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

ARTHUR M. LEVY, M.D., AND BARThOLOMew J. BonazInG A, 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 electrophysiol- ogic 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),

From the Cardiology Division, Medical Center Hospital of Vermont, University of Vermont College of Medicine, Burlington, Vermont.
Address for correspondence: Arthur M. Levy, M.D., Cardiology Division, Medical Center Hospital of Vermont, Burlington, Vermont 05401.
Received October 15, 1981; revision accepted July 8, 1982.
Circulation 67, No. 1, 1983.

84
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 A1A2 intervals plotted against H1H2 intervals during increasingly more premature atrial stimulation. These criteria include H1H2 interval increases of 40 msec or more for a decrement of 10 msec or less in A1A2 interval and a concomitant increase in conduction time represented by the A3H3 interval with no change in H3V3 intervals. In our patients, the sudden prolongation in H1H2 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 H1H2 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 H1H2 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.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** 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.

---

Visually interpreting delimiters of table

Visually interpreting delimiters of figure

Visually interpreting delimiters of equation

Visually interpreting delimiters of graph

Visually interpreting delimiters of figure

Visually interpreting delimiters of table

Visually interpreting delimiters of equation

Visually interpreting delimiters of graph

Visually interpreting delimiters of figure
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 combina-
tion with propranolol or quinidine. One would cer-
tainly be concerned that in the presence of bradycardia-
duced tachycardia as described above, digoxin or
propranolol or both might indeed have the paradoxical
effect of producing more frequent episodes of tachy-
cardia 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 in-
stance, these drugs, alone or combined, caused a sig-
nificant 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 tachy-
cardia, the greatest increase in episodes during therapy
was in the nonsustained variety. Perhaps totally differ-
ent antiarrhythmic agents should be used in children
with SVT who have this mechanism of onset, for the
problem is really the bradycardia and junctional esca-
pe. Primary chronotropic agents might be consid-
ered. Hydralazine increases heart rate, both as a reflex
response to lowered systemic pressure and as a primary
chronotropic agent. It has been tried on a limited
basis in sick sinus syndrome with some success.

In conclusion, careful review of Holter monitor recor-
dings done in our hospital suggests that SVT in pediat-
ric 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.

Acknowledgment

We are indebted to Diane Prevost, R.N., for her expert help in
analyzing electromagnetic ECG tape recordings described in this
manuscript.

References

1. Thapar MK, Gillette PC: Dual atrioventricular nodal pathways: a
   common electrophysiologic response in children. Circulation 60:
   1369, 1979
2. Casta A, Wolff GS, Mehta AV, Tamer D, Garcia OL, Pickoff AS,
   Ferrer PL, Sung RJ, Gelband H: Dual atrioventricular nodal path-
   ways: a benign finding in arrhythmia-free children with heart
   Shinebourne EA: Supraventricular tachycardia in healthy newborn
electrocardiographic study of heart rate and rhythm patterns in
5. Välimäki I, Tarlo PA: Heart rate patterns and apnea in newborn
tachycardias in Wolff-Parkinson-White syndrome. II. Role of
   atypical cycle length dependency and nodal-His escape beats in
   initiating reciprocating tachycardias. Br Heart J 38: 897, 1976
7. Krikel D, Curry P: Atypical initiation of reciprocating tachy-
cardias in the Wolff-Parkinson-White syndrome. In Re-enterant Ar-
   rythmias, Mechanisms and Treatment, edited by Kulbertus HE.
   Baltimore, University Park Press, 1976, pp 144-152
8. Mandel WJ, Laks MM, Obayashi K, Bilder P: Prolongation of the
tachycardia zone. Eur J Cardiol 3: 117, 1975
9. Lubbers WJ, Losekoot TG, Anderson RH, Wellens HJ: Paroxys-
   mal supraventricular tachycardia in infancy and childhood. Eur J
   Cardiol 2: 91, 1974
10. Khatri I, Venura N, Notargiacomo A, Freis ED: Direct and reflex
cardiostimulating effects of hydralazine. Am J Cardiol 40: 38,
    1977
11. Weiss AT, Rod JL, Gotsman MS, Lewis BS: Hydralazine in the
    management of symptomatic sinus bradycardia. Eur J Cardiol
    12: 261, 1981
Sudden sinus slowing with junctional escape: a common mode of initiation of juvenile supraventricular tachycardia.

A M Levy and B J Bonazinga

Circulation. 1983;67:84-87
doi: 10.1161/01.CIR.67.1.84

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 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/67/1/84