Demonstration of Sustained Sinus and Atrial Re-entry as a Mechanism of Paroxysmal Supraventricular Tachycardia

By Delon Wu, M.D., Fernando Amat-y-Leon, M.D., Pablo Denes, M.D., Ramesh C. Dhingra, M.D., Raymond J. Pietras, M.D., and Kenneth M. Rosen, M.D.

SUMMARY
Electrophysiological studies in five patients with documented (4) or suspected (1) paroxysmal supraventricular tachycardia (PSVT), suggested sinus or atrial re-entry (SR or AR). Two of the patients had pre-excitation, three had evidence of atrial enlargement, and all had organic heart disease. The following observations supported a diagnosis of SR and AR: 1) induction of sustained PSVT with atrial extrastimulus technique allowing definition of an echo zone; 2) induction of sustained PSVT during constant rapid atrial pacing at a rate less than that producing A-V nodal Wenckebach periods, or producing normalization of QRS complex in patients with pre-excitation; 3) P waves preceding each QRS during PSVT with an A-H interval appropriate for the rate of the PSVT; 4) antegrade P wave morphology during PSVT, and normal high to low sequence of right atrial activation (SR), or P wave morphology and atrial activation sequence different from sinus (AR); 5) lack of correlation of PSVT induction with critical A-H interval. The rates of induced sustained PSVT ranged from 114 to 143 beats/min, and were similar to those observed during spontaneous episodes of PSVT in the four patients. PSVT could be terminated with critically timed extra-stimuli or carotid massage.

In conclusion, SR and AR appear to be mechanisms of spontaneous PSVT in man. Rates of SR and AR PSVT tend to be relatively slow.

Additional Indexing Words:
Atrioventricular nodal re-entry
Atrial extrastimulus
Pre-excitation
Refractory period
His bundle electrogram
atrial re-entrant PSVT is compared to that of A-V nodal re-entrant PSVT.

Methods
Patient Selection
The study group consisted of four patients with previously documented PSVT (Cases 1,2,4,5), and one patient with palpitations and suspected PSVT (Case 3). (table 1). Clinical and electrocardiographic data are summarized in table 1. Two of the patients had pre-excitation (Cases 2 and 3), and all of the patients had organic heart disease. Three of the patients had electrocardiographic and/or radiologic evidence of atrial enlargement.

Two of the patients were on cardiac medications prior to study, Case 2 receiving digitalis and quinidine, and Case 5 receiving digitalis. These drugs were discontinued 48 hours prior to study.

Electrophysiological Studies
Electrophysiological studies were performed in the postabsorptive, nonsedated state. Informed consent was obtained. Two electrode catheters were utilized, both passed percutaneously to the right heart, via the femoral veins. The first tripolar catheter was placed across the tricuspid valve for the recording of low right atrial and His bundle electrograms. The second quadrripolar catheter was placed against the high lateral right atrial wall (close to sinus node) for atrial stimulation, and for recording of high right atrial...
PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

Clinical Features and Electrocardiographic Findings

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Cardiovascular diagnosis</th>
<th>ECG</th>
<th>Atrial enlargement</th>
<th>Clinically documented PSVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>66</td>
<td>M</td>
<td>ASHD</td>
<td>RBBB + LASH</td>
<td>No</td>
<td>140 Positive in leads II, III, aVF</td>
</tr>
<tr>
<td>2</td>
<td>37</td>
<td>M</td>
<td>Mitral stenosis</td>
<td>Pre-excitation (Type A)</td>
<td>Yes (L)</td>
<td>130 Positive in leads II, III, aVF</td>
</tr>
<tr>
<td>3</td>
<td>13</td>
<td>M</td>
<td>Aortic stenosis</td>
<td>Pre-excitation (Type A)</td>
<td>No</td>
<td>Not documented</td>
</tr>
<tr>
<td>4</td>
<td>61</td>
<td>M</td>
<td>HHD</td>
<td>RBBB</td>
<td>Yes (L)</td>
<td>120 Positive in leads II, III, different from sinus</td>
</tr>
<tr>
<td>5</td>
<td>63</td>
<td>F</td>
<td>ASHD</td>
<td>Old antero-septal myocardial infarction, nonspecific ST-T changes</td>
<td>Yes (L)</td>
<td>140 Positive in lead II, different from sinus</td>
</tr>
</tbody>
</table>

Abbreviations: PSVT = paroxysmal supraventricular tachycardia; ASHD = arteriosclerotic heart disease; HHD = hypertensive heart disease; RBBB = right bundle branch block; LASH = left anterior superior hemiblock; L = left atrium.

electrograms. When retrograde conduction was studied, the latter catheter was advanced to the right ventricle for ventricular pacing. Electrocardiographic leads I, II, III, and V₁, as well as high right atrial and His bundle electrograms were simultaneously recorded on a multichannel oscilloscopic photographic recorder (Electronics for Medicine DR-16, White Plains, New York), at paper speeds of 100 and 200 mm/sec. Recordings were also recorded on an 8-channel tape system (Honeywell 5600), to facilitate subsequent analysis.

The atria were paced at a rate slightly faster than sinus rhythm. Pacing rates were increased at 10 beats/min increments until A-V nodal Wenckebach periods were observed. Refractory periods and echo zones were determined, utilizing extra-stimulus technique, during sinus rhythm and at one or more driven cycle lengths shorter than sinus rhythm.¹,²,¹³,¹⁴ The test stimulus (S₂) was introduced after every tenth driven (S₁) or spontaneous sinus beat. The coupling interval was decreased in 5 to 10 msec increments. Test stimuli were repeated several times at critical coupling intervals to accurately identify echo zones. Retrograde conduction was studied in three of the patients. The ventricles were paced at a rate slightly faster than sinus rhythm, and pacing rates were increased in 10 beats/min increments.

Electrophysiological Definitions

HRA₁, A₁, H₂, and V₁ are the high right atrial, the low right atrial, the His bundle, and ventricular electrograms, respectively, of sinus or driven beats. S₁ was the stimulus artifact of the driven beats. HRA₂, A₂, H₃, and V₂ were the electrograms in response to the extra-stimulus (S₂). Conduction intervals and refractory periods were defined as previously described.¹³,¹⁴

An echo zone was defined as the zone in which atrial extra-stimuli induced atrial echoes, with or without PSVT (rate ≥ 100 beats/min).¹,² This zone was defined in terms of its longest and shortest A₁-A₂ coupling interval. Sustained PSVT was defined as an episode of induced PSVT lasting longer than two minutes.

Since the diagnosis of sinus or atrial re-entrance in the present series of cases will depend upon exclusion of A-V nodal re-entry and re-entrance using anomalous pathways (in the two pre-excitation cases), criteria for the above diagnosis are presented in this section.

Criteria for the diagnosis of A-V nodal re-entrance in the catheterization laboratory would include the follow-

In all five patients, an echo zone, with sustained PSVT following the initiating echo, was defined with atrial extra-stimulus technique (table 2, and figs. 1-4). This echo zone could be demonstrated only during atrial driving in two of the patients (Cases 1 and 2), and during both sinus rhythm and atrial driving in three of the patients (Cases 3-5). In all patients, the inner limit of the echo zone was the atrial functional refractory period (FRP), or a short zone of atrial vulnerability at a coupling interval close to the atrial FRP (fig. 1C).

In all five patients, PSVT was observed following sudden cessation of atrial pacing at a critical rate or range of rates (table 2 and figs. 5-7). In all the patients, the lowest pacing rate inducing PSVT was slower than the pacing rate inducing A-V nodal Wenckebach periods (or normalization of conduction in the two patients with pre-excitation). When PSVT was also induced following cessation of pacing at

Results

Induction of Paroxysmal Supraventricular Tachycardia

In all five patients, an echo zone, with sustained PSVT following the initiating echo, was defined with atrial extra-stimulus technique (table 2, and figs. 1-4). This echo zone could be demonstrated only during atrial driving in two of the patients (Cases 1 and 2), and during both sinus rhythm and atrial driving in three of the patients (Cases 3-5). In all patients, the inner limit of the echo zone was the atrial functional refractory period (FRP), or a short zone of atrial vulnerability at a coupling interval close to the atrial FRP (fig. 1C).

In all five patients, PSVT was observed following sudden cessation of atrial pacing at a critical rate or range of rates (table 2 and figs. 5-7). In all the patients, the lowest pacing rate inducing PSVT was slower than the pacing rate inducing A-V nodal Wenckebach periods (or normalization of conduction in the two patients with pre-excitation). When PSVT was also induced following cessation of pacing at
Atrial extra-stimulus (in msec)  Range of \( \Delta \)-E (msec)  AP rates with induction of PSVT (beats/minute)  Earliest AP rate with AVWP (beats/minute)  Ventricular pacing

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Cycle length</th>
<th>Echo zone (( \Delta )-A)</th>
<th>Atrial FRP</th>
<th>( \Delta )-E</th>
<th>PSVT (beats/minute)</th>
<th>AVWP</th>
<th>PSVT (beats/minute)</th>
<th>Ventricular pacing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>750 (Sinus)</td>
<td>0</td>
<td>250</td>
<td>530-560</td>
<td>160-200</td>
<td>190</td>
<td>No V-A conduction</td>
<td></td>
</tr>
<tr>
<td></td>
<td>545</td>
<td>280-180</td>
<td>180</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>780 (Sinus)</td>
<td>0</td>
<td>340</td>
<td>480-580</td>
<td>140-150</td>
<td>&gt;150*</td>
<td>Induction of PSVT</td>
<td></td>
</tr>
<tr>
<td></td>
<td>555</td>
<td>440-320</td>
<td>320</td>
<td></td>
<td></td>
<td></td>
<td>&gt;150 beats/minute.</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>700 (Sinus)</td>
<td>300-250</td>
<td>250</td>
<td>290-350</td>
<td>150-190</td>
<td>190†</td>
<td>1:1 V-A conduction</td>
<td></td>
</tr>
<tr>
<td></td>
<td>470</td>
<td>370-250</td>
<td>250</td>
<td></td>
<td></td>
<td></td>
<td>130 beats/minute.</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>700 (Sinus)</td>
<td>450-315</td>
<td>315</td>
<td>380-580</td>
<td>130-170</td>
<td>170</td>
<td>Not done</td>
<td></td>
</tr>
<tr>
<td></td>
<td>600</td>
<td>450-260</td>
<td>260</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>685 (Sinus)</td>
<td>400-325</td>
<td>325</td>
<td>390-500</td>
<td>150</td>
<td>&gt;150†</td>
<td>Not done</td>
<td></td>
</tr>
<tr>
<td></td>
<td>575</td>
<td>420</td>
<td>310</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Antegrade Kent Bundle Conduction up to a Pacing Rate of 150/min, faster rates not tested.
†Type II Antegrade Kent Bundle Block Occurred at a Pacing Rate of 150/min.
‡1:1 Conduction up to a Pacing Rate of 150/min, faster rates not tested.
§Faster pacing rates not tested.

**Abbreviations:** FRP = functional refractory period; AP = atrial pacing; PSVT = paroxysmal supraventricular tachycardia; AVWP = A-V nodal Wenckebach period; \( \Delta \)-E = the interval between atrial extra-stimulus (\( \Delta \)) and first echo (E) of PSVT.

Faster rates (those rates at which A-V nodal Wenckebach periods were occurring and/or normalization of conduction was noted), PSVT induction occurred, even when the last paced beat was blocked in the A-V node (in the patients without preexcitation) or blocked in both normal and anomalous pathways (in the pre-excitation patients) (figs. 5C and 6B).

Ventricular pacing was performed in three patients (Cases 1-3) (table 2). Ventriculo-atrial conduction was absent in Case 1, and present in Cases 2 and 3. In Case 2, with 1:1 ventriculo-atrial conduction, PSVT was induced during cessation of ventricular pacing at...
a rate comparable to the critical atrial pacing rate inducing PSVT (fig. 8). The induction of PSVT during ventricular pacing was not related to retrograde A-V nodal Wenckebach periods. In Case 3, who had 1:1 ventriculo-atrial conduction up to a pacing rate of 130 beats/min, PSVT was not induced, and faster pacing rates were not tested.

In all of the patients, multiple A-H intervals were noted during the study. These varying A-H were achieved during atrial pacing with 1:1 A-V nodal conduction, during paced A-V nodal Wenckebach periods (with frequent sudden cessation of atrial pacing in an attempt to identify concealed A-V nodal re-entry), and during coupled atrial stimulation in both sinus rhythm and during atrial driving. PSVT induction could not be correlated with achievement of a critical A-H interval in any of the patients. In the two patients with pre-excitation, the outer limit of the echo zone was longer than the Kent bundle effective refractory period.

**Figure 2**

Induction of paroxysmal sinus re-entrant tachycardia with atrial extra-stimulus in Case 3 with pre-excitation. S2 was coupled to sinus rhythm at a cycle length of 700 msec.

In panel A, A1-A2 was 310 msec, and PST was not induced. QRS of the sinus beats showed incomplete type A pre-excitation, and that of the test beat showed total pre-excitation.

In panel B, A1-A2 was 270 msec and PST was induced. Note that during PST, atrial depolarization sequence was from high to low, similar to that of the sinus P, and that P waves were conducted to the ventricles with total pre-excitation.

Characteristics of Induced PSVT

The rates of induced PSVT were between 114 and 143 beats/min (table 3). The cycle length of the PSVT was stable immediately following initiation. The interval between the inducing beat (during constant atrial pacing or extra-stimulus), and the first beat of the PSVT varied somewhat without demonstrable relationship to either the pacing rate or coupling interval (table 2).

The P wave during PSVT was similar to the P wave during sinus rhythm in three of the patients (Cases 1-3) (figs. 1,2,5,6, and 8). In these three patients, atrial activation sequence during PSVT was from high to low. In two of the patients, P polarity was positive in lead II during PSVT, but different from the sinus P (Cases 4 and 5) (figs. 3,4 and 7). In Case 5, the P wave was inverted in lead III (fig. 9D). Atrial activation se-
quences were low to high (but only slightly asynchronous) in Case 4, and simultaneous or slightly low to high in Case 5. Analysis of P wave morphology and atrial activation sequences allowed separation of PSVT into those with sinus node re-entry (Cases 1-3), and those with probable atrial re-entry (Cases 4 and 5).

In all five patients, the P wave preceded each QRS complex during PSVT. In the three patients without pre-excitation, the A-H interval during PSVT was appropriate for the rate as determined with atrial pacing (table 3). In the two patients with pre-excitation (Cases 2 and 3), PSVT with both wide (anomalous pathway conduction) and narrow (normal pathway conduction) QRS complexes was noted (fig. 6).

Termination of PSVT

In all five patients, tachycardia could be terminated by properly-timed single (fig. 4D) or double atrial stimulation, or with a short period of repetitive atrial pacing at a faster heart rate. In four of the five patients (Cases 1-4), tachycardia could also be terminated with carotid massage. In one patient, spontaneous cessation of PSVT was also observed (Case 3).

Comparison of Induced to Spontaneous PSVT (tables 1 and 3)

Spontaneous episodes of PSVT were documented in Cases 1, 2, 4, and 5. The rates of these spontaneous episodes of PSVT were similar to the rates of induced PSVT (fig. 9). In all the cases of spontaneous PSVT, the P wave morphology resembled that of the induced PSVT (except for Case 1, in which P wave morphology during spontaneous PSVT was not clearly visualized) (fig. 9). In case 2, both normal pathway and anomalous pathway QRS complexes were identified during spontaneous PSVT (fig. 9B).

Discussion

Disparity in the refractory periods of a conduction tissue can predispose to re-entry. An early impulse may encounter the effective refractory period of one pathway, conduct slowly in an alternate pathway, and then return in a retrograde or circular fashion, to re-excite the previously blocked pathway. In order for the re-entry to occur, the early impulse must come within a critical interval, so that block occurs in one pathway, while conduction delay in the other pathway is sufficient for the previously blocked pathway to recover for re-excitation.

Figure 4

Induction of atrial re-entrant paroxysmal tachycardia (PAT) with delineation of an echo zone and termination of PAT by atrial extrastimulus in Case 5. S₃ was coupled to sinus rhythm at a cycle length of 685 msec.

In panel A, A₁-A₂ was 410 msec, and atrial echo did not occur.

In panels B and C, A₁-A₂ was between 400 to 325 msec, atrial echo with (panel C) and without (panel B) PAT was noted. Note the atrial echo had antegrade P wave morphology different from sinus P wave and had simultaneous high and low atrial depolarization. Atrial FRP was 325 msec.

In panel D, a timed atrial extrastimulus (S) terminated PAT.
PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

A

NSR = 80/min.

B

Pacing HR = 160/min.

C

Pacing HR = 200/min.


Figure 5

Induction of paroxysmal sinus re-entrant tachycardia during cessation of rapid atrial pacing in Case 1. Panel A demonstrates normal sinus rhythm (NSR) at a rate of 80 beats/min. In panel B, sinus echoes with PST were induced during sudden cessation of rapid atrial stimulation (S) at heart rate (HR) of 160 beats/min. During PST, P wave morphology and atrial depolarization sequence were identical to sinus P. In panel C, pacing HR was 200 beats/min. PST was induced even when the initiating beat was blocked in the A-V node.

Re-entry can also be induced during cessation of rapid pacing, since rapid stimulation can increase dispersion of refractoriness in conducting tissues. Re-entry could occur in any portion of the atrioventricular conducting system. Re-entry has been demonstrated in the sinus node, A-V node, and His-Purkinje system, in both human and animal studies.1-11, 18-19 In addition, intra-atrial and intra-ventricular re-entry have been suggested to be mechanisms of tachycardia.20, 21

A-V Nodal Re-entry

Both animal and human studies using direct and indirect recording methods have presented evidence that A-V nodal re-entry is a common mechanism of PSVT.1-4, 22-27 Criteria for the diagnosis of A-V nodal re-entrant PSVT in the catheterization laboratory was presented under electrophysiological definitions. In addition, A1-A2, H1-H2 curves suggestive of dual A-V nodal pathways have been described in approximately half the patients meeting the above criteria for A-V nodal re-entrant PSVT.5, 6

Figure 6

Induction of sinus re-entrant paroxysmal tachycardia (PST) in Case 3 with pre-excitation. Panel A demonstrates NSR at a rate of 80 beats/min. QRS complex showed type A pre-excitation. In panel B, pacing HR was 200 beats/min. PST was induced even when the initiating beat was blocked in both normal and anomalous pathways. During PST, P wave morphology and atrial depolarization sequence were identical to sinus P. P waves were conducted with both the narrow and pre-excited QRS complexes during PST.

In the present series of five cases, the induced PSVT did not appear to reflect A-V nodal re-entrance for a number of reasons. In all cases, multiple A-H intervals were observed with atrial pacing and during extra-stimulus testing during sinus rhythm and at faster driven cycle lengths. Induction of PSVT was indepen-

Figure 7

Induction of paroxysmal atrial re-entrant tachycardia (PAT) during cessation of rapid atrial pacing in Case 4. In panel A, NSR at a rate of 82 beats/min was demonstrated. QRS complex showed RBBB and LASH. In panel B, PAT was induced during cessation of rapid atrial pacing at a rate of 160 beats/min. Note that during PAT, P wave morphology was different from sinus P and atrial depolarization sequence was slightly low to high.
Induction of paroxysmal sinus tachycardia (PST) during cessation of rapid ventricular pacing in Case 2 with pre-excitation. High right (HRA) and coronary sinus electrogram (CSA) are shown. In panel A, NSR at a rate of 77 beats/min was noted. QRS complex showed incomplete type A pre-excitation. In panel B, pacing HR was 160 beats/min. One-to-one ventriculo-atrial conduction was noted. Sinus echoes with PST developed during sudden cessation of ventricular pacing. Note the P wave morphology and atrial depolarization sequence during PST was identical to sinus P wave. QRS complexes during PST were conducted via normal pathway (the patient had RBBB during normal conduction).

Figure 8

Electrocardiograms during spontaneous episodes of paroxysmal supraventricular tachycardia (PSVT) in Cases 1, 2, 4, and 5.

Panel A (case 1): ECG lead II, showing PSVT induced by a premature atrial beat (4th beat). P waves during PSVT were positive, although detailed morphology was unclear. The rate of PSVT was 140 beats/min.

Panel B (case 2): ECG leads III (left side) and V1 (right side), showing PSVT recorded on two occasions. Lead III showed sinus P waves at a rate of 125 beats/min with narrow QRS complex. Lead V1 showed PSVT pre-excitation at a rate of 130 beats/min. P waves were unclear.

Panel C (case 4): ECG lead II showing termination of PSVT (rate = 120 beats/min) with resumption of sinus rhythm (last 3 beats). Note the P wave morphology was different from sinus P and was identical to the induced PAT in figures 3 and 7.

Panel D (case 5): ECG lead III, showing spontaneous induction of PSVT with a rate of 140 beats/min. During PSVT, P wave morphology was different from sinus P (1st and 3rd beats), and was similar to the induced PAT in figure 4. Premature ventricular beats (2nd, 4th, and 9th QRS complexes) were also noted.

Figure 9

Table 3

Characteristics of Induced Paroxysmal Sinus and Atrial Tachycardia

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Rate (beats/min)</th>
<th>P wave morphology</th>
<th>Atrial activation sequence</th>
<th>A-H interval (msec)</th>
<th>QRS complex</th>
<th>Mode of termination</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>During PSVT</td>
<td>During AP at equivalent rate</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>143</td>
<td>Sinus</td>
<td>H→L</td>
<td>120</td>
<td>Identical to sinus rhythm</td>
<td>C &amp; E</td>
</tr>
<tr>
<td>2</td>
<td>136</td>
<td>Sinus</td>
<td>H→L</td>
<td>150</td>
<td>Both narrow and anomalous QRS</td>
<td>C &amp; E</td>
</tr>
<tr>
<td>3</td>
<td>131</td>
<td>Sinus</td>
<td>H→L</td>
<td>170</td>
<td>Unclear*</td>
<td>C, E, &amp; S</td>
</tr>
<tr>
<td>4</td>
<td>114</td>
<td>Positive in II, III, Different from sinus</td>
<td>Slightly L→H</td>
<td>100</td>
<td>Identical to sinus rhythm</td>
<td>C &amp; E</td>
</tr>
<tr>
<td>5</td>
<td>133</td>
<td>Positive in II, Different from sinus</td>
<td>Simultaneous L and H or slightly L→H</td>
<td>150</td>
<td>Identical to sinus rhythm</td>
<td>E</td>
</tr>
</tbody>
</table>

*Pre-excitation at rate of 130/min.

Abbreviations: AP = atrial pacing; PSVT = paroxysmal supraventricular tachycardia; H = high; L = low; C = carotid massage; E = properly timed single or double atrial stimulations; S = spontaneous cessation.
Atrial re-entry (in the low atrium) seemed most likely as an explanation for the findings in this case. During the induced episodes of PSVT in our five cases, P waves preceded each QRS complex with an A-H interval appropriate for the rate of the PSVT, unless pre-excitation was present. This latter finding contrasts strikingly with the previously described cases of induced A-V nodal re-entrant PSVT.

Re-entry Utilizing Normal and Anomalous Pathways in Patients with Pre-excitation

Recent pathologic and electrophysiologic studies in patients with pre-excitation suggest the presence of two A-V connections, these being: 1) an anomalous pathway (the Kent bundle) in one of the A-V rings, and 2) the normal A-V node-His bundle pathway. The presence of an anomalous pathway predisposes to PSVT, in which the circus movement, utilizing both normal and anomalous pathways, occurs. In the usual case, an atrial impulse blocks in the anomalous pathway, and conducts in the normal pathway, with prolongation of P-R intervals, and normalization of QRS complex. The slow antegrade normal pathway conduction allows recovery of the anomalous pathway for retrograde conduction. During PSVT, the normal pathway serves as antegrade route, the anomalous pathway serves as the retrograde route, and QRS complexes are narrow. The reverse type of circus movement utilizing the anomalous pathway as antegrade and the normal pathway as retrograde is rare. In both types of PSVT, the atrial activation sequence is from low to high, and the P wave morphology is retrograde.

Circus movement PSVT did not appear to explain the induced PSVT in our two cases of pre-excitation in that: 1) P waves during the PSVT had an antegrade morphology, and a high to low sequence of atrial activation; 2) the echo zones did not coincide with the coupling intervals encompassing the anomalous pathways, and A-V nodal effective refractory periods; 3) both anomalous and normal QRS complexes were demonstrated during the same episode of PSVT; and 4) PSVT was induced with P waves blocked in both normal and anomalous pathways during atrial pacing induction.

Sinus Node and Atrial Re-entry

Microelectrode studies in isolated rabbit hearts by Han et al. have demonstrated evidence of sinus node re-entry. He suggested that an early impulse may fail to engage a part of the node, enter at another part, and traverse the nodal tissue so slowly that the emerging impulse could re-excite the atrium. Subsequently, evidence of sinus node re-entry in both animal and human hearts was provided by Bonke et al. and Paulay et al. These workers suggested that sinus node re-entry could be a mechanism of clinical PSVT.

Cases 1-3 of the present series appear to reflect sustained PSVT due to sinus node re-entry. In these three cases, the induction with atrial extra-stimuli and during atrial pacing, the prevention of PSVT by the early atrial response provided by stimulation during atrial vulnerability, and the conversion with timed extra-stimuli and carotid massage, are consistent with re-entry somewhere in the supraventricular conduction system. Transient acceleration of sinus activity due to shift of pacemaker within the sinus node or local release of catecholamine induced by stimuli are unlikely causes because the PSVT was induced only within the echo zone and within the range of critical pacing rates and was stable and maintained once induced. Atrioventricular nodal re-entry was excluded, as was Kent bundle re-entry. The P wave morphology being similar to sinus P, and the high to low sequence of atrial activation during PSVT in these three patients strongly suggested the sinus node or perisinus nodal tissue as the site for re-entrance. The induction during atrial pacing probably reflected a Wenckebach induction in the sinus node or its approaches (type 1 sino-atrial entrance block), analogous to the Wenckebach induction of A-V nodal re-entrant PSVT. The presence of organic heart disease in our patients suggested that diseased sinus nodal tissue might predispose to sinus node re-entry.

Circus movement of the impulse in the atrium has frequently been proposed as the underlying mechanism of some supraventricular tachycardias. Bonke et al. showed that a series of atrial discharges can be induced by an early premature stimulation in a small piece of atrial muscle. Allessie et al., using surface electrode recording and extra-stimulus techniques, clearly demonstrated sustained circus movement of an impulse in a small piece of atrial muscle containing no anatomical obstacle.

Cases 4 and 5 of the present series appeared to reflect atrial re-entrance. Re-entrance somewhere in the supraventricular conduction system was implied in these two cases by the modes of PSVT induction and conversion. This induction differed from repetitive atrial firing observed following stimulation with an extra-stimulus during the atrial vulnerable period. In the case of atrial vulnerability, the interval between A and the first atrial response is 250 msec or less, and intracardiac electrograms reveal short (or sometimes sustained) episodes of atrial fibrillation or flutter.

Sinus node re-entrance did not seem likely in these two cases, since P wave morphology and atrial activation sequences were not suggestive of re-entrance in-
itiated in, or close to the sinus node. However, the possibility that these cases (particularly case 4) reflected sinus re-entrance with a different mode of emergence of impulse out of the SA node, producing an alternation of atrial activation cannot be ruled out. In both cases, electrophysiological studies did not suggest A-V nodal re-entrance, despite the retrograde P morphology in Case 5. Since the S-A and A-V nodes were unlikely sites for re-entry in these two cases, the atria seemed to be the most likely site for sustained re-entry. Whether re-entry involved atrial myocardium, the internodal tracts, or electrophysiologically active tissue in the mitral valve, or venous connections of the atria, could not be determined. It could also not be determined whether sustained re-entry occurred in the right or left atria. The presence of organic heart disease and atrial enlargement in the three patients suggested that diseased atrial muscle and/or atrial stretch predisposed to sustained atrial re-entry.

The electrophysiological features of sinus and atrial re-entrant PSVT were virtually identical, except for the P wave morphology and atrial activation sequence during induced episodes of PSVT. Both sinus and atrial re-entrant PSVT as described had a number of similar characteristics to the more commonly encountered A-V nodal re-entrant PSVT. These common characteristics included induction with critically timed atrial extra-stimuli, or during rapid atrial pacing and conversion with critically timed atrial extra-stimuli and carotid massage. Both A-V nodal re-entrant and sinus or atrial re-entrant PSVT could also be induced with ventricular pacing; however, the former would be dependent upon induction of A-V nodal retrograde Wenckebach sequences, while the latter would be dependent upon intact 1:1 retrograde V-A conduction. Heart rates during induced sinus or atrial re-entrant PSVT appeared to be slower than those described for A-V nodal re-entrant PSVT. Organic heart disease was present in our five patients with sinus or atrial re-entrant PSVT. Whether organic heart disease is a prerequisite for the occurrence of sustained sinus or atrial re-entrance in man cannot be determined with the small number of patients studied.

Clinical Implications

This study suggests that sinus node and atrial re-entry may be mechanisms of clinical PSVT. The clinical behavior and pharmacological responses of paroxysmal sinus and atrial tachycardia are probably different from those of A-V nodal re-entrant PSVT. The presence of organic heart disease with or without atrial enlargement in these patients suggests that diseased atrial tissue and/or stretched atrium might predispose to paroxysmal sinus and atrial tachycardia.

This study also suggests that paroxysmal sinus and atrial tachycardia are mechanisms of clinical PSVT in patients with pre-excitation. This has important therapeutic implication in regard to current surgical interventions in patients with pre-excitation and recurrent PSVT. Patients with pre-excitation and paroxysmal sinus or atrial tachycardia would probably not benefit from surgical destruction of anomalous pathway. Secondly, paroxysmal sinus or atrial tachycardia could produce wide QRS PSVT resembling reversed type of circus movement in patients with pre-excitation. Accurate diagnosis of arrhythmias could only be achieved with electrophysiological studies and careful analysis of data.

References

15. Dubois D, Schoo L, Schultenin B, Wellens HJ: The role of premature beats in the initiation and the termination
26. JANCE MJ, VAN CAPELLE FJL, FREUD GE, DUBBER D: Circus movement within the A-V node as a basis for supraventricular tachycardia, as shown by multiple micro-electrode recording in the isolated rabbit heart. Circ Res 25: 403, 1971
33. SPACH MS, BARR RC, JEWETT PH: Spread of excitation from the atrium into thoracic veins in human beings and dogs. Am J Cardiol 30: 844, 1972
Demonstration of sustained sinus and atrial re-entry as a mechanism of paroxysmal supraventricular tachycardia.
D Wu, F Amat-y-leon, P Denes, R C Dhingra, R J Pietras and K M Rosen

Circulation. 1975;51:234-243
doi: 10.1161/01.CIR.51.2.234
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1975 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/51/2/234

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/