The Mechanism of Supraventricular Tachycardia

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

SUMMARY
In six successive patients, none of whom had the Wolff-Parkinson-White syndrome, recurrent episodes of paroxysmal supraventricular tachycardia (SVT) were analyzed to determine the mechanism by which this arrhythmia is initiated and sustained. In each patient, simultaneous intracavitary atrial electrograms and surface electrocardiograms were recorded during the onset of numerous spontaneous episodes of SVT. Atrial premature depolarizations (APD) produced by programmed stimulation sequences were used to measure atrioventricular refractory periods and to produce atrial echoes and episodes of SVT. Stimulated APDs introduced during sustained episodes of SVT either altered its behavior or terminated it. The electrophysiologic behavior of SVT in these patients strongly suggests that the mechanism responsible for paroxysmal supraventricular tachycardia is atrial reentry utilizing the A-V conducting system.

Additional Indexing Words:
- Atrial arrhythmia
- Atrial echo beats
- Paroxysmal atrial tachycardia
- A-V refractory period
- Ventriculoatrial condition
- A-V nodal tachycardia
- Reentry phenomenon
- Reciprocal rhythm
- Atrial stimulation

The controversy over the mechanism of paroxysmal supraventricular tachycardia in man is of long standing. Many authors have thought a rapid regularly firing ectopic pacemaker located either in the atrium or atrioventricular junction was responsible for this arrhythmia.1–8 Others have felt that paroxysmal supraventricular tachycardia was due to “circus movement” or “reciprocal beating,” that is, the arrhythmia was due to reentry involving the atria and sinoatrial or atrioventricular nodes.9–11

It is not possible to establish beyond any doubt that the mechanism of a given, clinically encountered arrhythmia is due either to an ectopic pacemaker or to reentry mechanisms. It is possible, however, to perform electrophysiologic studies on patients with cardiac arrhythmias, which produce evidence strongly favoring one or the other hypothesis.12 We studied a group of six patients with recurrent paroxysmal supraventricular tachycardia in an attempt to establish the nature of its onset and persistence. Observations were made during spontaneous and evoked onset and termination of supraventricular tachycardia. In addition, the behavior of the tachycardia was studied, and the functional properties of the atrioventricular conducting system were determined by specific atrial and ventricular pacing sequences. Our results lead us to the conclusion that the mechanism of paroxysmal supraventricular

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tachycardia in this group of patients is reentry utilizing the tissues of the atrium and the atrioventricular conducting system.

Methods

During a 1-year period (1968-69) six patients were referred for evaluation of paroxysmal supraventricular tachycardia; none had the Wolff-Parkinson-White syndrome. The salient clinical features of these patients are presented in table 1. A.P., who had been followed for 30 years for ankylosing spondylitis, had a brief aortic decrescendo diastolic murmur but no evidence of hemodynamically significant aortic regurgitation. Two patients, J.L. and M.H., had ECG abnormalities in the absence of drugs, and M.H. had an enlarged heart; the cause of the heart disease was not found in these cases. Although arteriosclerotic heart disease was suspected, none had a history of angina pectoris or myocardial infarction; coronary arteriography was not performed.

The history of palpitations varied widely in duration, but certain characteristics of the palpitations were identical in all these patients. All paroxysms were sudden in onset without prodromata, all were rapid, regular, and although of variable duration, ceased abruptly and spontaneously. None of these patients had ever attempted vagal maneuvers to terminate episodes of palpitations.

Electrocardiographic documentation of a regular supraventricular tachycardia (SVT) was made in each patient, and all were admitted to the hospital. At the time of admission, P.M., J.L., and A.P. had congestive heart failure associated with almost continuous SVT. Congestive heart failure cleared when the arrhythmia was controlled. Only patient M.H. was taking medication on admission; she had taken 0.25 mg of digoxin a day for 3 years and had normal serum potassium and blood urea nitrogen. Routine laboratory evaluation failed to demonstrate pertinent chemical abnormalities; serum sodium, potassium, chloride, carbon dioxide, calcium, urea nitrogen, and thyroid function were all normal. Endocrine causes for the recurring arrhythmias were absent. Cardiac configuration was normal on chest x-rays in all patients except M.H. in whom cardiomegaly was demonstrated. Electrocardiographic data are summarized in table 2.

All patients had continuous monitoring of the ECG in order to record the spontaneous onset of their arrhythmia. Five patients had frequent episodes of tachycardia during the monitoring, and each identified his symptoms during SVT as characteristic of his palpitations; one patient (P.M.), admitted after 16 days of continuous SVT, was so ill that cardioversion was performed. Tachycardia did not recur spontaneously during the subsequent 10 days of continuous monitoring.

All patients were brought to the cardiopulmonary laboratory in the nonsedated, postabsorptive state. Under local procaine anesthesia, two electrode catheters were introduced via the basilic or saphenous vein and positioned high in the right atrium under fluoroscopic and electrocardiographic control.

### Table 1

**Clinical Characteristics of Patients with Paroxysmal SVT**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Etiology</th>
<th>Cardiac disease</th>
<th>Congestive heart failure</th>
<th>Palpitations</th>
<th>Medications on admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>P.A.</td>
<td>19</td>
<td>M</td>
<td>No heart disease</td>
<td>Normal</td>
<td>No</td>
<td>Minutes to days</td>
<td>None</td>
</tr>
<tr>
<td>O.F.</td>
<td>44</td>
<td>F</td>
<td>No heart disease</td>
<td>Normal</td>
<td>No</td>
<td>Minutes to days</td>
<td>None</td>
</tr>
<tr>
<td>P.M.</td>
<td>53</td>
<td>M</td>
<td>No heart disease</td>
<td>Normal</td>
<td>No*</td>
<td>Minutes to days</td>
<td>None</td>
</tr>
<tr>
<td>A.P.</td>
<td>58</td>
<td>M</td>
<td>Rheumatoid spondylitis with AI</td>
<td>Normal</td>
<td>No*</td>
<td>Minutes to hours</td>
<td>None</td>
</tr>
<tr>
<td>J.L.</td>
<td>78</td>
<td>M</td>
<td>Unknown heart disease</td>
<td>Normal</td>
<td>No*</td>
<td>Minutes to hours</td>
<td>None</td>
</tr>
<tr>
<td>M.H.</td>
<td>85</td>
<td>F</td>
<td>Unknown heart disease</td>
<td>Slightly enlarged</td>
<td>No</td>
<td>Minutes to hours</td>
<td>Digoxin, 0.25 mg/day (since 1965)</td>
</tr>
</tbody>
</table>

*CHF when arrhythmia was uncontrolled.
SUPRAVENTRICULAR TACHYCARDIA

Table 2

<table>
<thead>
<tr>
<th>ECG Characteristics During Sinus Rhythm and SVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
</tr>
<tr>
<td>---------</td>
</tr>
<tr>
<td>P.A.</td>
</tr>
<tr>
<td>O.F.</td>
</tr>
<tr>
<td>P.M.</td>
</tr>
<tr>
<td>A.P.</td>
</tr>
<tr>
<td>J.L.</td>
</tr>
<tr>
<td>M.H.</td>
</tr>
</tbody>
</table>

Abbreviations: MLAD = marked left axis duration (> −30°); LBBB = left bundle-branch block; IRBBB = incomplete right bundle-branch block; NL = normal.

Intra-atrial electrograms and surface electrocardiograms were monitored on a switched-beam oscilloscope and recorded on magnetic tape. The spontaneous onset of numerous paroxysms of SVT was recorded in every patient except P.M. In all patients the A-V conduction and refractory periods were measured by use of the extra-stimulus method. The right atrium was paced slightly faster than the spontaneous sino-atrial rate using rectangular cathodal stimuli, 1.8 msec in duration, provided by a series of wave form and pulse generators; a premature (test) stimulus was introduced after every tenth drive stimulus. The drive stimulus following the test cycle was omitted to prevent rapidly successive stimuli from entering the atrium and to allow for the observation of spontaneous reentrant activity should it occur. Successive test stimuli were applied prematurely by 5 to 10-msec decrements until functional and effective refractory periods were determined. In one patient (M.H.) episodes of SVT occurred with such frequency that refractory periods could not be accurately measured. In all patients, selectively placed premature stimuli were used to begin episodes of SVT.

In all patients except P.M. (the first studied), the time zone during which premature atrial responses caused atrial reentry (echoes) or SVT was determined. The atrial electrogram during sinus rhythm was used to trigger an oscilloscope. The gated output of this oscilloscope was counted electronically and, after every tenth sino-atrial depolarization, atrial premature depolarizations (APDs) were produced by cathodal stimuli. The stimuli were progressively made more premature by 5 to 10-msec decrements, and the interval during which atrial echoes and episodes of SVT could be induced was determined.

In all patients episodes of SVT were terminated by single stimulated APDs. In the first two patients (P.M. and P.A.) atrial stimuli were allowed to "wander" through the atrial cycle during SVT. In all subsequent patients the stimulating system was triggered from the atrial depolarizations during SVT and atrial stimuli, introduced in the manner described for determining the echo zone.

After these studies, the stimulating catheter was advanced into the ventricle. Ventriculo-atrial conduction time and ventriculo-atrial refractory periods were measured, and an attempt was made to induce episodes of SVT with ventricular premature depolarizations induced with test stimuli.

Results

The spontaneous onset of SVT recorded in five of the six patients is shown in figure 1. In most of these patients, the onset of 20 to 100 episodes of SVT was recorded. SVT always began with an atrial premature depolarization (APD). The average length of the initial atrial cycle (shaded area of fig. 1) of SVT in all patients was 432 ± 34 msec (mean ± sd). In any given patient there was some variation in the length of this initial cycle. Furthermore, the APD initiating SVT always showed prolonged A-V conduction; in other words, it fell in the relative refractory period of the A-V conducting system. There was considerable variation in atrial cycle length during the
The spontaneous onset of SVT recorded in five patients. Each panel is identified by patient's initials and shows the atrial electrogram (EGM) and surface electrocardiogram (ECG). Time markers at 50-msec and 1-sec intervals are shown at the bottom panel of the figure.

The wide vertical bar aligns the last of three atrial depolarizations during sinus rhythm (NSR), that is, two atrial cycles during NSR appear to the left of this bar in each panel. Intervals, in milliseconds, between consecutive atrial depolarizations appear above the atrial electrogram. In every panel, a spontaneous atrial premature depolarization (the first atrial depolarization to the right of the heavy vertical bar) initiates SVT. Diagonal lines between atrial electrogram and ECG are used to represent electrical events within the A-V conduction system. The area between conduction of the last sinus beat and conduction of the APD is shaded to emphasize the cycle initiating SVT and the prolonged A-V conduction of the APD. In each patient, the duration of atrial cycles varied markedly during the first few beats of SVT.

Initial cycles of spontaneous SVT. The variation usually consisted of alternation of shorter and longer cycles which, in some patients, persisted for quite a few cycles before a stable
Table 3

Characteristics of SVT and A-V Conduction System

<table>
<thead>
<tr>
<th></th>
<th>P.A.</th>
<th>O.F.</th>
<th>P.M.</th>
<th>A.P.</th>
<th>J.L.</th>
<th>M.H.</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVT CL (msec)</td>
<td>435</td>
<td>326</td>
<td>330</td>
<td>320</td>
<td>495</td>
<td>395</td>
</tr>
<tr>
<td>Rate (beats/min)</td>
<td>138*</td>
<td>184</td>
<td>182</td>
<td>187</td>
<td>122*</td>
<td>152*</td>
</tr>
<tr>
<td>QRS configuration</td>
<td>NL</td>
<td>NL</td>
<td>IRBBB</td>
<td>NL</td>
<td>LBBB</td>
<td>NL</td>
</tr>
<tr>
<td>AVRP:CL (msec)</td>
<td>500</td>
<td>600</td>
<td>513</td>
<td>600</td>
<td>650</td>
<td>820</td>
</tr>
<tr>
<td>FRP (msec)</td>
<td>446†</td>
<td>378</td>
<td>372</td>
<td>340</td>
<td>440†</td>
<td>450†</td>
</tr>
<tr>
<td>ERP (msec)</td>
<td>333‡</td>
<td>260</td>
<td>&lt;246</td>
<td>247</td>
<td>335‡</td>
<td>340‡</td>
</tr>
<tr>
<td>Conduction time (msec)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A-V</td>
<td>160</td>
<td>160</td>
<td>160</td>
<td>160</td>
<td>250</td>
<td>200</td>
</tr>
<tr>
<td>V-A</td>
<td>194</td>
<td>120**</td>
<td>175</td>
<td>182</td>
<td>210**</td>
<td>130**</td>
</tr>
<tr>
<td>V-A ERP (msec)</td>
<td>354</td>
<td>265</td>
<td>292</td>
<td>&lt;380</td>
<td>300</td>
<td>-</td>
</tr>
<tr>
<td>Limits of echo zone (msec)</td>
<td>310-549</td>
<td>265-420</td>
<td>-</td>
<td>252-312</td>
<td>345-425</td>
<td>370-670</td>
</tr>
<tr>
<td>Duration of echo zone (msec)</td>
<td>239</td>
<td>155</td>
<td>-</td>
<td>60</td>
<td>80</td>
<td>300</td>
</tr>
</tbody>
</table>

*Slow rate, see text.
†Long FRP.
‡Long ERP.
§Measured at CL similar to AVRP CL.
**V-A < A-V unusual, see text.

Abbreviations: IRBBB = incomplete right bundle-branch block; LBBB = left bundle-branch block; NL = normal.

SVT cycle length became established (see M.H., fig. 1). In other patients, a stable atrial cycle length was more quickly established (see J.L., fig. 1). The interval between the spontaneous atrial premature depolarization (A₂) which initiated the tachycardia and the preceding atrial depolarization in sinus rhythm (A₁) was rather long in each of these cases, and one would expect the following atrial interval (A₂A₃) to be short if a reentrant mechanism utilizing the A-V conducting system was involved. This was generally true. The A₂A₃ interval was shorter than the A₁A₂ interval in all of the cases except J.L.

The differences seen in J.L. are explained in the following way: J.L. was the only patient with prolonged A-V conduction during sinus rhythm. Furthermore, the functional and effective refractory periods of his A-V conducting system were prolonged (table 3). In addition, the duration of the relative refractory period was longer in J.L. than in the other patients. This is demonstrated in figure 1 in which the prematurity of the APD initiating SVT is similar to that occurring in other patients but the APD propagates to the ventricles much more slowly. In J.L. impaired conduction in the A-V conducting system also causes prolonged retrograde conduction in the first reentry cycle, resulting in a long A₂A₃ cycle. J.L. also has the longest V-A conduction time during ventricular pacing (table 3).

To obtain further information on the mechanism of SVT onset and to define the functional characteristics of the A-V conducting system, studies were done using the extra-stimulus method. A-V conduction of premature atrial depolarizations and the effective and functional refractory periods of the A-V conduction system were determined. Table 3 summarizes these results. Even at atrial pacing rates 25% above the sinus rate prevailing at the time of study, A-V conduction time was normal in all of the patients except J.L. During ventricular pacing at similar cycle lengths, 1:1 ventriculo-atrial conduction was present in each patient. The A-V effective and
The similarity between spontaneous atrial echoes and either the spontaneous or electrically stimulated onset of supraventricular tachycardia (SVT) is shown in these recordings of M.H. Each of the four panels displays the atrial electrogram (EGM) above and electrocardiogram (ECG) below. One-second and 50-msec time markers are shown at the bottom of the figure. The wide vertical bar that runs through the entire figure aligns the last of three atrial depolarizations during sinus rhythm in each panel; the first atrial depolarization to the right of the bar is an atrial premature depolarization (APD) which is either spontaneous (A, B, and C) or electrically stimulated (D). The intervals (in milliseconds) between successive atrial depolarizations appear above the atrial electrogram in each panel.

In A, a spontaneous APD occurs 650 msec after a sinus beat, exhibits prolonged A-V conduction, and results in an atrial echo 320 msec later. Antegrade conduction of this echo is blocked within the A-V conduction system and sinus rhythm continues.

In B, a spontaneous APD 460 msec following a sinus beat results in two consecutive atrial echoes.

In C, a spontaneous APD occurs 10 msec earlier than that shown in panel B, resulting in atrial reentry similar to that shown in panels A and B, but in this instance reentrant activity persists as an episode of SVT.

In D, an evoked APD (stimulus indicated by the arrow) 370 msec after a sinus beat results in an episode of SVT similar to that shown in panel C.
The onset of 27 spontaneous episodes of SVT was recorded and analyzed in patient P.A. The interval from the spontaneous APD \( A_0 \) to the first beat of SVT (the \( A_2A_3 \) interval) is plotted as a function of the interval from the last sinus beat \( A_1 \) to the APD \( A_1A_2 \) interval. There is a strong reciprocal relationship between these intervals \( (df = 25, r = 0.82, P < 0.001) \). This finding suggests that the first atrial depolarization in the tachycardia \( A_0 \) is an echo beat induced by \( A_2 \).

Functional refractory periods are slightly prolonged in J. L., M. H., and P. A. and normal in the other patients.

In the five patients in whom multiple episodes of SVT were observed, we demonstrated a discrete period of time within the atrial cycle during which spontaneous or stimulated atrial premature depolarizations invariably resulted in either atrial echoes or SVT (table 3). In each case the inner limit of this echo zone was fairly early in the atrial cycle; yet in four of the five patients, it was not as early as the A-V effective refractory period. In every case the echo zone was completely contained within the relative refractory period; the duration of the echo zone varied from 60 to 300 msec. An example of events occurring during the echo zone is shown in figure 2. The similarity between atrial echoes and SVT demonstrated in M. H. (fig. 2) was typical of the group in every way except for the very long duration of the atrial echo zone. The relationship between the cycle initiating an echo or SVT \( A_1A_2 \) and the second cycle \( A_2A_3 \) is reciprocal, as might be expected of atrial reentrant beats. In panel A of figure 2, after a long \( A_1A_2 \) interval (650 msec) the \( A_2A_3 \) interval is short (320 msec) whereas, in panel D, after a short \( A_1A_2 \) interval (370 msec) the \( A_2A_3 \) interval is long (500 msec). Figure 3 shows the reciprocal relationship between the first \( A_1A_2 \) and second \( A_2A_3 \) cycles of SVT in patient P. A. during 27 spontaneous episodes. An equally strong relationship was found in two of the three additional patients in whom data were sufficient for analysis.

Not only did spontaneous SVT occur in each of the patients, but in all, single electrically stimulated APDs evoked during the atrial echo zone resulted in episodes of SVT identical to those occurring spontaneously (fig. 4). Although the initiating intervals \( A_1A_2 \) shown in figure 4 are shorter than those depicted for the spontaneous onset in figure 2, we analyzed all cycles initiating SVT (spontaneous and stimulated) and found considerable overlap in the duration of the first cycles of stimulated and spontaneously occurring SVT in four patients. In the other two patients data were insufficient for analysis; in patient P. M. spontaneous onset was not observed, and in patient P. A. only seven episodes of SVT were initiated by electrical stimuli. During the determination of the ventriculo-atrial refractory period (table 3), premature ventricular responses were elicited throughout the ventricular cycle. Although retrograde propagation of premature ventricular responses activated the atria prematurely, SVT never resulted. V-A conduction time of very premature ventricular responses was so prolonged that the atria were never activated in the echo zone.

SVT was not the result of stimulation during the atrial vulnerable period. In every case in which the atrial vulnerable period was identified, it was brief and occurred considerably earlier in the atrial cycle than the echo zone (fig. 5). Furthermore, repetitive atrial responses evoked by stimulation during the

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The onset of SVT produced by electrical stimulation in all six patients. Each panel consists of atrial electrogram (EGM) and surface electrocardiogram (ECG) identified by the initials of the patient in whom they were recorded. Time markers at 1-sec and 50-msec intervals are shown at the bottom of the figure.

The wide vertical bar aligns the last of three atrial depolarizations prior to a stimulated atrial premature depolarization (APD). This APD begins a paroxysm of SVT in each patient. Two sinus cycles appear to the left of the vertical bar in patients O.F., A.P., J.L., M.H. In patients P.A. and P.M. two cardiac cycles during atrial pacing are illustrated, stimulus artifacts precede the P waves prior to the onset of SVT. Intervals between consecutive atrial depolarizations in milliseconds appear above the atrial electrogram in each panel. In every panel, an arrow denotes the stimulus used to evoke the APD which begins SVT. The APD is the first atrial depolarization to the right of the wide vertical bar. Diagonal lines between atrial electrogram and ECG represent electrical events within the A-V conduction system (see discussion). The area between conduction of the last beat prior to SVT and conduction of
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The different responses obtained by stimulating at various times during the atrial cycle. Only the atrial electrogram is displayed in each panel as test stimuli are made increasingly premature. Time marks at 50-msec intervals are shown at the bottom of the figure. In this study the atria were paced at a constant cycle length of 610 msec, and premature stimuli introduced every tenth drive cycle at varying times after the basic pacing stimulus; stimuli are indicated by arrows.

The heavy vertical bar aligns the last paced beat prior to the introduction of a stimulated atrial premature depolarization (APD). To the left of the bar, is one paced atrial cycle. To the right of the bar, the interval in milliseconds between the last basic drive stimulus and the premature stimulus is given above the atrial electrogram. In A to D, the atrial premature stimulus is introduced successively more prematurely in the atrial cycle.

In A, a premature stimulus, 320 msec after the last paced beat, results in an atrial echo and episode of SVT which is sustained at a cycle length of 450 msec.

In B, the interstimulus interval is decreased to 270 msec. The premature stimulus elicits an APD which falls earlier than the echo zone so that neither an echo nor an episode of SVT results. The premature stimulus also falls within the atrial relative refractory period so that the latency between the stimulus and response is increased.

In C, the interstimulus interval is further decreased by 10 msec to 260 msec. This stimulus falls within that portion of the atrial relative refractory period called the atrial "vulnerable" period. After a latency of more than 50 msec, the single stimulus results in a premature period had very short cycle length, terminated spontaneously after two or three beats, and never resulted in SVT (fig. 5 C). Since the atrial vulnerable period resides in the atrial relative refractory period, stimuli falling in this period evoke responses only after a long latency.\(^\text{12}\) Stimuli which evoked SVT in our patients never showed an increased latency between stimulus and response.

Once SVT was established, it was usually maintained for long periods of time unless interrupted by electrical stimulation or by carotid sinus massage. The effects of electrically induced atrial premature responses during SVT were of great interest; an example is shown in figure 6. Premature atrial activation had one of three effects on the subsequent course of the tachycardia; it either (1) was followed by a full compensatory pause, (2) caused a pause greater than compensatory, or (3) terminated the tachycardia. Full compensatory pauses only occurred following premature atrial activation late in the SVT cycle. Activation of the atria earlier in the SVT cycle caused pauses greater than fully compensatory pauses. As the atria were activated progressively earlier in the SVT cycle, pauses became progressively longer than the compensatory pause until the tachycardia was terminated (fig. 6). Behavior compatible with pacemaker reset was never observed during the course of atrial stimulation in SVT. In each case we were able to alter the behavior of the tachycardia in this manner and with appropriately timed stimuli to terminate the tachycardia (fig. 7). The events following introduc-

two atrial responses 200 msec apart. Stimulation during the atrial "vulnerable" period does NOT result in an episode of SVT.

In D, the interval between stimuli is decreased to 250 msec and no atrial response results, that is, this interval is shorter than the atrial effective refractory period. Sinoatrial escape occurs before the next atrial drive stimulus is delivered.

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The APD is shaded, in order to emphasize the cycle initiating SVT and the prolonged conduction of the APD. Note the variation in atrial cycle length during the first cycles of SVT in each patient.
Effect of induced APDs (A) during established SVT in patient M.H., electrical stimulation of the atrium was used to evoke atrial premature depolarizations (APD) after every sixth atrial beat of SVT. In the three panels to the right of the graph, atrial electrogram and surface electrocardiogram are shown during atrial cycles in which a stimulated APD (A') was evoked. A wide vertical bar aligns the last of six atrial depolarizations (A') before A' is introduced. Intervals between successive atrial depolarizations are given in milliseconds above each atrial electrogram. Events postulated to occur within the atrioventricular capture cycle (AVCS) are represented by diagonal lines connecting atrial and ventricular depolarizations. The heavy dashed vertical line indicates where A' would be expected to appear after A' if there were a fully compensatory pause. Panels 1, 2, and 3 demonstrate the effect of inducing A' with increasing prematurity (A' A' intervals of 370, 285, and 270 msec). The cycle length of this SVT is 400 msec. In panel 2, for example, the interval between A' and A' (A'A' = capture cycle) is 285 msec. The interval between A' and the next beat of SVT (A'A' = return cycle) is 625 msec. The sum of these two cycles, 910 msec, is 110 longer than would have been expected if a full compensatory pause had occurred following A'.

On the graph to the left of the figure, return cycles are plotted as a function of capture cycles as the interval between A' and A' is made progressively shorter. The diagonal line shows where points would fall if the pause following A' were fully compensatory. Similarly, the shaded area of the graph indicates where points would be expected to fall if an atrial pacemaker were depolarized and reset by A'. All the observed points fall above the line labeled full compensatory pause; none fall within the area of pacemaker reset (see discussion).

In panel 3, at a capture cycle (A'A') of 270 msec, the episode of SVT is terminated, and A' fails to conduct to the ventricle. Capture cycles which resulted in termination of SVT are indicated by points which fall below the interruption in the ordinate of the graph.

Figure 6

The diagram illustrates the relationship between atrial premature depolarizations (APDs) and the occurrence of atrial premature atrial contractions (APCs). The graph shows the effect of induced APDs (A) during established SVT in patient M.H., electrical stimulation of the atrium was used to evoke atrial premature depolarizations (APD) after every sixth atrial beat of SVT. The intervals between successive atrial depolarizations are given in milliseconds above each atrial electrogram. The effect of inducing A' with increasing prematurity (A' A' intervals of 370, 285, and 270 msec) is demonstrated in panels 1, 2, and 3. The cycle length of this SVT is 400 msec. In panel 2, for example, the interval between A' and A' (A'A' = capture cycle) is 285 msec. The interval between A' and the next beat of SVT (A'A' = return cycle) is 625 msec. The sum of these two cycles, 910 msec, is 110 longer than would have been expected if a full compensatory pause had occurred following A'.

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The tachycardia ceased immediately after the premature atrial activation; in others (O. F. and A. P.) one atrial echo occurred following

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Termination of SVT by single stimulated APDs evoked in each patient. An atrial electrogram (EGM) and surface electrocardiogram (ECG) are shown for each patient. Time marks at 50 msec and 1-sec intervals appear at the bottom of figure.

The last of four atrial depolarizations during SVT prior to the introduction of a stimulated SPD (indicated by an arrow) are shown to the left of the wide vertical bar. The numbers above the atrial electrogram indicate intervals in milliseconds between consecutive atrial depolarizations. Diagonal lines represent electrical events postulated to occur within the A-V conduction system at the termination of SVT.

In patients P.A. and P.M. electrical stimuli were allowed to “wander” through the atrial cycle. As a result, stable atrial pacing resulted when SVT was terminated; a stimulus artifact can be seen to precede each P wave in the surface electrocardiogram. In the other four patients a single APD synchronized to atrial depolarization was introduced. Termination of SVT results in sinus rhythm in each case (see discussion).

In patients O.F. and A.P., the stimulated APD causes a single reciprocal beat which alters antegrade and retrograde conduction times sufficiently to interrupt the SVT (see discussion).
SVT rhythm resumed. Atrial activity was noted to be able to terminate SVT by prematurely stimulating the ventricles. Since sinus cycle length and the refractoriness of the A-V conducting system both vary inversely with increases in intrinsic sympathetic tone and directly with intrinsic parasympathetic tone, this provided the opportunity to determine that the SVT rate varied with changes in sinus cycle length and presumably with change in A-V refractory period. At a sino-atrial cycle length of 905 msec, the cycle length of SVT was 470 msec (figs. 2 and 4), whereas after a sinus cycle of 640 msec the cycle length of SVT decreased to 400 msec (fig. 2). Furthermore, during SVT with a cycle length of 470 msec, the intravenous administration of atropine resulted in a change in cycle length to 405 msec.

**Discussion**

In 1968, we encountered a patient with recurrent attacks of paroxysmal SVT. Electrophysiologic stimulating and recording procedures demonstrated the cause of SVT in that case to be reentry via the A-V conducting system. This led us to ask if this mechanism was characteristic of paroxysmal SVT. Accordingly, we began to study the mechanism of SVT in unselected patients with a history of spontaneous paroxysms of this arrhythmia. Our evaluation of the genesis and sustaining mechanism of paroxysmal SVT was facilitated by four considerations: (1) paroxysms of tachycardia are usually disturbing enough to prompt investigation, (2) the paroxysmal nature of the arrhythmia provides an opportunity to observe the spontaneous onset and termination of the arrhythmia, (3) a short period of SVT is not usually hemodynamically or electrically hazardous, and (4) episodes are easily terminated. Therefore, we felt that induction and termination of these tachycardias were justified.

Accordingly, we employed stimulation and recording methods selected to differentiate between arrhythmias caused by ectopic automatic foci and those caused by reentrant mechanisms. In our six patients the following observations favor reentry mechanisms: (1) spontaneous episodes of SVT were initiated only by APDs occurring in the relative A-V refractory period, (2) the capacity for retrograde (V-A) conduction was demonstrated during ventricular pacing, (3) spontaneous single or multiple atrial echo beats were observed, (4) APDs elicited by electrical stimuli during certain portions of the relative-A-V refractory period initiated SVT, (5) the
first and second cycles of SVT were reciprocally related, (6) electrical stimuli during the atrial vulnerable period did not elicit SVT, (7) the cycle length during SVT was a positive linear function of the refractoriness of the A-V conducting system, (8) APDs evoked during sustained SVT were followed by atrial cycles longer than fully compensatory, and (9) appropriately timed electrical stimuli evoked APDs which terminated SVT.

**Onset of Supraventricular Tachycardia**

If SVT were initiated by an ectopic supraventricular pacemaker, the onset could occur in any portion of the atrial cycle where the atrial tissues are not refractory, that is, one might expect some episodes to begin with APDs which fall outside the relative A-V refractory period. On the other hand, SVT initiated by reentry in the A-V conducting system could only occur when the initial beat falls during the relative A-V refractory period. Experimentally, atrial reentry had been shown to depend upon the fact that a premature depolarization entering the A-V conducting system during its relative refractory period must experience sufficient antegrade conduction delay to allow the propagating impulse to return to (reenter) its chamber of origin.14-16 Clinically, atrial reentry has been shown to depend on prolonged A-V conduction of the initiating APD.17-20 Every episode of SVT in our patients began with an APD evoked in the A-V relative refractory period. Atrial premature depolarizations which did not demonstrate prolonged A-V conduction never resulted in SVT.

**Capacity for V-A Conduction**

It is apparent that atrial reentry via the A-V conducting system depends on the ability of this system to sustain retrograde conduction; atrial reentry is impossible unless V-A conduction occurs in some portion of the A-V conducting system. In every patient we demonstrated retrograde conduction traversing the entire A-V conducting system by pacing the right ventricle and observing the atrial responses. Although it is distinctly unusual to produce V-A conduction in patients with any impairment of A-V conduction, even J. L., who had a prolonged P-R interval, showed retrograde conduction. Thus, the retrograde conduction required to sustain reentry was shown to occur in all these patients.

**Atrial Echo Beats and Onset of Supraventricular Tachycardia**

If an ectopic pacemaker assumed control of the atria for one, two, or any number of cycles, one would expect the atrial cycle lengths to (1) be invariable, (2) show progressive shortening to a stable cycle length, as expected in the warm up behavior of an ectopic pacemaker or in type I (Wenckebach) exit block from the pacemaker, (3) show progressive lengthening due either to inherent pacemaker slowing or to conduction impairment progressing to total exit block, or (4) show grouped beating of the type expected during type II exit block from the pacemaker. In spontaneously occurring atrial echoes of SVT it may be necessary to observe many episodes to demonstrate adequately the mechanism involved. For example, episodes with short atrial length were analyzed in patient M. H. (fig. 2). Consideration of panels A, B, and C in figure 2 conclusively shows that the short atrial cycles are not invariable in duration, do not progressively shorten or progressively lengthen, and do not show alternation compatible with type II exit block from an ectopic focus. The analysis of sequences of atrial cycles during spontaneous SVT did not suggest origin in an ectopic pacemaker in any case.

The behavior of the short cycles was, however, compatible with a reentrant mechanism. Atrial reentrant beats (atrial echoes, reentrant SVT) are characterized by a reciprocal relationship between the cycle initiating reentry (see A1A2 interval, the shaded cycle in figs. 1 and 2) and the return atrial cycle (A2A3). An example of this reciprocal relationship is shown in figure 3. APDs occurring late in the relative A-V refractory period (long A1A2) return rapidly to the atria (short A2A3); APDs occurring early in the relative A-V refractory period (short A1A2) return more
slowly (long $A_2A_3$). Such behavior not only explains atrial echo beats but also the oscillation of the initial atrial cycles in SVT. The sequences of atrial depolarization in both atrial echo beats and initial cycles of SVT are strikingly similar; in SVT the echo phenomenon (reentrant activity) is merely sustained.

**Electrically Stimulated Atrial Echoes and Supraventricular Tachycardia**

Despite extensive investigation there is no experimental evidence to suggest that stimulated APDs can elicit automatic activity in the cells of the atrium or A-V junction.\textsuperscript{14, 15} Therefore we thought if SVT could be reproduced in our patients by single stimulated APDs, it would strongly favor the reentrant hypothesis. This was accomplished several times in every case. In each patient test stimuli revealed a discrete zone within which reentrant activity was reproduced. This zone was completely contained within the relative A-V refractory period but distinct from the atrial vulnerable period. We have previously discussed in detail the differences between atrial responses evoked by stimulating during the echo zone as opposed to those evoked by stimulating during the vulnerable period.\textsuperscript{12} It is important to note here that stimulation during the vulnerable period never resulted in SVT (fig. 5).

**Rate of Supraventricular Tachycardia and A-V Refractoriness**

If SVT were the result of reentry via the A-V conducting system, then the rate would be determined by the refractoriness of this system. We found a direct linear relationship between the rate of SVT and both the A-V effective and functional refractory period in our group of patients (fig. 8), that is, the longer the A-V refractory periods the slower the rate. Conceivably, such a relationship could be found if SVT were caused by an ectopic pacemaker under strong autonomic neural control. This hypothesis predicts that vagal maneuvers would cause progressive deceleration of the pacemaker. However, the well-known effect of carotid sinus massage or other vagal maneuvers on SVT is to terminate it abruptly or not to affect it. The effect of vagal maneuvers on SVT is more consistent with acetylcholine causing conduction block in a reentrant circuit within the A-V node.\textsuperscript{22}

**Premature Depolarization of the Atria During Supraventricular Tachycardia**

Assuming that established SVT is caused by an ectopic pacemaker one could predict that an APD induced during SVT would either depolarize and reset the pacemaker or not. The former occurrence would produce a pause less than compensatory and the latter a fully compensatory pause. We did this experiment in each patient and found that most APDs elicited during SVT resulted in pauses longer than fully compensatory (fig. 6). This behavior is inconsistent with the hypothesis that SVT is caused by a pacemaker but would be expected of a reentrant rhythm utilizing the A-V conducting system. We found that the earlier an APD was elicited in the SVT cycle, the longer was the following pause and the greater the discrepancy between the next SVT beat and a fully compensatory pause; very premature APDs terminated the tachycardia (figs. 6 and 7). We propose that these stimulated APDs during SVT entered the reentrant pathway earlier and earlier in its relative refractory period and experienced progressive conduction delay until conduction failed altogether.

**General Considerations**

Some form of reentry has been considered as a possible mechanism for paroxysmal tachycardia since the description of circus movement by Mines in 1913.\textsuperscript{23} Iliescu and Sebastiani\textsuperscript{9} first suggested that paroxysmal SVT was due to circus movement. In 1925 Lewis\textsuperscript{2} rejected circus movement as the basis of this rhythm because he reasoned that the conduction velocity in the atrial musculature should result in a faster rate and there should be no isoelectric interval between atrial depolarizations in circus rhythm. Barker and associates\textsuperscript{11} countered these objections by suggesting that the part of the reentrant path might be either the sinoatrial or atrioventricular node. They supported their view with extensive observations on the clinical and electrocardiographic features of SVT and on
its response to vagal maneuvers and drug therapy. For reasons that are not apparent to us, this view does not seem to have been widely accepted.

In recent years, extensive laboratory investigations have delineated the electrophysiological properties of the A-V conducting system and shown its capacity to sustain reentry and reentrant tachycardia.14,15,24,25 In addition, a number of recent clinical reports demonstrate supraventricular reentrant tachycardias.12,17-19,26-30 Most of these reports describe patients selected by electrocardiographic features noted at the onset of SVT. We were surprised to find overwhelming evidence for a reentry mechanism in six consecutive cases selected for study only because of a history of recurrent supraventricular tachycardia. It should be noted that from use of electrocardiographic criteria alone, we would have suspected the reentrant mechanism in only two of the six patients. Intracardiac electrograms and special stimulation sequences were required to demonstrate the mechanism in the other four. We conclude that Barker and associates' suggestion is correct—paroxysmal supraventricular tachycardia is most often due to reentry utilizing the A-V conducting system.

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**Style, Rhetoric, and Social Intercourse**

In the very act of addressing someone we acknowledge a wish to push him around, and in our zeal to push a little harder, it is no wonder our voices begin to sound strident. It is with style that we try to behave like a decent person, one who ruefully concedes his drive for power while remaining aware of his reader's well-chosen resistance. Thus style is our way of becoming a person worth listening to, worth knowing.

A moral justification for the study of rhetoric lies right here. We improve ourselves by improving the words we write. We make our performance less monstrous, by *acting* like human beings. Just what comprises a satisfactory human performance is every man's complicated decision. But at least, by looking at rhetoric, we may begin to know more about who it is we are making believe we are. And then, perhaps, we can do something about it.—From GIBSON W: Tough, Sweet & Stuffy: An Essay on Modern American Prose Styles. Bloomington, Indiana, Indiana University Press, 1966, p 110.
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