The Mechanisms of Supraventricular Tachycardia in Children

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SUMMARY The mechanisms of supraventricular tachycardia were investigated in 35 children. Intracardiac electrogams including His bundle potentials were recorded. Atrial pacing and single premature atrial stimuli were performed in the right atrium. Tachycardia was observed and the mechanism elucidated in 33 patients. The atrial activation sequence during tachycardia, including high right atrium, low lateral right atrium, low septal right atrium, and left atrium, together with the ability to initiate or terminate the tachycardia with premature stimuli, were keys to defining the mechanisms. The observed mechanisms included atrioventricular (A-V) node re-entry (8), sino-atrial node re-entry (5), re-entry through manifest or concealed lateral anomalous pathway (8), re-entry through A-V node bypasses (3), and atrial (7) and junctional (2) ectopic foci. The frequency of the various mechanisms of SVT is more varied in children than adults, with ectopic mechanisms being more common in children.

SUPRAVENTRICULAR TACHYCARDIA (SVT) is a common problem in children. It has been said to occur in one of every 25,000 children. The electrophysiologic mechanism of supraventricular tachycardia has been controversial for many years. It has been suggested that a rapid ectopic pacemaker is responsible for this dysrythmia; others have felt that it was due to re-entry in the sino-atrial or atrioventricular nodes. Recent electrophysiologic investigations have defined several mechanisms of supraventricular tachycardia in adults. These have included atrioventricular (A-V) node re-entry due to either dual A-V nodal pathways or reflection, sino-atrial node re-entry, re-entry in the His-Purkinje system, atrial ectopic foci, and re-entry through anomalous bypasses, either of the James or Kent type. To date, no studies have been reported with regard to the mechanism of supraventricular tachycardia in children utilizing the new electrophysiologic techniques.

It was the purpose of this investigation to elucidate the mechanisms of SVT in children using the techniques of His bundle recording, atrial pacing, and the extrastimulus method.

Materials and Methods

The studies were performed in 35 subjects ranging in age from one week to 18 years with either documented or suspected supraventricular tachycardia (SVT). There were 18 females and 17 males. Nine subjects had congenital heart disease, three of whom had undergone surgical repair, and four had a cardiomyopathy. The other 23 patients had no detectable heart disease. All cardioactive medications were discontinued 36 hours before the study. Seventeen patients were taking digoxin up to 36 hours prior to study; hence, about 50% of their serum digoxin probably remained at the time of study. Each subject was sedated with meperidine, 1 mg per pound, promethazine, 0.25 mg per pound, and chlorpromazine, 0.25 mg per pound (one-half hour prior to study). Catheters with either two, three, or four electrodes, 1, 5, or 10 mm apart were used to give bipolar electrograms. Depending upon the type of study one, two, or three catheters were inserted percutaneously into the femoral or brachial veins with fluoroscopic control, according to techniques previously described from this laboratory. The His bundle potential was recorded with one of the catheters, by previously described techniques, in each subject.

In 28 subjects a second catheter with four electrodes was positioned at the high right atrial-superior vena caval junction and used for both recording and atrial pacing. In three subjects the left atrium could be entered with the quadrirpolar catheter through an atrial communication. In two subjects a quadripolar catheter was positioned in the distal coronary sinus. The catheters were connected to a photographic recorder through a junction box. In 28 subjects, after control intervals had been recorded, sequentially more rapid high right atrial pacing was performed. Pacing was interrupted if 2:1 A-V block occurred, or if a dangerous decrease in arterial pressure was detected by the continuously monitored arterial pressure. The sinus node recovery time was then measured using previously described techniques. Next, single progressively more premature atrial stimuli were introduced using a Medtronic #5837 pacemaker. The cardiac cycle was tested from late diastole to the atrial effective refractory period.

In the other seven subjects, who were studied early in the series, no atrial pacing was attempted. Recordings were made during sinus rhythm and during spontaneous SVT.

Results

In 33 of the 35 subjects, it was possible to determine the electrophysiologic mechanism of the patients' tachycardia (table 1). In the other two subjects no episodes of spontaneous SVT occurred during the investigation and none could be induced with either atrial pacing or single atrial stimulation. Since it is not possible to define the mechanism of the dysrythmia if tachycardia is not recorded, no statement can be made as to the mechanism in these two subjects. A-V nodal re-entry was accepted as the mechanism if the following criteria were met (table 1):

1) SVT could be induced by a single premature atrial depolarization (PAD) which resulted in delayed conduction in the A-V node or by a similar A-V nodal delay caused by atrial pacing.
2) A low-to-high atrial activation sequence was present during SVT.

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3) If left atrial activation was recorded it followed low septal right atrial activation during SVT.

Eight subjects satisfied the above requirements. Figure 1 shows His bundle and atrial recordings during sinus rhythm and SVT in one of these patients. During tachycardia the low right atrium was activated before the high right atrium and the LRA-H interval was prolonged. Left atrial activation was recorded in only one of these subjects. Evidence for dual A-V nodal pathways was searched for, using the criteria of Denes et al.,11 but none was found in any of these subjects. In all of these patients the tachycardia was terminated by either single premature atrial stimulation (2) or rapid overdrive pacing (6).

Re-entry through a manifest lateral anomalous pathway was defined by the following criteria:

1) Wolff-Parkinson-White syndrome was present on the surface ECG or provoked by atrial pacing.

2) Single PADS resulted in tachycardia with a low-to-high atrial activation sequence.

3) During tachycardia the QRS became normal (fig. 2). Five subjects satisfied these criteria. In the four in whom it was performed, atrial pacing resulted in widening of the QRS, confirming anomalous excitation of the ventricle (fig. 2).

Re-entry through an A-V node bypass was diagnosed by the following criteria:

1) The surface ECG showed a short P-R interval and normal QRS (Lown-Ganong-Levine).

2) SVT was induced by a single PAC.

3) A low-to-high atrial activation sequence was present during SVT.

4) Atrial pacing resulted in a smaller than normal increase in LRA-H interval.

5) During SVT there was unexpectedly short conduction time, either antegrade or retrograde, through the A-V junction indicating that the anomalous pathway was part of the re-entry circuit.

Three subjects satisfied these criteria; all were found to

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**Table 1. Criteria for Diagnosis of Mechanism of Supraventricular Tachycardia**

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Init by PAC</th>
<th>Req A-VN delay</th>
<th>Warm-up seq</th>
<th>A-V dual</th>
<th>Diagnosed</th>
<th>LRA-H before LA</th>
<th>LRA-H before LLRA</th>
<th>LBBB slow rate</th>
<th>RBBB slow rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>A-VN re-entry</td>
<td>+</td>
<td>+</td>
<td>-</td>
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<td></td>
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<tr>
<td>SAN re-entry</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
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<tr>
<td>APR</td>
<td>+</td>
<td>-</td>
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<td>+</td>
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<td></td>
</tr>
<tr>
<td>Left Kent</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>±</td>
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<td></td>
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<tr>
<td>Right Kent</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>±</td>
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<tr>
<td>James</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
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<td></td>
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<tr>
<td>Atrial ectopic</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>±</td>
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<tr>
<td>Junctional ectopic</td>
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<td>-</td>
<td>+</td>
<td>+</td>
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</tbody>
</table>

Abbreviations: Init = initiated; Req = require; seq = sequence; PAC = premature atrial contraction; A-V = atrioventricular node; LSRA = low septal right atrium; HRA = high right atrium; LA = left atrium; LLRA = low lateral right atrium; LBBB = left bundle branch block; RBBB = right bundle branch block; SAN = sinoatrial node; APR = anomalous pathway re-entry.
have short A-H intervals and abnormally small increase in A-H interval with atrial pacing in agreement with previous reports.30, 31

Re-entry through a concealed lateral anomalous A-V conduction pathway. Three additional subjects with normal surface ECGs satisfied all the electrophysiologic criteria for re-entry. In each of them, however, recording of the left atrial activation showed that during tachycardia the left atrium

FIGURE 3. Induction of re-entry tachycardia in a patient with a concealed lateral anomalous pathway. Surface lead I, aVF, and V1 recorded with high right atrial (HRA), distal coronary sinus (DCS), His bundle electrograms (HBE), and femoral artery pressure (FAP). A premature atrial stimulus is introduced at a coupling interval of 340 msec, which results in a re-entry tachycardia at a cycle length of 330 msec. During SVT the left atrium (LA) is activated 93 msec after the ventricles while the low right atrium (LRA) is not activated until 139 msec after the onset of ventricular depolarization.
was activated before the low septal right atrium (fig. 3 and table 1). In the two subjects in whom it was performed, right ventricular premature stimulation resulted in earlier activation of the left atrium than the low septal right atrium.

In one subject the QRS pattern during tachycardia changed from that of LBBB to RBBB. Simultaneously, the rate of tachycardia increased (fig. 4), and the V-A conduction time decreased 80 msec. All of these observations favor a left-sided anomalous pathway which only conducted in a retrograde direction. In these subjects no indication of

![Image](http://circ.ahajournals.org/)
not conduct antegrade. In two of these subjects pacing from the coronary sinus also failed to produce pre-excitation of the ventricles.

**Sinus node re-entry** was identified as the mechanism of a patient's tachycardia when the following criteria were met:
1) Tachycardia or re-entry beats could be produced by single premature atrial depolarizations (fig. 5).
2) The atrial activation sequence of the re-entrant beats was from high-to-low and similar to the sinus beats.
3) If the P waves of the re-entrant beats could be seen, they were similar to sinus P waves.
4) The tachycardia could be terminated by rapid atrial pacing.

In the five patients in this series in whom sinus node re-entry was the mechanism of the tachycardia, two were later studied during spontaneous tachycardia and a normal high-to-low atrial activation sequence was identified.

In one patient in this group a high-to-low atrial activation sequence was further confirmed by epicardial atrial mapping during correction of her atrial septal defect (fig. 6).

**Automatic ectopic atrial tachycardia** was established in seven patients by the criteria of Goldreyer et al.:
1) Neither tachycardia nor re-entry beats were initiated, nor was tachycardia terminated by single PADs.
2) Overdrive pacing failed to permanently suppress the ectopic focus.
3) The initial complex of the tachycardia sequence had the same P wave morphology and atrial activation sequence as the ensuing complexes.

Seven subjects in our series satisfied the above criteria for ectopic atrial tachycardia. One had chaotic atrial rhythm with several different P wave configurations, and P-P intervals. Three patients (fig. 7) had frequent intermittent episodes of SVT. The other three had had chronic sustained tachycardia for several years.

**Nonparoxysmal junctional tachycardia** (NPJT) was seen in two patients. This type is characterized by periods of sinus rhythm alternating with periods of junctional rhythm at a
faster rate. Atroventricular node re-entry was excluded by
the following observations:

1) The onset of the NPJT was not abrupt but the junctional focus gradually increased the rate until it usurped the sinus mechanism.

2) There was no evidence of A-V node delay at the onset of tachycardia.

3) Atrial pacing and progressive premature atrial stimulation failed to induce or terminate the tachycardia.

Thus, in 35 children with supraventricular tachycardia, there were seven different mechanisms. The types of heart disease associated with each mechanism are listed in table 2.

**Discussion**

The techniques of His bundle recording, atrial pacing, and progressive premature atrial stimulation allowed identification of the mechanism of supraventricular tachycardia in 33 of 35 children studied. The frequency distribution of the mechanisms found in these children was apparently different from that found in adult patients by previous investigators. It is difficult to determine the exact incidence of each mechanism in adults from publications since each of the reports deals with only one mechanism. There have been few reports which give the incidence of the various mechanisms in adults. There have been two reports concerning five adult patients who had automatic ectopic atrial tachycardia documented by intracardiac recording.20,21 On the other hand, there have been many reports of A-V node re-entry, based on 29 patients.8-13 These studies concluded that A-V node re-entry accounted for most episodes of SVT. Our study documented equal prevalence of automatic atrial and A-V node re-entry tachycardias.

We found an incidence of SVT due to anomalous A-V conduction in our pediatric patients similar to that reported in adults. In all of our patients with WPW the mechanism of supraventricular tachycardia was by antegrade conduction through the A-V node-His pathway and retrograde conduction was through the anomalous pathway.

The exact mechanism of SVT in patients with the Lown-Ganong-Levine syndrome is not yet defined. Two of our patients, as well as all those in the literature, had prolonged antegrade atioventricular conduction during tachycardia which may indicate that the impulse is conducted antegrade through the normal A-V node pathway and retrograde over the anomalous pathway. In one of our patients the LRA-H remained short during tachycardia and the H-LRA conduction time accounted for most of the delay. In this patient there must have been antegrade conduction through the bypass tract and retrograde through the A-V node.

The occurrence of anomalous pathways which conduct only in a retrograde manner has only recently been reported.22 In order to exclude this mechanism in cases of junctional re-entry it is necessary to show that the low septal right atrium (LSRA) is activated before the left atrium and the low lateral right atrium (LLRA) during tachycardia. This demonstration was attempted only in two of our patients with A-V node re-entry, so it is possible that some of them represent additional examples of retrograde pre-excitation. None of the previously reported cases of A-V node re-entry in the literature has excluded this mechanism. It is important to detect this mechanism in patients since medical treatment differs and because these patients may be helped by surgical incision of the anomalous bundle.

Sinus node re-entry is another recently recognized mechanism of supraventricular tachycardia. In our series we found this mechanism to be almost as common as either A-V node re-entry or anomalous pathway re-entry. In two of the five patients with this mechanism there was an associated sinus bradycardia and prolongation of sinus node recovery after overdrive pacing,29 indicating sinus node disease.

Rosen and his associates have found evidence of dual A-V node conduction pathways in some patients with A-V node re-entry tachycardias.10-13 This has been documented by finding interruption in the usually smooth curve obtained when the interval between a sinus beat and a premature beat is plotted against the interval between the His potential conducted from the sinus beat to the His potential from the premature beat. These curves were plotted in all of our patients with A-V node re-entry tachycardia, but none exhibited an interruption in the curve.

The present study has shown that there are at least seven different mechanisms of supraventricular tachycardia in children. This information is important in the management of these arrhythmias since the different mechanisms respond to different medications. For example, S-A node re-entry appears to be best treated with quinidine rather than digoxin.37 In exceptional cases, identification of an anomalous pathway which only conducts retrogradely may allow surgical therapy.34 The technique used in this study may also be expanded by giving drugs during the study to patients in whom it is possible to initiate tachycardia. If, after a drug has been given, it is impossible to initiate tachycardia it is likely that this drug will be effective when given orally.38,39 A drug that is administered during the study that facilitates the induction of SVT is not likely to be effective when given orally.

**References**


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**Table 2. Classification of Supraventricular Tachycardia with Associated Heart Disease**

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Associated heart disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>A-V node re-entry(8)</td>
<td>N(6), ASD(1), P/O TOF(1)</td>
</tr>
<tr>
<td>S-A node re-entry(5)</td>
<td>N(3), ASD(1), P/O ASD(1)</td>
</tr>
<tr>
<td>Anomalous pathway re-entry</td>
<td>N(3), SV(1), Eb(1)</td>
</tr>
<tr>
<td>WPW(5)</td>
<td>N(2), TGA(1)</td>
</tr>
<tr>
<td>LGL(3)</td>
<td>N(3)</td>
</tr>
<tr>
<td>Retrograde Kent(3)</td>
<td>N(3)</td>
</tr>
<tr>
<td>Automatic atrial(7)</td>
<td>N(3), P/O ASD(1), CM(3)</td>
</tr>
<tr>
<td>NPJT(2)</td>
<td>VSD(1), CM(1)</td>
</tr>
</tbody>
</table>

Abbreviations: ASD = atrial septal defect; A-V = atioventricular; CM = cardiomyopathy; Eb = Ebstein's anomaly; LGL = Lown, Ganong, and Levine syndrome; N = normal; NPJT = nonparoxysmal junctional tachycardia; P/O = postoperative; S-A = sino-atrial; SV = single ventricle; TGA = transposition of great arteries; TOF = tetralogy of Fallot; VSD = ventricular septal defect; WPW = Wolff-Parkinson-White syndrome.
22. Wu D, Denes 12, Paulay 16, Childers 10, Wu 11, Denes 31:
31:
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