Supraventricular Tachycardia in Infancy

Evaluation, Management, and Follow-up

Susan P. Etheridge, MD; Victoria E. Judd, MD

Background: Supraventricular tachycardia (SVT) occurs frequently in infancy. However, some infants have no recurrences after the initial presentation of SVT, and approximately 30% of infants lose SVT inducibility by 1 year of age.

Objective: To determine whether features at presentation, tachycardia characteristics, or data from an esophageal electrophysiology (EP) study could predict which infants will not require antiarrhythmic medication and which infants will not have inducible SVT at 1 year.

Design: Clinical and tachycardia characteristics at presentation of SVT and data obtained from an esophageal EP study were evaluated prospectively. Patients were followed up for 1 year, and an esophageal EP study was performed to evaluate for continued SVT inducibility.

Setting: Primary Children’s Medical Center is a tertiary care hospital affiliated with the University of Utah that provides primary care to local patients and is a referral center for a 4-state region.

Patients: All infants aged 3 months or younger who presented with SVT between August 1995 and October 1997 were evaluated.

Interventions: An esophageal EP study was performed at diagnosis and at 1 year.

Results: The SVT was controlled in all 33 infants. At the initial esophageal EP study, the mechanism of SVT was atrioventricular node reentry in 5 patients (15%) and orthodromic reciprocating tachycardia via an accessory atrioventricular connection in 28 patients (85%). One infant was lost to follow-up, 5 never required medication, 11 had SVT controlled with propranolol hydrochloride, 10 had SVT controlled with amiodarone, and 6 required more than 1 medication. Of the 21 patients who have reached 1 year of age, 16 (76%) were not taking any medication and were free of SVT at the time of follow-up. All 16 patients without clinical SVT have undergone a follow-up esophageal EP study, and 11 of 16 had inducible SVT on esophageal EP study. Thus, of the 21 one-year-old patients, 5 (24%) no longer had clinical or inducible SVT.

Conclusions: Control of SVT was possible in all patients. Clinical episodes of SVT were uncommon after discharge, yet most still had inducible SVT at 1 year of age. No data at presentation or initial esophageal EP study were predictive of the clinical course or of continued SVT.

Abbreviations: EP = electrophysiology, SVT = supraventricular tachycardia

Editor’s Note: Okay, so we need a different crystal ball.

Catherine D. DeAngelis, MD

From the Department of Pediatrics, University of Utah School of Medicine, Primary Children’s Medical Center, Salt Lake City.
PATIENTS AND METHODS

PATIENT POPULATION

This was a prospective evaluation of all infants aged 3 months or younger who presented to Primary Children’s Medical Center, Salt Lake City, Utah, with SVT between August 1995 and October 1997. Infants with atrial flutter were excluded. Approval for this study was obtained from the institutional review board at the University of Utah and Primary Children’s Medical Center, and parental consent was obtained. We evaluated data at presentation including age, tachycardia rate and QRS morphology, signs and symptoms of congestive heart failure, and mechanism of successful and sustained tachycardia termination. All infants had either a 12-lead electrocardiogram (ECG) or rhythm strip in tachycardia and a 12-lead ECG in sinus rhythm to evaluate for the presence of ventricular preexcitation. The association of ventricular preexcitation and SVT establishes the diagnosis of Wolff-Parkinson-White syndrome (WPW). A complete echocardiogram was performed to evaluate for congenital heart disease and to assess ventricular function.

INITIAL ESOPHAGEAL EP STUDY

During the initial hospitalization, an esophageal EP study was performed on all infants in the absence of antiarrhythmic medication. The technique for performing an esophageal EP study has been described previously.1-3 Parental informed consent was obtained and intravenous access was secured. The study was performed with all patients in the fasting state and with some patients under light sedation by means of intravenous midazolam hydrochloride (0.1 mg/kg). An esophageal pacing electrode was placed through the infant’s nostril to a depth estimated by the infant’s length.2 Using the esophageal pacing catheter, premature atrial extrastimuli and incremental atrial pacing were performed to induce SVT. Tachycardia was initiated with esophageal pacing in all patients, although isoproterenol hydrochloride (0.1 µg/kg per minute) was used in 1 infant in whom SVT was not initiated in the baseline state. To determine the tachycardia mechanism, the ventriculoatrial (VA) interval was measured on the esophageal lead. The VA esophageal interval is the time in milliseconds that it takes the electrical impulse to travel backwards from the ventricle to the atrium during SVT. A VA esophageal interval of less than 70 milliseconds excludes the presence of an accessory connection,3,4 making AV node reentry tachycardia the likely mechanism of SVT. A long VA esophageal interval (≥70 milliseconds) suggests the presence of an accessory AV connection. The VA interval is longer because the electrical impulse must travel through the ventricular myocardium before reaching the accessory AV connection to travel to the atrium.

If a wide QRS tachycardia with a bundle branch block (BBB) morphology was seen during SVT, the VA esophageal interval with BBB was compared with the VA esophageal interval with narrow QRS tachycardia.3,6 Lengthening of the VA interval with a BBB localizes the accessory connection to the side ipsilateral to the BBB. Tachycardia was terminated with esophageal pacing in all patients. Subsequent to the esophageal EP study, patients remained in the hospital until the SVT was under control, which was defined as more than 3 days without sustained SVT.

CLINICAL FOLLOW-UP

One patient was lost to follow-up. The remaining patients were followed up in clinic. Follow-up included a detailed history of tachycardia episodes and a record of all medications, adverse effects of the medication, and length of therapy. The addition of antiarrhythmic therapy and all changes in antiarrhythmic therapy were noted. If there were no recurrences of SVT, medication was discontinued at approximately age 9 months to 1 year of age. A follow-up ECG and an esophageal EP study were performed at 1 year of age. The purpose of the follow-up esophageal EP study was to determine whether SVT was still inducible. If SVT was not initiated in the baseline state, isoproterenol (0.05 µg/kg to 0.1 µg/kg) was administered.

DATA ANALYSIS

The clinical and tachycardia characteristics at presentation and data from the initial esophageal EP study from patients who never required antiarrhythmic medications were compared with those from patients who required medications. Patients whose medication was discontinued successfully at age 9 months were compared with infants still receiving medication because of continued episodes of SVT. Data from patients with continued clinical or inducible SVT were compared with data from patients without inducible SVT at the 1-year follow-up esophageal EP study. Comparisons between the groups were performed using the Student t test for continuous variables and the Fisher exact test for nominal data. P<.05 was considered significant. Data are expressed as mean ± SEM.

with ventricular septal defects, 1 with a left ventricular aneurysm, and 1 with a combination of a ventricular septal defect, coarctation of the aorta, and pulmonary stenosis. Four patients were born prematurely. At presentation, signs and symptoms of congestive heart failure were present in 10 patients. No patient with associated congenital heart disease presented with congestive heart failure. Of the 10 patients with congestive heart failure, 3 were in shock. Ten patients (30%) had WPW. With the use of the surface ECG and application of a well-known algorithm, the accessory connection location was determined to be in the posteroseptal region of the heart in 3 patients with WPW and on the left side in 7 patients.7

At presentation, the average SVT rate was 266 ± 6 beats per minute. Four patients presented with a wide QRS tachycardia. Successful and sustained termination of SVT was spontaneous in 5 patients. Vagal maneuvers were successful in 3, and ice to the face was successful in 1 patient. Adenosine was successful and sinus
rhythm was sustained in 14 patients. In 8 patients, adenosine resulted in the termination of SVT initially, but rapid recurrence of SVT necessitated the use of intravenous procainamide hydrochloride to sustain sinus rhythm. In 1 patient, a short burst of esophageal atrial pacing after a bolus of procainamide resulted in sustained sinus rhythm. One critically ill patient had reonset of tachycardia after adenosine administration and esophageal pacing. Procainamide, esmolol hydrochloride, and intravenous digoxin failed to terminate the SVT, but it was successfully controlled with intravenous amiodarone. All patients presenting with congestive heart failure regained normal cardiac function and did not have further symptoms once SVT was controlled. No patient required anticongestive medication at discharge.

INITIAL ESOPHAGEAL EP STUDY

Supraventricular tachycardia was inducible in all patients at the initial esophageal EP study. The SVT rate was 240 ± 6 beats per minute in the esophageal EP study. The average VA esophageal interval was 103 ± 7 milliseconds. The VA interval was 70 milliseconds or more in 28 patients (85%), which is consistent with the presence of an accessory AV connection and the diagnosis of orthodromic reciprocating tachycardia. 5 There were 5 infants with a VA esophageal interval of less than 70 (40-60) milliseconds, suggesting AV node reentry tachycardia. The VA intervals were constant unless there was a change in QRS morphology. During the esophageal EP study, both narrow and wide QRS tachycardias were seen in 16 patients. All wide QRS tachycardias had a left BBB morphology. In 14 of these patients, the VA esophageal interval increased by an average of 31 ± 4 milliseconds during tachycardia with left BBB morphology, suggesting that the tachycardia used a left-sided accessory connection. 5,6 The accessory connection was localized to the left side in 17 patients (52%). In 3 patients, the accessory connection was localized using the surface ECG alone, and in 10 patients it was localized by observing a lengthening of the VA esophageal interval during tachycardia with a left BBB morphology. In 4 patients, both the surface ECG pattern of preexcitation and VA prolongation supported the presence of a left-sided accessory AV connection. In 3 patients with WPW, the accessory connection was localized to the posteroseptal region. In 8 patients, the location of the accessory AV connection could not be established. There were no complications of the esophageal EP study. The infants remained in the hospital until SVT was controlled and ventricular function was normal.

CLINICAL FOLLOW-UP

One patient, who was not receiving medication, was lost to follow-up soon after being discharged from the hospital. The remaining 32 patients were followed up; of these, 11 were not receiving medication at the time of discharge. Figure 2 details the initial medication used and changes in medication made during follow-up. We used propranolol hydrochloride as the initial therapy in most of the infants who required long-term therapy. Propranolol was successful for control of SVT in 11 (55%) of the 20 patients in whom it was used. Treatment with oral amiodarone was successful in 10 (71%) of 14 patients when used as a single agent and in all 5 patients when used in combination with propranolol. Five infants never required medication, 11 were successfully treated with propranolol, 10 were successfully treated with oral amiodarone, and 6 required more than 1 medication for successful SVT control. There were no adverse effects of antiarrhythmic medication.

Twenty-one patients have reached 1 year of age. A total of 16 patients (76%) were not on antiarrhythmic medication at the 1-year follow-up and were free of SVT. Medication was discontinued at an average age of 9 months. Five patients (24%) continued receiving antiarrhythmic medication because of episodes of clinically significant SVT.
Follow-Up Esophageal EP Study

All 16 of the 21 patients who reached 1 year of age and were without clinical episodes of SVT underwent a follow-up esophageal EP study in the absence of antiarrhythmic medication. Five of the 16 did not have inducible SVT in the baseline state or after isoproterenol. The remaining 11 patients had inducible SVT. Five (24%) of 21 patients no longer had clinical or inducible SVT at 1 year of age. Eight patients in the group followed up for 1 year had WPW. Three of these patients did not have ventricular preexcitation at 1 year of age, but 2 continued to have inducible SVT during the esophageal EP study. All 3 patients with loss of preexcitation on the surface ECG had left-sided accessory connections at presentation.

Data Analysis

Infants requiring medication for control of SVT were compared with infants who did not require antiarrhythmic medications. Infants who needed medication after age 9 months were compared with those who no longer needed medication. Infants with clinical or inducible SVT were compared with infants in whom SVT could not be induced. Data at presentation, characteristics of the tachycardia, and data from the initial esophageal EP study were not predictive of the clinical course, need for medication, or continued SVT inducibility at the 1-year follow-up esophageal EP study. However, all 5 patients with AV node reentry tachycardia had inducible SVT at 1 year of age, and only patients with left-sided accessory AV connections lost ventricular preexcitation on the surface ECG.

Conclusions

Supraventricular tachycardia occurs in 1 of 250 to 1000 children, often presenting before the age of 4 months. Although episodes of SVT are common during infancy, most patients are free of tachycardia episodes during early childhood and some infants do not have a recurrence of SVT after the initial presentation. We were unable to identify factors at presentation, characteristics of the tachycardia, or data from an initial esophageal EP study that would allow us to predict which infants will have continued SVT recurrences and require antiarrhythmic medication and which infants will not need medication. However, all infants with the diagnosis of AV node reentry tachycardia continued to have inducible SVT at 1 year of age.

Several of the infants who were very ill at presentation and had SVT that was initially very difficult to control were easily managed with long-term medications and were successfully withdrawn from medications without recurrences of clinical SVT in infancy. Two of the infants who presented with congestive heart failure and SVT that was difficult to control had no clinical recurrences after discharge and did not have inducible SVT at the 1-year follow-up esophageal EP study. Neither the severity of the illness at presentation nor the presence of congestive heart failure or associated congenital heart disease were predictive of the clinical course or loss of SVT inducibility. Furthermore, neither SVT rate, VA esophageal interval during SVT, location of the accessory connection, nor mechanism of SVT were predictive of the clinical course or inability to induce SVT at follow-up. All patients with AV node reentry tachycardia remained inducible, but this group included too few patients to reach statistical significance.

In this series, both short-term and long-term control of tachycardia was possible in all infants. No infant required radiofrequency catheter ablation. It is rare to encounter an infant who is unresponsive to medical therapy. Although radiofrequency catheter ablation has been performed successfully in infants, they are at greater risk of morbidity and mortality than are older children and adults. Considering the natural history of SVT presenting in infancy, wherein some children lose the ability to have SVT by 1 year of age and many do not have recurrences of SVT until 5 to 10 years of age, aggressive attempts should be made to control tachycardia with medication.

Figure 2. Diagram of the medications used at the time of discharge from the hospital, results, and changes in medication during follow-up.
We evaluated the mechanism of successful long-term SVT control. Long-term antiarrhythmic medication was not required in 15% of patients. In an article by Benson and Deal, 10% of infants with SVT did not require treatment. In children with SVT episodes that were infrequent, non–life threatening, and self-terminating, long-term chronic antiarrhythmic medication is not always necessary or practical.

Few controlled trials have evaluated the efficacy of individual antiarrhythmic agents for a specific rhythm abnormality in pediatric patients. Most of the information about antiarrhythmic agents has been extrapolated from studies of adults. Because more clinical trials have evaluated experience with conventional antiarrhythmic agents, these are usually the initial agents chosen. We did not use digoxin because it is ineffective in the prophylaxis of SVT and is not recommended in patients with WPW. Many clinicians use beta blockers for management of SVT. We found that treatment with propranolol was successful for complete SVT control in only 11 (35%) of 20 patients in whom it was used. Treatment with oral amiodarone was successful in 10 (71%) of 14 patients when used as a single agent and in all 5 patients when used in combination with propranolol. Successful therapy requires compliance, and this makes treatment with oral amiodarone an ideal choice for many patients, because, after initial loading, it is administered only once per day. There were no major adverse effects of medical therapy and no patient had to stop taking medication because of adverse effects. All patients treated with oral amiodarone had liver function tests and thyroid function tests every 3 months and there were no abnormal findings.

Most patients presenting with SVT in infancy did not require medication beyond 9 months of age. Although clinical episodes of SVT were uncommon at 1 year and is not recommended in patients with WPW. Most patients presenting with SVT in infancy did not require medication beyond 9 months of age. Although clinical episodes of SVT were uncommon at 1 year of age, only 5 (24%) of 21 patients were not inducible. Benson et al found that 32% of infants with accessory AV connections were not inducible at a 1-year follow-up esophageal EP study and Ding et al reported that 4 of 7 patients were not inducible in a follow-up esophageal EP study. The higher rate of continued inducibility in our patients may be due to the inclusion of patients with AV node reentry tachycardia, all of whom were inducible at 1 year. Others have demonstrated a change in the conduction properties of accessory connections with age. Loss of function of the accessory connection in the retrograde direction results in loss of the ability to induce SVT, as was seen in 5 of 9 patients, including 1 with associated loss of preexcitation. Two other patients had the loss of anterograde accessory connection conduction demonstrated by loss of preexcitation on the surface ECG, but had continued SVT inducibility. Thus, both anterograde and retrograde properties of the accessory connections may undergo developmental changes.

As expected, the most common diagnosis in our patients was orthodromic reciprocating tachycardia using the AV node in the anterograde direction and an accessory AV connection in the retrograde direction. However, 15% of infants, all of whom were inducible at 1 year, had AV node reentry tachycardia. Although AV node reentry tachycardia is common in adults and older children, it has been thought to be rare or absent in child-

Accepted August 31, 1998.

We thank Kris Sjoblom, BS, for her support in the editing of the manuscript.

Corresponding author: Susan P. Etheridge, MD, Division of Pediatric Cardiology, University of Utah School of Medicine, Primary Children’s Medical Center, 100 N Medical Dr, Salt Lake City, UT 84113.

REFERENCES