Sudden Sinus Slowing with Junctional Escape: A Common Mode of Initiation of Juvenile Supraventricular Tachycardia

ARTHUR M. LEVY, M.D., AND BARTHOLOMIEW J. BONAZINGA, M.D.

SUMMARY After noting bradycardia-induced supraventricular tachycardia (SVT) in two successive children with SVT, we analyzed Holter monitor recordings done on 66 children with suspected or proved SVT. Ten children had apparent reentry SVT. The most common mode of initiation (eight of 10 patients) was not premature atrial beats, but sudden sinus pause with a junctional escape beat (JEB), usually fused with the delayed sinus P wave, initiating the tachycardia. Electrophysiologic studies in five children who had this mode of initiation showed evidence of reentry in four, possibly by dual atrioventricular nodal (AVN) pathways. Since sudden sinus pause and JEB are relatively uncommon in adults, the disappearance of this phenomenon with age may be the most significant reason why children often have less tachyarrhythmia as they get older. Both propranolol and digoxin significantly increased the numbers of episodes of SVT in the three patients tested with serial Holter monitoring.

REENTRY supraventricular tachycardia (SVT) can be initiated by a number of mechanisms, but the most common is a premature atrial beat (APB) producing unidirectional block in one of two pathways. SVTs in childhood have been considered similar to those in adults, although Holter monitor data on children are more sparse. However, recent data demonstrate that dual atrioventricular nodal (AVN) pathways are extremely common in children.1,2 We were surprised to observe two successive children with SVT whose Holter monitor recordings showed SVT with a different mechanism of onset from the classic APB. Both had multiple episodes of sudden sinus pauses followed by a junctional escape beat (JEB) that fused with the delayed sinus P wave and initiated SVT. Review of all our Holter monitor tracings on children with suspected or proved SVT showed that this mode of initiation was the most common one in patients who had tachycardia during the recording. Five children had limited electrophysiologic studies as well, which demonstrated reentry as the most likely mechanism of the SVT. Three of these five children had serial Holter recordings during treatment with digoxin and propranolol because of the continued apparent failure of these standard antiarrhythmic agents to prevent recurrences, with documentation that therapy sometimes produced apparent worsening.

Material and Methods

The Holter records of 66 children 16 years and younger done between 1974 and 1980 because of suspected or proved SVT were analyzed. These children had no evidence of an underlying heart disorder. Thirteen children had episodes of SVT during the recording period, and these tapes were reviewed to determine the most likely type of tachycardia. Three patients were considered to have ectopic atrial tachycardia based on the presence of positive P waves preceding each QRS and sometimes associated with the “warm-up” phenomenon. Ten seemed most likely to have reentry SVT, and these form the basis for this report. Evidence for reentry included SVT starting after a period of impaired atrioventricular (AV) conduction after APBs (seen in only two), atrial echoes during sinus rhythm, and either no visible P waves or negative P waves occurring just after the QRS during SVT. The tapes from these 10 patients were carefully analyzed for the exact mechanism of onset of each episode of SVT.

Five of the 10 children underwent electrophysiologic evaluation, having had persistent problems with tachycardia despite antiarrhythmic therapy. The procedure was explained and signed consent was obtained. The children were sedated with an intramuscular demerol compound consisting of meperidine, 2 mg/kg, promethazine, 0.5 mg/kg, and chlorpromazine, 0.5 mg/kg. Propranolol was stopped at least 24 hours before the study and digoxin at least 7 days before. Both bipolar and quadripolar electrode catheters were introduced percutaneously in the femoral vein. The bipolar catheter was positioned across the tricuspid valve to record His potentials, and the quadripolar catheter was positioned high in the right atrium near its junction with the superior vena cava. Four surface electrocardiographic leads and intracardiac electrograms from the high right atrium and the His bundle were recorded on an Elema multichannel recorder at a paper speed of 100 mm/sec. The right atrium was paced up to rates greater than 200 beats/min until AV block occurred or a tachycardia was instituted. Then, right atrial extrastimuli were delivered at decreasing coupling intervals of 10 msec during sinus rhythm and then during atrial pacing at rates of 100 and 130 beats/min. In the latter two instances, extrastimuli were given after nine paced beats. A programmable stimulator (Digitimer Co.) was used throughout the studies. The stimuli were delivered at twice diastolic threshold and were 2 msec in duration.

Finally, three of the patients who underwent electrophysiologic testing also underwent a series of 24-hour outpatient Holter monitor recordings while receiving no medication and then during therapy with propranolol alone (4 mg orally three times daily in younger patients and 10 mg four times daily in older patients),
digoxin alone (0.06 mg/kg loading dose and 0.015 mg/kg daily maintenance in younger patients and 0.125 mg/kg orally daily in older patients) and digoxin plus propranolol. Patients were treated for at least 1 week before each recording. These tapes were analyzed in real time both quantitatively for numbers of episodes of tachycardia and qualitatively with special attention to the mechanism of onset of each episode of tachycardia.

Results

Results of Initial Holter Analysis

Of the 66 children who underwent Holter monitor recordings for known or suspected SVT, 13 had episodes of SVT during the recording period. Ten of these 13 had apparent reentry tachycardia and the other three had atrial tachycardia. Based on the mechanism of onset of the tachycardia, three subgroups of patients were identified within this group of 10 patients. Group 1 consisted of six patients who had multiple episodes of SVT, always initiated by sinus pause and JEB, fusing with the delayed sinus P wave (fig. 1). Group 2 consisted of two patients who had tachycardia initiated by junctional beats that escaped after a sinus pause or, at other times, appeared to be accelerated rather than escape beats. Group 3 consisted of two patients who showed initiation of the tachycardia with APBs; one of these patients met the criteria for an incessant or permanent reciprocating SVT, i.e., a chronic SVT occasionally interrupted by only a few sinus beats, with negative P waves in inferior leads and a long RP' interval.

Results of Electrophysiologic Studies

Group 1

Four of the six patients in group 1 underwent electrophysiologic study (table 1). Three fulfilled Thapar and Gillette’s criteria for dual AVN pathways by demonstrating a discontinuous graph of A,A2 intervals plotted against H,H2 intervals during increasingly more premature atrial stimulation. These criteria include H,H2 interval increases of 40 msec or more for a decrement of 10 msec or less in A,A2 interval and a concomitant increase in conduction time represented by the A,A2 interval with no change in H,V intervals. In our patients, the sudden prolongation in H,H2 intervals varied as to whether it occurred during sinus rhythm with atrial stimulation or during atrial pacing with atrial stimulation. The single patient who did not have sudden H,H2 prolongation did have a Mahaim pathway. Evidence for reentry was found in three of the four studied patients in this group, either by initiating SVT with programmed atrial extrastimuli (two patients) or by spontaneous occurrence of SVT with retrograde P waves (one patient). In the two patients without a Mahaim pathway, the HV interval during tachycardia was normal and the VA intervals were 25 msec and 75 msec. Attempts to simulate the sequence seen in the Holter monitor tracings by rapidly stimulating the atrium to produce sinus suppression and junctional escape and initiate tachycardia were unsuccessful. Sinus node suppression was minimal in these children, and the sinus node rapidly took over with cessation of atrial pacing without JEBs.

Group 2

One of the two patients in group 2 (MB, table 1) underwent electrophysiologic testing as described above. This patient also had discontinuous H,H2 intervals during programmed premature atrial stimulation, as well as a reentry phenomenon (echo beats), normal HV intervals and a VA interval of 50 msec.

Group 3

No electrophysiologic studies were done in group 3.

Results of Pharmacologic Intervention on SVT Initiation

Two patients from group 1 and one patient from group 2 had serial Holter recordings on a program consisting of no medication, digoxin alone, propranolol alone, and combined digoxin-propranolol therapy (fig. 2). Every instance of SVT, defined as two or more echo beats after sinus pause and JEB, was counted. All three patients had more episodes of tachycardia with either drug alone or when combined than with no medications. In two patients, the most striking increase was with digoxin alone, and in the third one, the most striking increase was with propranolol alone.

Discussion

This study shows that bradycardia facilitating SVT appears to be very common in the pediatric age group. This discovery has a number of implications relating to the potential for disappearance of paroxysms of tachycardia with advancing age, worsening of the clinical situation with the use of the more common antiarrhythmic agents (digoxin and propranolol), and the search for newer forms of therapy aimed at smoothing sinus function rather than at preventing initiating APBs or changing the electrophysiologic characteristics of reentrant pathways.

The finding of frequent sinus pauses with JEBs in children is not unexpected, especially in view of some
TABLE 1. Data on the Children with Apparent Reentry Supraventricular Tachycardia

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age at onset</th>
<th>Age at 1st Holter</th>
<th>EPS</th>
<th>Dual AVN pathways</th>
<th>Reentry demonstrated</th>
<th>Episodes/24 hr on antiarrhythmic Rx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>JG</td>
<td>6 mo</td>
<td>4 yr</td>
<td>Yes</td>
<td>No (Mahaim)</td>
<td>Yes</td>
<td>Prop ↑, Dig ↑, Prop-Dig ↑</td>
</tr>
<tr>
<td>RD</td>
<td>4 yr</td>
<td>5 yr</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>—</td>
</tr>
<tr>
<td>JJ</td>
<td>2 yr</td>
<td>2 yr</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Prop ↑, Dig ↑, Prop-Dig ↑</td>
</tr>
<tr>
<td>WB</td>
<td>4 yr</td>
<td>5 yr</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>—</td>
</tr>
<tr>
<td>JB</td>
<td>2 yr</td>
<td>2 yr</td>
<td>No</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>JM</td>
<td>12 yr</td>
<td>12 yr</td>
<td>No</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Group 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MB</td>
<td>6 yr</td>
<td>10 yr</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Prop ↑, Dig ↑, Prop Dig ↑</td>
</tr>
<tr>
<td>SS</td>
<td>6 yr</td>
<td>6 yr</td>
<td>No</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Group 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>JM</td>
<td>2 yr</td>
<td>4 yr</td>
<td>No</td>
<td>—</td>
<td>—</td>
<td>(Incessant reciprocating SVT)</td>
</tr>
<tr>
<td>AP</td>
<td>8 mo</td>
<td>1½ yr</td>
<td>No</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Abbreviations: SVT = supraventricular tachycardia; JEB = junctional escape beat; APB = atrial premature beat; AVN = atrioventricular nodal; Prop = propranolol; Dig = digoxin; EPS = electrophysiologic studies. ↑ = increased number of episodes of SVT (number of arrows denotes degree).

recent studies. Southall et al. reported on electromagnetic electrocardiographic tape recordings on newborns, and in children ages 7–16 years. Seventy-one healthy, full-term neonates underwent 24-hour recordings and 89% had sinus pauses. In the study of 92 healthy school children ages 7–12 years, 65% had sinus pauses that appeared to be sinoatrial exit block or sinus arrest. The frequency of JEBs in normal children is unknown, although in a study of newborns, Välimäki and Tarlo found that nodal escape beats, chiefly during bradycardia, were the most common rhythm alteration.

The lack of specificity of the criteria for dual AVN pathways in children based on discontinuous H1H2 intervals during programmed atrial stimulation places the anatomic site of the tachycardia circuit in doubt. However, the mechanism of the SVT in our patients was thought to be reentry, for four of the five patients who underwent electrophysiologic studies had reentry phenomena during the study (i.e., initiation of SVT or atrial echo beats with APBs). Figure 3 shows two possible mechanisms for initiation of tachycardia. In figure 3A, the sinus P wave after the sudden pause is responsible for initiating the tachycardia. The prolonged PR interval is produced by the JEB, which presumably entered the fast pathway in a dual pathway. This explanation would fulfill an important criterion for diagnosing junctional reentry tachycardia, i.e., SVT starting after a period of impaired AV conduction. In figure 3B, the sinus P wave is totally blocked by conduction of the JEB into the AV node. The JEB itself initiates the tachycardia by retrograde conduction in one limb of a dual pathway that is sufficiently delayed to allow return to the ventricles in the other limb.

Coumel et al. and Krikler and Curry described the initiation of SVT by His bundle escape beats in adult patients with Wolff-Parkinson-White syndrome (WPW), although it is postulated that this phenomenon could occur with dual AVN pathways as well. These authors suggested that this mechanism applies particularly to older subjects in whom the JEBs, perhaps originating in the His bundle, occur more frequently as a consequence of disease affecting proximal physiologic pacemaking cells, i.e., sinoatrial disease. Our experience suggests it also applies to young subjects without evidence of WPW in whom JEBs occur more frequently as a consequence of an apparently normal or physiologic rather than pathologic irregularity of the proximal pacemaking cells. Since young and middle-aged adults demonstrate much less sudden sinus slow-

![Figure 2](http://circ.ahajournals.org/) Number of episodes of supraventricular tachycardia in three patients during four 24-hour ambulatory ECG recordings. Each patient had a control recording followed by recordings while on three drug regimens: digoxin, propranolol, and digoxin plus propranolol.
ing in ECG recordings, this may be a major reason behind the observation that children with recurrent SVT frequently have less and less difficulty as they grow older.

There is evidence that digitalis and propranolol are frequently ineffective in the long-term prevention of paroxysmal SVT. Lubbers et al. found that in their eight children with proved AVN reentry tachycardia, five had many (and often severe) recurrences, despite prophylaxis with either digoxin alone or in combination with propranolol or quinidine. One would certainly be concerned that in the presence of bradycardia-induced tachycardia as described above, digoxin or propranolol or both might indeed have the paradoxical effect of producing more frequent episodes of tachycardia by promoting more sinus bradycardia or, in the case of digoxin, by accelerating junctional escape. Counsel et al. expressed the same concern in their older patients with WPW whose SVT was initiated by sinus slowing and junctional escape and stated that depressant antiarrhythmic drugs in this setting may paradoxically induce incessant tachycardias.

Our serial Holter studies on three patients did indeed show some alarming worsening during treatment with digoxin or propranolol or both (fig. 2). In every instance, these drugs, alone or combined, caused a significant increase in numbers of episodes of tachycardia initiated by sudden pause plus JEB. Overall, children could improve while taking these drugs because they might still prevent sustained tachycardias, which are the only ones that result in significant hemodynamic impairment in this age group. In fact, one child has not had sustained SVT while taking digoxin (MB), one is doing well without therapy (JJ), and one has been lost to follow-up (JG). Although these three children all had episodes of both sustained and nonsustained tachycardia, the greatest increase in episodes during therapy was in the nonsustained variety. Perhaps totally different antiarrhythmic agents should be used in children with SVT who have this mechanism of onset, for the problem is really the bradycardia and junctional escape. Primary chronotrophic agents might be considered. Hydralazine increases heart rate, both as a reflex response to lowered systemic pressure and as a primary chronotrophic agent. It has been tried on a limited basis in sick sinus syndrome with some success.

In conclusion, careful review of Holter monitor recordings done in our hospital suggests that SVT in pediatric patients is often induced by bradycardia. As sinus rhythm becomes more regular with increasing age, this mechanism may well disappear, resulting in fewer episodes of tachycardia. (No episodes of SVT with this mode of onset have been found in over 16,000 adult Holter recordings scanned by our group.) Standard drug therapy may be harmful rather than beneficial given this mechanism of onset. Therefore, newer agents should be investigated.

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References

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