Site of Reentry in Paroxysmal Supraventricular Tachycardia in Man

By Bruce N. Goldreyer, M.D., and J. Thomas Bigger, Jr., M.D.

SUMMARY

His bundle recordings during spontaneous and induced episodes of paroxysmal supraventricular tachycardia (SVT) demonstrated that the site of reentry responsible for this arrhythmia was in the A-V node. In five patients studied, A-V conduction time and refractory periods were normal, but atrial premature depolarizations (APD) within a portion of the relative refractory period of the A-V node consistently produced atrial reentry—echoes or sustained SVT. In two patients, APDs falling within the A-V effective refractory period failed to excite the His bundle but still reentered within the A-V node and began SVT. It was noted that the various relationships between atrial and ventricular depolarizations on the surface ECG did not reflect different mechanisms for SVT, but only variations in A-V nodal conduction time.

THERE IS A long-standing controversy as to whether the mechanism of paroxysmal supraventricular tachycardia (SVT) in man is caused by an ectopic pacemaker or by reentry. Recent studies strongly suggest that the mechanism for this arrhythmia is most often reentry. However, the site of reentry has not been demonstrated.

Intraatrial reentry was postulated as early as 1923. This site seemed unlikely because the rate of SVT is much slower than would be predicted from the conduction velocity in ordinary atrial tissue. Barker, Wilson, and Johnston overcame this objection by postulating that the reentrant pathway responsible for SVT might include either the sinoatrial or atrioventricular nodes. They pointed out that the relatively slow conduction velocity of either node would account for the observed rate of SVT.

In the present study, recordings of His bundle depolarization in a group of patients with SVT demonstrated that the site of reentry lies within the A-V node.

Methods

We studied nine consecutive patients who were referred for evaluation of paroxysmal SVT. Recordings of His bundle depolarization were attempted and obtained in the last five patients. Four of these five patients had no evidence of heart disease by history, physical examination, chest X-ray, or electrocardiogram. The fifth patient with ankylosing spondylitis had minimal aortic insufficiency but no evidence of congestive
heart failure. The patients' ages ranged between 19 and 58 years, and all had a long history of paroxysmal palpitations. No patient was receiving any medication at the time of study, and results of pertinent laboratory examinations were normal. In each patient, a paroxysmal regular SVT was recorded during a brief period of in-hospital monitoring (fig. 1).

Patients were brought to the catheterization laboratory in the nonsedated, postabsorptive state. With the use of sterile technique and local procaaine anesthesia, a single bipolar catheter was introduced via the right basilic vein under fluoroscopic and electrocardiographic control to a position high in the right atrium, adjacent to the region of the sinoatrial node. A second electrode catheter was introduced via the saphenous vein into the right atrial appendage for use as a stimulating catheter.

A decapolar catheter of our design was similarly introduced through the right saphenous vein and positioned fluoroscopically so that its tip was within the right ventricular inflow tract. Accordingly, the electrodes were distributed across the A-V valve so that one pair "bracketed" the His bundle. Recordings from each adjacent pair of electrodes were then scanned until His bundle depolarization was identified as a sharp depolarization occurring within the isoelectric portion of the PR segment of the surface electrocardiogram.

A bipolar atrial electrogram, a His bundle electrogram, and a surface electrocardiogram were simultaneously observed on a switched beam oscilloscope and recorded, unfiltered, on magnetic tape. When transferred to photographic paper for analysis, the His bundle recording was filtered with frequency settings of 40 and 500 Hz.

Atrioventricular refractory periods were measured by previously described stimulating and recording techniques.\(^1\) \(^2\) \(^8\) Recordings were made in all patients during: (1) spontaneous episodes of SVT, (2) SVT induced by stimulated atrial premature depolarizations (APD), (3) the termination of episodes of SVT by single stimulated APD, and (4) carotid sinus massage.

**Results**

In all five of these patients, the His bundle electrogram was easily recorded. His bundle depolarization was identified as a rapid deflection in the isoelectric PR segment of the surface electrocardiogram; it was verified during fixed rate atrial pacing and measurement of the A-V refractory periods when the interval between atrial and His bundle depolarizations increased appropriately. His bundle pacing was not attempted because of the high stimulus amplitudes required.\(^9\)

Atrial echoes and spontaneous episodes of SVT were observed in each patient while atrial electrogram, His bundle electrogram, and surface electrocardiogram were recorded. Previously established criteria supporting a reentrant mechanism in the initiation and persistence of these SVTs\(^8\) were satisfied in all five patients: (1) spontaneous episodes of SVT were always and only initiated by APDs arising within the relative A-V refractory period; (2) the capacity for ventriculoatrial conduction was present; (3) spontaneous atrial echoes were observed, and the sequence of atrial and ventricular depolarizations closely paralleled that seen during the initiation of SVT; (4) electrically stimulated APDs evoked

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**Figure 1**

*Six-lead standard electrocardiogram demonstrates the onset of SVT in patient A.Y. The third beat during sinus rhythm (NSR) is an atrial premature depolarization (APD) which does not fall within the relative refractory period of the A-V conduction system, i.e., the PR interval is no longer than that of the preceding sinus beats. At the arrow, a spontaneous APD occurs early in the relative A-V refractory period, demonstrates markedly prolonged A-V conduction, and initiates an episode of SVT. The first two beats of SVT are conducted aberrantly.*

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*GOLDFREYER, BIGGER*
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Table 1
Characteristics of A-V Conduction and SVT

<table>
<thead>
<tr>
<th>Patients</th>
<th>O.F.</th>
<th>A.P.</th>
<th>A.Y.</th>
<th>J.B.</th>
<th>T.P.</th>
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<tr>
<td>NSR CL (msec)</td>
<td>590</td>
<td>565</td>
<td>830</td>
<td>715</td>
<td>750</td>
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<tr>
<td>A-H (msec)</td>
<td>115</td>
<td>95</td>
<td>90</td>
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<td>85</td>
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<tr>
<td>H-V (msec)</td>
<td>45</td>
<td>45</td>
<td>45</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>P-R (msec)</td>
<td>160</td>
<td>140</td>
<td>140</td>
<td>130</td>
<td>135</td>
</tr>
<tr>
<td>AVRP CL (msec)</td>
<td>600</td>
<td>600</td>
<td>600</td>
<td>500</td>
<td>600</td>
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<tr>
<td>FRP (msec)</td>
<td>378</td>
<td>340</td>
<td>384</td>
<td>375</td>
<td>380</td>
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<td>ERP (msec)</td>
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<td>247</td>
<td>310</td>
<td>&lt;230</td>
<td>242</td>
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<tr>
<td>SVT rate (beats/min)</td>
<td>190 176</td>
<td>176 187</td>
<td>146 185</td>
<td>158</td>
<td></td>
</tr>
<tr>
<td>CL (msec)</td>
<td>315 340</td>
<td>340 320</td>
<td>410 325</td>
<td>380</td>
<td></td>
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<td>A-H (msec)</td>
<td>270 150</td>
<td>265 150</td>
<td>115 250</td>
<td>260</td>
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<tr>
<td>H-V (msec)</td>
<td>45 45</td>
<td>45 45</td>
<td>45 50</td>
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<td></td>
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<td>P to QRS relationship</td>
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<td>P S S</td>
<td></td>
<td></td>
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<td>Echo zone duration (msec)</td>
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<td>290-370 230-470</td>
<td>220-310</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Echo zone duration (msec)</td>
<td>60 80 240 90</td>
<td></td>
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</table>

Abbreviations: S = simultaneous; P = precedes; CL = cycle length; AVRP CL = cycle length at which atrioventricular refractory periods were measured; FRP = functional refractory period; ERP = effective refractory period; SVT = supraventricular tachycardia.

during a specific portion of the relative A-V refractory period always resulted in atrial echoes or episodes of SVT; (5) the first and second atrial cycles of SVT were reciprocally related, and there tended to be an initial oscillation of atrial cycle lengths during SVT; (6) single or multiple atrial responses evoked by stimuli within the atrial “vulnerable” period never resulted in episodes of SVT; (7) the cycle length of SVT was a direct linear function of the refractoriness of the A-V conduction system; (8) during sustained episodes of SVT, appropriately timed single APDs terminated SVT.

During sinus rhythm, recordings of His bundle depolarization allowed A-V conduction to be more specifically characterized. Conduction across the A-V node, as determined from the interval between the initiation of atrial and His bundle depolarizations (A-H interval), was normal in all patients (table 1). Conduction in the ventricular specialized conduction system (the interval between His bundle and ventricular depolarizations, or H-V interval) was normal. In each patient, very premature atrial depolarizations were blocked above the His bundle, i.e., in the A-V node. Therefore, the effective and functional refractory periods listed in table 1 characterize the refractoriness of the A-V node. In each patient, A-V nodal refractoriness was normal.

During the initiation of spontaneous and stimulated episodes of SVT, the markedly prolonged A-V conduction necessary for atrial reentry always occurred within the A-V node (fig. 2). The H-V interval was never longer during SVT than during sinus rhythm or atrial pacing. A-V nodal conduction times varied during sustained SVT, but were always longer than during sinus rhythm (figs. 2 to 4). In two patients (A.P. and A.Y.) atrial echoes and episodes of SVT were initiated by APDs which failed to propagate to the bundle of His (fig. 3); in these same two patients, SVT was also produced by APDs that did conduct to the bundle of His (fig. 2). These observations suggest that activation of the His bundle is not a prerequisite for atrial reentry, and, therefore, the site of reentry must lie above the His bundle, i.e., within the A-V node.

The atrial cycle length and the A-H and H-V intervals during SVT are shown for each patient in table 1. The A-H intervals of APDs initiating SVT varied in individual patients depending upon the degree of prematurity of the APD; in all patients, however, this A-H interval was prolonged when compared to sinus cycles. In every case, it was longer than...
His bundle activity during the spontaneous and stimulated onset of SVT (patient A.Y.).

Atrial electrogram (AE), His bundle electrogram (HBE), and surface electrocardiogram (ECG) are recorded. Intervals in msec between successive atrial depolarizations are given above each atrial electrogram. AV conduction times in msec are given on each diagonal line. The heavy vertical bar aligns the last atrial depolarization prior to an APD; one atrial cycle appears to the left of the bar. The shaded area highlights the prolonged conduction of each APD.

A. During sinus rhythm, cycle length is 890 msec, A-H interval is 90 msec, H-V interval is 45 msec. A spontaneous APD occurs 365 msec after the sinoatrial beat and shows a prolonged A-H intercal (130 msec), indicating the APD occurred during the A-V nodal relative refractory period. The H-V interval is unchanged. Atrial reentry does not occur, and sinus rhythm resumes after a 940-msec interval.

B. A spontaneous APD at a 285-msec interval exhibits prolonged A-V conduction (A-H interval, 265 msec); the H-V interval remains constant. Atrial reentry occurs 400 msec after the APD, and SVT is initiated. During subsequent beats of SVT, the A-H interval progressively shortens to 110 msec but remains longer than during sinus rhythm. The first three beats of SVT have a right bundle branch block configuration-rate-related aberration.

C. During atrial pacing at a cycle length of 610 msec, the A-H interval is 110 msec and the H-V interval, 50 msec. Stimuli are indicated by arrows. An APD at a 325-msec interval shows a markedly prolonged A-H interval (230 msec), whereas the H-V interval again remains constant. The prolonged A-V nodal conduction time allows atrial reentry to occur 450 msec after the APD, and a sustained episode of SVT is initiated.
His bundle activity during the spontaneous and stimulated onset of SVT. The format is the same as in figure 2.

A. During sinus rhythm, the A-H interval is 90 msec and the H-V interval, 45 msec. A spontaneous APD arises 300 msec after the sinoatrial beat, indicated by the vertical bar. The APD falls within the effective A-V nodal refractory period and fails to conduct to the His bundle or ventricle. Nonetheless, prolonged conduction within the A-V node results in a reentrant atrial beat 440 msec later. Only a single atrial echo results, because the A-H interval of the reentrant beat (95 msec) is not prolonged sufficiently for reentry to continue. Sinus rhythm resumes.

B. A spontaneous APD at a longer interval (340 msec) still falls within the A-V nodal effective refractory period (fails to reach the His bundle). Atrial reentry occurs 350 msec after the APD. The shorter interval between the APD and the first beat of SVT results in prolonged A-V nodal conduction of the atrial reentrant beat (A-H interval, 120 msec). Reentry continues as SVT.

C. During atrial pacing, a stimulated APD 285 msec after the atrial paced beat fails to conduct to the His bundle, and SVT is initiated. A stimulus 445 msec after the first reentrant beat depolarizes the atrium 5 to 15 msec earlier than expected, but reentry is maintained, and SVT continues.

The relationship between atrial and ventricular depolarizations in reentrant SVT is dependent upon the A-H interval (fig. 4). When A-H intervals are long, the P waves are superimposed upon the QRS complexes; when

the subsequent A-H intervals during SVT. In some individuals the A-H intervals during SVT quickly became constant; in others, this process took a number of beats (fig. 2). The H-V interval always remained constant.
A-H intervals are shorter, P waves precede QRS complexes on the surface electrocardiogram (fig. 5). This was most strikingly demonstrated in two patients (O.F., and A.P.) when during sustained SVT, the A-H interval varied in such a way that the P waves assumed both relationships to the QRS complexes (figs. 6 and 7). In figure 6, after block within the A-V node, the next A-H interval is markedly shortened and subsequent A-H intervals become stable at an intermediate interval. In figure 7, for no apparent reason, the A-H interval progressively lengthens to assume a new stable and markedly longer A-V nodal conduction time.

The electrophysiologic events, occurring at the termination of episodes of SVT by either stimulated APDs or carotid sinus massage, were clarified by His bundle recordings. Stimulated APDs which terminated SVT always showed A-H intervals that were dramatically prolonged in comparison with those during SVT (fig. 8). During SVT, the entire atrial cycle was explored with APDs induced by electrical stimuli at 10-msec intervals. In figure 8, successively more premature stimulated APDs are shown. When stimuli are introduced late in the atrial cycle, the A-H interval lengthens but reentry is sustained and SVT continues. APDs intro-
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The A-H intervals during stable episodes of SVT are plotted and identified with patients' initials. During SVT, when the P preceded the QRS on the surface ECG, the A-H intervals were between 100 and 150 msec (circles). When during SVT, P and QRS were superimposed, A-H intervals were uniformly greater than 250 msec (squares).

produced early in the atrial cycle propagated with very long A-H intervals and ended SVT (see Discussion). Moreover, when SVT was terminated by carotid sinus massage, progressive lengthening of the A-H interval was observed without significant alteration in atrial cycle length (fig 9). When the A-H interval became markedly prolonged, SVT was terminated.

Discussion

We demonstrated in a previously studied group of patients that paroxysmal SVT is caused by reentry in the A-V conduction system. The patients discussed here were all studied in the same manner, and satisfied all our previously described criteria for reentry as the mechanism of their SVT. In the present study, we used recordings from the bundle of His to define more precisely the characteristics of conduction in the A-V conduction system (AVCS) of patients with SVT, and to localize the site of reentry within the AVCS during sustained SVT.

The events occurring within the several components of the AVCS take place during the time span of the PR interval of the surface electrocardiogram, i.e., the interval between the initiation of atrial and ventricular depolarizations. The PR interval is composed of overlapping time periods representing intra-atrial conduction, delay at the upper margin of the A-V node, conduction across the A-V node, and propagation through the bundle of His and ventricular specialized conduction system. By recording the initiation of His bundle depolarization within the PR interval, we can divide A-V conduction into two segments—an interval representing atrial and A-V nodal conduction time and an interval reflecting transmission within the ventricular specialized conduction system.

His bundle recordings from the intact human heart were first obtained by Giraud et al. in 1960, and by Watson et al. in 1967. These investigators thought His bundle depolarization might be easily and safely recorded in normal patients during cardiac catheterization. A-V conduction may be more precisely characterized with this technique, and in several reports the site of block within the AVCS was delineated. V-A conduction was also studied by recordings from the bundle of His. We felt that the ability to separate A-V nodal conduction delay from delay within the ventricular specialized conduction system could be useful in the analysis of the site of reentry responsible for SVT.

Reentry has been suggested as a possible mechanism for paroxysmal tachycardias since 1913. Barker, Wilson, and Johnston suggested that the clinical and electrocardiographic feature of SVT could best be explained by assuming that, in SVT, reentry occurred within the sinoatrial or A-V nodes. Laboratory studies have shown that in atrial echoes reentry occurs within the A-V node. In a laboratory study of SVT in a single dog, reentry occurred within the AVCS. The site was not demonstrated, but it was postulated to be within the A-V node. We demonstrated...
The variable relationship between atrial and ventricular depolarizations during SVT.

A. During sinus rhythm, the A-H interval is 90 msec.

B. During one episode of SVT, the A-H interval was markedly prolonged (280 msec). This caused atrial and ventricular depolarizations to occur simultaneously on the surface ECG. This is emphasized by the dashed vertical bar.

C. During another episode of SVT, the A-H interval was longer than in sinus rhythm but shorter than the A-H interval in the episode of SVT shown in B. As a result, atrial depolarization precedes ventricular depolarization on the surface ECG.

D. The mechanism of SVT is the same as in B and C. During an episode of sustained SVT with a very prolonged A-H interval (280 msec), the fifth atrial depolarization propagates through the site of reentry in the A-V node but is blocked above the His bundle. The reentrant tachycardia continues, but the A-H interval following this blocked beat is considerably shortened (110 msec). The second dashed vertical bar indicates the beginning of a period of progressive A-V nodal conduction delay during which the relationship between atrial and ventricular depolarizations is constantly changing. The A-H interval (150 msec) then remains constant for a long period of time.

If one regards only the surface ECG, atrial depolarization may be before, during, or, apparently, after ventricular depolarization during the same episode of SVT. Thus the relationship between atrial and ventricular depolarizations is not a clue as to the mechanism of SVT.

that this arrhythmia is caused by sustained reentry within the AVCS in a total of nine unselected patients with a history of paroxysmal SVT.2

In the five patients reported here, His bundle electrograms were obtained and A-V nodal conduction delay (the A-H interval) distinguished from conduction delay within the ventricular specialized conduction system (the H-V interval). The A-H and H-V
Figure 7

Spontaneous variation in the relation of atrial depolarization to ventricular depolarization at a constant atrial cycle length (340 msec). Fifty-msec and 1-sec time markers are shown below the ECG. After a long period of stable SVT with a constant cycle length (340 msec) and A-H interval (150 msec), during which the R-R interval in the ECG remains constant, a period of spontaneous lengthening of the A-H interval from 150 to 280 msec is shown. The variation in A-V nodal conduction time allows the relationship between atrial and ventricular depolarizations to continuously “shift” over the course of these eight beats. Note that the shift occurs at a constant atrial cycle length but that variation in the R-R interval results. The dashed vertical bar at the left shows a cycle where atrial depolarization precedes ventricular depolarization; at the right, the dashed vertical bar shows atrial and ventricular depolarizations occurring simultaneously.

Intervals were normal during sinus rhythm in each patient (table 1). Functional and effective refractory periods of the AVCS were normal; these reflected the refractoriness of the A-V node, since A-V block occurred only above the bundle of His. We could distinguish no functional differences in A-V conduction between these five patients and almost 30 others studied in a similar fashion, except that in these patients sustained atrial reentry could be initiated by APDs. The fact that refractoriness and conduction are no different in these patients with a history of SVT and in 30 others with no history of SVT, and that in the former group SVT can be initiated at will while in the latter it never was, suggests that an anatomic alteration in or about the A-V node may be responsible for their paroxysmal tachycardia.

Every episode of SVT, whether spontaneous or induced by electrical stimuli, was initiated by an APD with markedly prolonged conduction from atrium to His bundle (figs. 2 and 3). The H-V interval was never longer than during sinus rhythm. Since prolonged conduction is a necessary prerequisite of reentry and only the A-H interval was prolonged, this observation by itself suggests that reentry must be occurring within the A-V node. Observations in two patients (A.P. and A.Y.) conclusively demonstrate that this is the case. In these two patients, SVT was seen to begin with APDs that failed to propagate across the
His bundle activity during the termination of SVT by single stimulated APDs. The heavy vertical bar aligns the last atrial depolarization of SVT before the introduction of a stimulated APD.

A. During SVT at a cycle length of 420 msec, the A-H interval is 110 msec. A stimulated APD with a coupling interval of 395 msec results in prolongation of the A-H interval (118 msec), but reentry is sustained and SVT continues.

B. During the same episode of SVT at cycle length 420 msec and A-H interval 110 msec, a stimulated APD is introduced 366 msec after the atrial depolarization indicated by the vertical bar. The A-H interval of this APD is markedly prolonged (195 msec) and the episode of SVT is terminated. After 1100 msec, the stimulator initiates another atrial beat with normal A-H interval (92 msec); when the stimulator was turned off, sinus rhythm (NSR) resumed.

entire A-V node to excite the bundle of His. Since His bundle depolarization was not required for initiation of SVT, the His bundle and ventricle can not be part of the reentrant pathway (fig. 3). This observation confirms the hypothesis that reentry must occur within the A-V node, and is the first demonstration of the precise site of reentry in SVT in any mammalian species.

Observations made at the termination of SVT provide further evidence that the A-V node is the site of reentry. Precisely timed stimulated APDs which terminated SVT pre-excite the atrium, producing refractory tissue in a portion of the reentrant pathway. On the other hand, when carotid sinus massage terminated SVT, vagotonia and increased acetylcholine release at the A-V node prolonged intranodal conduction to such an extent that reentry was impossible (fig. 9).

We have suggested that the relationship between P and QRS complexes on the surface
SITE OF REENTRY IN SVT

CAROTID SINUS STIMULATION

<table>
<thead>
<tr>
<th>AE</th>
<th>440</th>
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<th>465</th>
<th>465</th>
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Figure 9

Termination of sustained SVT by carotid sinus massage. AE, HBE, and ECG are recorded along with time markers at 1-sec and 50-msec intervals. Intervals in msec between successive atrial depolarizations are shown above AE. Here, during stable SVT with a cycle length of 440 msec, carotid sinus massage is begun. During massage, conduction time across the A-V node (A-H interval) progressively lengthens (110, 120, 130, 130, 215 msec). The interval between atrial depolarizations lengthens only slightly (440 to 465 msec). During the terminal cycle of SVT, the A-H interval is markedly prolonged (215 msec), and retrograde conduction fails. Sinus rhythm resumes 1175 msec later. The A-H interval of the first sinus cycle is 130 msec, showing the effect of carotid sinus massage. Over the next few sinus cycles the A-H interval returns to the control value of 90 msec.

Electrocardiogram does not help in understanding the mechanism of any paroxysmal SVT.\(^1\) The P wave is said to precede the QRS complex in atrial tachycardia, to be simultaneous with the QRS in "nodal" tachycardia, and to follow the QRS in reciprocal tachycardia.\(^2\) No matter what the P to QRS relationship became during SVT in our patients, this arrhythmia always began with an atrial premature depolarization.\(^2\) Despite this mode of onset, we saw examples of all three P to QRS relationships, and in every case the relationship was determined by the antegrade A-V nodal conduction time during atrial reentry. Figures 6 and 7 demonstrate that, even during the same episode of SVT, the surface electrocardiogram might resemble either "atrial" or "nodal" tachycardia. The clinical similarities in all paroxysmal SVTs probably result from the fact that they have the same reentrant mechanism and differ only in their degree of conduction delay in the A-V node.

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Circulation. 1971;43:15-26
doi: 10.1161/01.CIR.43.1.15

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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