Supraventricular Tachycardia Mechanisms and Their Age Distribution in Pediatric Patients

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To better define the natural history of supraventricular tachycardia (SVT) in young patients, age distribution of SVT mechanisms was examined in 137 infants, children and adolescents. Patients with a history of cardiac surgery or neuromuscular diseases were excluded. An electrophysiologic study was performed in each patient: transesophageal (110 patients) or transvenous (14 patients) or both (13 patients). Mechanisms were classified as SVT using accessory atrioventricular (AV) connection (SVT using accessory connection, including orthodromic and antidromic reciprocating tachycardia), primary atrial tachycardia (including chaotic, automatic and reentrant atrial tachycardia), and tachycardia due to reentry within the AV node. SVT using accessory connection occurred in 100 of 137 patients (73%) and was the most prevalent mechanism. Primary atrial tachycardia and reentry within the AV node were present in 19 of 137 (14%) and 18 of 137 (13%) patients, respectively. Using a multinomial logit model, relative probabilities for tachycardia mechanisms for 5 age groups — prenatal, <1, 1 to 5, 6 to 10 and >10 years — were determined. Primary atrial tachycardia (11 to 16%) and SVT using accessory connection (58 to 84%) appeared throughout infancy, childhood and adolescence. On the other hand, tachycardia due to reentry within the AV node (0 to 31%) rarely appeared before age 2 years. Mechanisms of SVT appear to have age-dependent distributions. SVT using accessory connection is the most common mechanism in young patients. We speculate that the propensity to tachycardia due to reentry within the AV node occurs during postnatal development.

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Supraventricular tachycardia (SVT) or paroxysmal atrial tachycardia, as it is also known, is a common abnormality of heart rhythm in infants, children and adolescents. In the past 2 decades, much has been learned regarding electrocardiographic and electrophysiologic features of SVT mechanisms.[1–9] It is now known that tachycardias requiring participation of both ventricular and supraventricular tissues have been termed SVT. For example, in SVT using an accessory atrioventricular (AV) connection, both atria and ventricles are essential for continuation of tachycardia. On the other hand, in SVT thought to result from reentry within the AV node, the precise role of the atria and ventricles in maintaining tachycardia is unclear. Finally, there are SVT types (e.g., ectopic atrial tachycardia), where the atria are the principal site of the electrophysiologic disturbance; these types have been termed atrial[8] or primary atrial tachycardias.[6]

It is generally agreed that accessory AV connections used in certain SVT types are developmental anomalies. However, much less is known of the developmental aspects of either primary atrial tachycardias or tachycardias due to reentry within the AV node. We hypothesized that SVT mechanisms are age-dependent. To test this hypothesis, we evaluated the frequency and age distribution of SVT mechanisms in pediatric patients.

METHODS

Patients: The records of electrophysiologic studies performed from July 1986 through January 1991 on 137 patients with electrocardiographically documented SVT were reviewed. There were 57 female and 80 male patients. Patients with electrocardiographic documented tachycardia with prolonged QRS duration and AV dissociation, as might be seen in patients with ventricular tachycardia or tachycardia using a nodoventricular connection, were not included.[10] Additionally, patients who had undergone open heart surgery, as well as patients known to have neuromuscular diseases were not included. However, we included 8 patients with nonsurgically treated congenital heart disease and 1 infant of a diabetic mother with hypertrophic cardiomyopathy.

Definition and diagnosis: Based on tachycardia characteristics, patients were divided into 3 groups: primary atrial tachycardia, SVT using accessory AV connection, and tachycardia due to reentry within the AV node. The classification of an SVT mechanism was made independently by 3 of the authors. SVT classification was based on analysis of the initiation method of SVT, electrophysiologic features, and response to pharmacologic agents or maneuvers that impaired AV or myocardial conduction.

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Primary atrial tachycardia[6] is that in which the primary electrophysiologic disturbance is restricted to atrial tissue, and it includes atrial ectopic tachycardia, intraatrial reentry tachycardia, atrial flutter and chaotic atrial tachycardia.[11] Electrophysiologic characterization was based on the presence or absence of 2° block during tachycardia, whether tachycardia could be induced or terminated by pacing, and the response to pharmacologic agents (e.g., adenosine) or maneuvers (e.g., Valsalva) that impaired AV conduction.

SVT using accessory connection includes orthodromic and antidromic reciprocating tachycardia, and the permanent form of junctional reciprocating tachycardia. Orthodromic reciprocating tachycardia is defined as a tachycardia with normal QRS, no evidence of AV dissociation, anterograde conduction over the AV node, and retrograde conduction through an accessory connection. During tachycardia, the ventriculoatrial interval, as measured from the onset of QRS to the rapid deflection of the atrial component of the transesophageal electrocardiogram, is >70 ms.[12,13] Prolongation of the cycle length or ventriculoatrial interval in the presence of bundle branch block ipsilateral to the accessory connection supported the diagnosis.[14] In addition, during transvenous electrophysiologic study, the presence of eccentric atrial activation and atrial preexcitation by ventricular extrastimuli at a time when the His bundle was refractory[15] supported the diagnosis of orthodromic reciprocating tachycardia. Antidromic reciprocating tachycardia shows a prolonged QRS duration with conduction anterograde over an accessory connection and retrograde conduction via either the AV node or an additional accessory connection; the QRS morphology resembles that present with preexcitation and can be duplicated with atrial pacing at a comparable rate. The permanent form of junctional reciprocating tachycardia is an incessant tachycardia considered to be a special case of orthodromic reciprocating tachycardia with anterograde conduction over the AV node and retrograde conduction over a septal accessory connection with decremental conduction properties[16]; the ventriculoatrial interval is prolonged, and there is no evidence of cycle length change with functional bundle branch block.

The diagnosis of tachycardia due to reentry within the AV node is made by excluding SVT using accessory connection and primary atrial tachycardia. In both typical (common) and atypical (uncommon) types, the critical elements of the reentry circuit are thought to be restricted to the region of the AV junction.[4,8,9,12,13] During the typical type, the ventriculoatrial interval during tachycardia is short whether measured during transvenous[12] or transesophageal[13] electrophysiologic study. The atypical form in association with longer ventriculoatrial intervals occurs less often.[9,12,13]

Electrophysiologic study: An electrophysiologic study was performed in each patient. Studies were performed in the postabsorptive and antiarrhythmic drug-free state. Before study, parental consent was obtained. During study, sedation was maintained using meperidine (1 mg/kg), promethazine (1 mg/kg), morphine (0.1 mg/kg) or midazolam (0.1 mg/kg) as needed. A transesophageal study was performed in 110 patients (2 days to 19 years), whereas in 14 patients (25 months to 17 years) a transvenous electrophysiologic study was performed. Both techniques were eventually performed in 13 patients (5 months to 19 years).

Transesophageal electrophysiologic study: The techniques for transesophageal pacing and electrocardiographic recording have been previously described.[17] A bipolar esophageal and 3 surface electrocardiograms were recorded using a strip-chart recorder at a paper speed of 50 and 100 mm/s. Anterograde conduction and refractory characteristics were assessed. During tachycardia, the esophageal electrocardiogram was recorded and special attention was paid to the atrial and ventricular cycle lengths and to the AV relationship. When appropriate, the ventriculoatrial interval was measured from the onset of QRS to the rapid deflection of the atrial waveform.

Transvenous electrophysiologic study: The techniques for transvenous electrophysiologic study using 3 or 4 percutaneously inserted quadripolar electrode catheters have been previously described.[18] Intracardiac electrograms were simultaneously recorded with a 3-lead electrocardiogram on a strip-chart recorder as in the transesophageal study. Anterograde and retrograde conduction and refractory characteristics were assessed, and atrial mapping procedure was performed at multiple sites in the right atrium and coronary sinus during both tachycardia and right ventricular pacing. Premature extrastimuli were administered in the right ventricular apex during tachycardia.

Pacing and pharmacologic maneuvers: Anterograde and retrograde conduction were assessed by pacing at cycle lengths ranging from slightly faster than the sinus cycle length to those producing 2° AV block. Refractory characteristics were assessed using the extrastimulus technique. To induce tachycardia, 1 or 2 atrial extrastimuli at progressively closer coupling intervals were introduced into normal sinus and paced rhythms, incremental atrial pacing to 2° AV block; burst pacing at cycle lengths similar to those producing AV block were performed. If tachycardia was not initiated under basal conditions, isoproterenol (0.02, 0.05, and 0.1 μg/kg/min to a maximal rate of 4 μg/min) was infused and the pacing protocol was repeated. If tachycardia was not initiated, atropine (0.04 mg/kg) was infused and the protocol was repeated.

Because the rationale for electrophysiologic study was to establish an electrophysiologic diagnosis and define therapeutic alternatives, the effects of the following intravenous medications were assessed during many studies: edrophonium (0.15 mg/kg), verapamil (0.15 mg/kg), propranolol (0.2 mg/kg), procainamide (15 mg/kg over 15 minutes) and adenosine (maximum 300 μg/kg). Additionally, in many patients the response to Valsalva maneuver was assessed.

STATISTICS

A multinomial logit model was used to evaluate the pattern of age distribution by SVT mechanism. Patients were divided into 5 age groups: prenatal, <1, 1 to 5, 6 to 10, and >10 years. For each age group, frequency estimates were generated for each tachycardia type and

conditional probabilities were computed from these frequency estimates. Models were examined to test for linear and quadratic effects. The difference in likelihood ratio statistics for logit models as well as comparing parameter estimates to their standard errors was used to determine the best model. The Kruskal-Wallis nonparametric test was used to compare the time interval between symptom onset and electrophysiologic study. Analyses were performed using SPSS PC+ (version 4.0).

RESULTS

An SVT mechanism was determined for each patient. In 135 of 137 patients (98%), there was uniform agreement between the 3 primary reviewers. In 2 patients, there was disagreement between the primary reviewers, and these 2 patients were classified by the majority opinion. SVT using accessory connection occurred in 100 of 137 patients (73%). Primary atrial tachycardia occurred in 19 of 137 patients, (14%) and tachycardia due to reentry within the AV node occurred in 18 of 137 patients (13%). SVT was first reported at a median age of 0.5 years (prenatal to 19 years); SVT was first detected at age <1 year in 78 patients (58%). SVT was recognized in utero in 21 of these 78 patients. Electrophysiologic study was performed at a median age of 4.3 years (1 day to 21 years), and in 51 of 137 patients (37%) the evaluation was performed at age <1 year.

Primary atrial tachycardia: Primary atrial tachycardia was present in 19 patients (8 female, 11 male). Tachycardia was present in 11 infants, and 4 of 11 infants had been diagnosed in utero. Electrophysiologic study was performed during the first year of life in 11 patients.

Tachycardia due to reentry within the atrioventricular node: This mechanism was present in 18 patients (9 female, 9 male). A classification difficulty was posed by 2 patients: a 7-year-old girl with block "in a lower final common pathway" associated with the typical form,[9] and a 13-year-old girl thought to have atypical form. The typical form was present in 17 patients. In 3 patients, tachycardia was first reported in infancy, but none was detected in utero. No patient had electrophysiologic study during the first year of life.

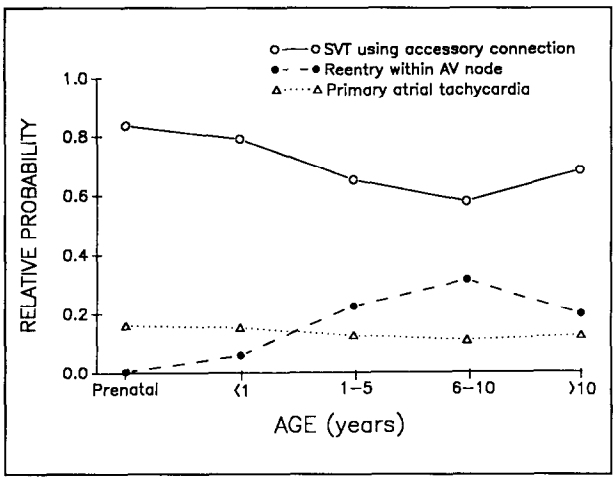

FIGURE 1. Graphic representation of the probability estimate of tachycardia type by age for supraventricular tachycardia (SVT) using accessory connection, tachycardia due to reentry within the atrioventricular (AV) node, and primary atrial tachycardia.

Supraventricular tachycardia using accessory connection: SVT using accessory connection was present in 100 patients (40 females, 60 males). Tachycardia was present in 64 infants and in 17 of 64 infants the diagnosis was in utero. Electrophysiologic study was performed in 40 patients during the first year of life. Ventricular preexcitation was present in 48 patients (48%).

Relation of tachycardia mechanisms with age: With use of a multinomial logit model, probabilities for tachycardia mechanisms by age were determined (Figure 1). Both the likelihood ratio chi-square statistic and the size of the parameter estimate, relative to the standard error indicated the quadratic model fit better than the linear model. SVT using accessory connection occured more often than the other 2 mechanisms (58 to 84%). Primary atrial tachycardia was relatively uncommon (11 to 16%) but appeared throughout infancy, childhood and adolescence. However, tachycardia due to reentry within the AV node (0 to 31%) rarely appeared before age 2 years.

Age at symptoms and age at study: The median interval between the age at symptom onset and the age at electrophysiologic study was 1.1 years (range 0.0 to 17.5). In 47% of patients, the interval was <1 year: 2 of 18 patients (11%) with tachycardia due to reentry within the AV node, in 14 of 19 patients (74%) with primary atrial tachycardia, and in 48 of 100 patients (48%) with SVT using accessory connection. Thus, patients with either SVT using accessory connection or primary atrial tachycardia tended to undergo electrophysiologic study within 1 year of symptom onset, whereas patients with tachycardia due to reentry within the AV node tended to undergo electrophysiology study >1 year after symptom onset ($p < 0.05$).

Comparison of transesophageal and transvenous electrophysiologic study: Both transvenous and transesophageal electrophysiologic studies were performed in 13 patients. Transesophageal study was performed initially, but transvenous study was eventually performed to evaluate suitability of ablation, antitachycardia pacemaker and investigational drug options. SVT using accessory connection was present in 10 patients, including orthodromic (6 patients), orthodromic and antidromic (1 patient), and antidromic-reciprocating tachycardia (1 patient), and permanent junctional reciprocating tachycardia (2 patients). Primary atrial tachycardia was present in 3 patients: atrial ectopic (1 patient) and intraatrial reentry (2 patients) tachycardia. In all 13 patients, the mechanisms of SVT determined by the 2 studies were identical.

Supraventricular tachycardia with associated heart disease: SVT using accessory connection was present in 9 patients with associated heart disease, including Ebstein's anomaly, scimitar syndrome, secundum atrial septal defect, primum atrial septal defect, and corrected transposition in 1 patient each and ventricular septal de-

fect in 2 patients. An infant of a diabetic mother had associated hypertrophic cardiomyopathy.

DISCUSSION

The main finding of this study in pediatric patients is that mechanisms of SVT appear to have age-dependent distributions. We grouped SVT patients into 3 major mechanisms: primary atrial tachycardia, SVT using accessory connection, and reentry within the AV node. Among the patients studied, SVT using accessory connection was the predominant mechanism (73%) of SVT, and most patients with SVT using accessory connection or primary atrial tachycardia developed symptoms before age 1 year. In contrast, tachycardia due to reentry within the AV node occurred nearly as often as primary atrial tachycardia, but no cases were detected in utero and symptoms rarely occurred before age 2 years.

In the patients we studied, SVT using accessory connection was more common than previous reports in children and adults.[1,2,8,9] Reentry within the AV node has been reported to be the most common mechanism of SVT in adults without ventricular preexcitation.[8,9,19] In our study of pediatric patients, this mechanism was more likely to occur in older children. The incidence of primary atrial tachycardia (14%) is also low compared to 21 to 34% present in previous studies in pediatric patients.[1,2]

There may be 2 reasons related to our patient selection to explain the predominance of SVT using the accessory connection. First, we were restrictive in patient selection and excluded all patients who had undergone open heart surgery or those who had neuromuscular disease which might be etiologic in the cause of SVT. These exclusion criteria may have resulted in a relative decrease in frequency of primary atrial tachycardia, which is known to be associated with these conditions.[20-23] Second, indications for study vary from center to center because there have been no generally agreed upon guidelines for either transesophageal or transvenous electrophysiology study. At our institution, transvenous study is usually reserved for patients with SVT who pose a diagnostic dilemma, or those in whom ablation, pacemaker or investigational drug therapy is considered. It is our practice to perform transesophageal studies in all pediatric patients presenting with SVT, especially when SVT is refractory to empiric antiarrhythmic therapy. This practice allowed electrophysiologic characterization of tachycardia features in many infants and young children who warranted evaluation but in whom intracardiac study would have been impractical and unwarranted.

We evaluated the age distribution of SVT mechanism both by age at symptom onset and age at time of study as a way to deal with the potential problem that a patient might have a different mechanism of tachycardia at an earlier versus later time in life. For example, an infant may have SVT using accessory connection but lose the capacity for this tachycardia during the first year of life, only to develop a propensity for tachycardia due to reentry within the AV node later in life. This possibility is supported by 2 considerations: The accessory connection may change electrophysiologic characteristics during maturation,[7,24-27] and the occurrence of tachycardia due to reentry within the AV node has been reported in patients with accessory connection.[28,29]

Study limitations: The possibility of selection bias or misclassification due to inadequate diagnostic criteria are the principal limitations of the study. Our study was limited to patients referred to a tertiary center; we estimate the indications for study at our center result in approximately 95% of patients with SVT undergoing some type of electrophysiologic study. The results of the present and previous[13] studies show that in the child and adolescent, transesophageal study can accurately determine SVT mechanisms. However, the lack of established diagnostic criteria for infants, in whom the standard multicatheter study is not feasible, poses a problem. Consequently, in the absence of an agreed-upon gold standard for infants, we relied on the classification by 3 experienced pediatric electrophysiologists.

Speculation: It was previously suggested that all infants with paroxysmal SVT may have accessory AV connections.[30] It is now generally accepted that accessory AV connections are developmental anomalies.[31] Much of the variation in prenatal and postnatal tachycardia occurrence may be explained by both developmental changes in the accessory connection[7,24] and developmental variation in the occurrence of initiating events.[32] The developmental aspects of the morphologic and functional basis of ectopic or reentrant primary atrial tachycardias in nonsurgically treated patients have been only partially described,[20,33] but based on results of the present study they may occur prenatally and postnatally throughout childhood. In the patients we evaluated, tachycardia due to reentry within the AV node occurred nearly as often as primary atrial tachycardia. However, no occurrence was detected prenatally and few cases were detected in the first year of life, suggesting an age-dependent anatomic or functional basis for tachycardia due to reentry within the AV node. Based on these considerations, we speculate that the propensity to tachycardia due to reentry within the AV node occurs during postnatal development.

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