with over 100 patients without a history of SVT whose A-V refractory periods were measured by introducing progressively premature atrial depolarizations during sinus rhythm, not one ever developed SVT. Yet, in all patients with a history of SVT similarly studied, appropriately timed APDs uniformly resulted in this arrhythmia. This suggests that, in patients with a history suggestive of SVT, documentation of which has been impossible due to the brief duration of palpitations, the methods used here might provide a diagnostic test to demonstrate if SVT is the cause of their clinical symptoms before drug therapy is instituted.

A word of caution is in order. At the present time, this type of testing should probably be limited to those centers where these stimulation techniques are frequently utilized. Secondly, although in 13 consecutive patients with a history of SVT, atrial reentry could be demonstrated to be the mechanism of their arrhythmia, this mechanism is undoubtedly not the cause of all atrial tachyarrhythmias. Some patients may have an ectopic atrial focus which is responsible for their arrhythmia. Were this the case in any given patient, the stimulation sequences described here would not be successful in initiating their tachycardia.

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BRUCE N. GOLDREYER and ANTHONY N. DAMATO

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