Follow-up evaluation of infant paroxysmal atrial tachycardia: transesophageal study

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ABSTRACT  We report results of follow-up transesophageal electrophysiologic studies in 35 infants seven to 27 months old (mean, 12 months) in whom paroxysmal atrial tachycardia (PAT) using an accessory atrioventricular (AV) connection had been previously evaluated by transesophageal study in the first 2 months (mean, 14 days) of life. No infants were receiving antiarrhythmic drug therapy at the time of follow-up study. To evaluate AV conduction and initiate PAT, a standard transesophageal pacing protocol was used: single extrastimuli in sinus rhythm, incremental pacing to second-degree AV block, and burst pacing at cycle lengths near those resulting in second-degree AV block. If PAT was not initiated during the baseline period, the protocol was repeated during the infusion of isoproterenol and after administration of atropine. At follow-up study, PAT was reinitiated in 24 of 35 (68%) infants, six of whom had exhibited recent spontaneous recurrence of PAT. AV nodal function did not differ in those with and those without inducible PAT. However, when initial and follow-up studies were compared, changes in antegrade conduction of the accessory AV connection were observed, since only five of 10 infants with preexcitation at initial study continued to exhibit preexcitation at follow-up study (1/5 infants only after isoproterenol). Additionally, changes in retrograde conduction of the accessory AV connection were observed; the ventriculoatrial interval in PAT induced at follow-up study increased by 20 to 40 msec in eight of 24 infants and by 50 to 80 msec in five of 24 infants compared with the interval at initial study. Thus, in 24 of 35 infants with PAT in the first 2 months of life, susceptibility to inducible PAT persisted whether or not spontaneous PAT was observed. Since AV nodal function was similar in infants with and those without inducible PAT, the loss of susceptibility to both inducible and/or spontaneous PAT may in part be due to observed developmental changes in electrophysiologic properties of accessory AV connections.


PAROXYSMAL atrial (or supraventricular) tachycardia (PAT) is a relatively common rhythm disturbance in infants. Its consequences may be life-threatening,1 but overall it is thought to carry a good prognosis since clinically apparent episodes of tachycardia usually cease during the first year of life.2-6 Some infants with PAT are recognized to have Wolff-Parkinson-White (WPW) syndrome.5-6 However, recent findings suggest that in many other infants with PAT an accessory atrioventricular (AV) connection may play a role, even though ventricular preexcitation is absent during sinus rhythm (i.e., there is a "concealed" accessory AV connection).2-8 Thus, PAT in infants is commonly an AV reentrant or reciprocating tachycardia, with ventricular activation occurring through the normal specialized conduction system and atrial activation initiated via an accessory AV connection (orthodromic reciprocating tachycardia).

In the present study, we compare results of transesophageal electrophysiologic studies in infants who had initially presented with spontaneous PAT that appeared to utilize an accessory AV connection. Each infant underwent transesophageal electrophysiologic study at the time of presentation with tachycardia in the first 2 months of life, and a follow-up study was performed at an average age of 12 months.

Methods

We evaluated 35 infants (23 boys; 12 girls) with tachycardia first recognized prior to 2 months of age (mean, 14 days). Tachycardia was initially documented in utero in seven infants (24 to 39 weeks gestation). Postnatal electrocardiographic (ECG) documentation of spontaneous tachycardia with the following characteristics was obtained in each infant: no evidence of atrioventricular dissociation, ventricular activation initiated via the normal conduction system, and a regular heart rate of 200 to 333 beats/min (cycle length of 180 to 300 msec). During
initial hospitalization after recognition of the tachycardia, each infant was evaluated by transesophageal study to document electrophysiologic characteristics of the tachycardia. Parents were advised at the time of initial study that a request for permission for a later follow-up study would be made. Long-term antiarrhythmic therapy was initiated as deemed necessary. Infants were discharged from the hospital and followed for recurrence of tachycardia. At 6 to 27 months of age (mean, 12 months), transesophageal study was repeated on an outpatient basis after cessation of antiarrhythmic therapy.

Patients. At the time of initial evaluation prolonged tachycardia had resulted in symptoms of congestive heart failure in 21 infants; in five of 21 infants, heart failure occurred prenatally (hydrops fetalis). In the remaining 14 infants duration of tachycardia was insufficient for the development of heart failure, but seven of 14 infants had had a prolonged (>3 hr) episode of tachycardia.

Each infant underwent cardiovascular evaluation by physical examination, electrocardiography, chest roentgenography, and two-dimensional echocardiography. One infant had corrected transposition with mild Ebstein’s anomaly of the left AV valve; all other infants had structurally normal hearts. In the absence of tachycardia, all infants had normal cardiac systolic function.

After initial evaluation, antiarrhythmic drug therapy was recommended for 30 of 35 infants; in five infants with infrequent episodes of tachycardia of short duration, no therapy was recommended. Long-term antiarrhythmic regimens included digoxin (8/30 infants), digoxin and propranolol (6/30 infants), propranolol (7/30 infants), propranolol and quinidine (3/30 infants), amiodarone (3/30 infants), and verapamil (2/30 infants).

Each infant was observed in the hospital until his or her condition was stable and significant spontaneous episodes of tachycardia subsided. Before discharge from the hospital, families were instructed with respect to recognition of signs that might indicate recurrence of tachycardia. The major emphasis in this instruction was on assessment of the infant’s overall well being rather than on documentation of pulse rate. Parents were contacted at 3-month intervals for follow-up reports. In each infant antiarrhythmic drugs were discontinued a minimum of 1 month before the follow-up study, with the exception of amiodarone, which was discontinued for 3 months before study.

Initial transesophageal study. Before the transesophageal study, infants fasted for 3 hr or more. Written informed consent was obtained from the parents of each infant. All studies were performed at the infant’s bedside. Sedation was not required. A silicone rubber-coated catheter with interelectrode spacing of 22 mm (Medtronic model 6904-A) was used for recording of the transesophageal electrocardiogram and pacing. The depth of catheter insertion was estimated from the infant’s height.

With the use of an available bedside monitor with one or two ECG channels, a bipolar esophageal electrocardiogram was recorded during both sinus rhythm and tachycardia. During tachycardia, five measures each of cycle length, ventriculoatrial (V-A) interval (onset of ventricular depolarization to rapid deflection on the esophageal waveform), and AV interval (rapid deflection on the esophageal waveform to the onset of ventricular depolarization) were averaged. Records were obtained at recording speeds of 25 and 50 mm/sec. Low- and high-frequency filters were 0.5 and 40 or 100 Hz, respectively.

The technique for transesophageal atrial pacing in infants has been previously described. A custom-made programmable stimulator that delivered constant-current square-wave pulses was used for the stimulation studies. Atrial pacing threshold was determined with use of a stimulus duration of 10 msec. To ensure consistent atrial capture, subsequent stimulation was performed with a current 25% to 50% higher than threshold. Incremental pacing was performed to the cycle length at which second-degree AV block occurred. Throughout the atrial pacing sequence QRS morphology was observed for appearance, disappearance, or change in extent of ventricular preexcitation. Characteristics of antegrade conduction in the accessory AV connection were assessed by noting the minimum paced cycle length with one-to-one conduction via this connection (i.e., maintenance of preexcited QRS complex). In each infant tachycardia was initiated by use of short bursts (two to eight stimuli) of rapid stimuli at cycle lengths that resulted in second-degree AV block. During tachycardia, bursts (two to eight stimuli) at cycle lengths 10 to 80 msec shorter than the tachycardia cycle length restored sinus rhythm in all infants.

Follow-up transesophageal study. Each infant underwent follow-up study as an outpatient at age 6 to 27 months (mean, 12 months). Before the study infants fasted for 3 hr or more. Written informed consent was obtained from the parents of each infant. A peripheral intravenous line was established and infants were sedated with meperidine (2 mg/kg) or diazepam (0.1 mg/kg) intravenously as needed. A bipolar catheter identical to that employed at the initial study was used. Depth of catheter insertion was estimated from the infant’s height.

Under baseline conditions, several pacing maneuvers were performed. Incremental pacing to the point of second-degree AV block and burst pacing at cycle lengths near those resulting in second-degree AV block were performed. After every eighth sinus beat a single extrastimulus was delivered at progressively shorter (10 msec) coupling intervals so that the effective (longest A-V) interval failing to conduct and functional (shortest V-V interval observed) refractory period of the normal AV conduction system (normal QRS morphology) or accessory connection (preexcitated QRS morphology) could be determined. The minimum paced cycle length with one-to-one conduction via the accessory connection and the effective refractory period of the accessory connection were used to describe conduction and refractory characteristics of the accessory connection.

If tachycardia occurred during stimulation, interval measurements were obtained as at initial study. Sinus rhythm was restored and isoproterenol was infused at 0.02, 0.05, and 0.10 μg/kg/min; QRS morphology was examined for evidence of preexcitation (short PR interval, delta wave, and prolonged QRS duration). If tachycardia did not result during the stimulation protocol, then isoproterenol was infused for at least 2 min at each of the following infusion rates: 0.02, 0.05, an 0.10 μg/kg/min. During each infusion rate, incremental and burst pacing were performed in an effort to initiate tachycardia. If tachycardia was initiated, the stimulation protocol was terminated. If tachycardia was not initiated, isoproterenol was infused at the next higher rate. If tachycardia was not initiated by stimulation during isoproterenol infusion, the infusion was stopped and approximately 15 min later 0.04 mg/kg atropine was administered intravenously and the stimulation protocol was repeated.

Stimulation was performed with a custom programmable stimulator that delivered constant-current square-wave pulses. Three or four surface ECG leads and a transesophageal electrocardiogram were recorded on an Electronics for Medicine DR-12 recorder at paper speeds of 25, 50, and 100 mm/sec. The transesophageal electrocardiogram was recorded with low- and high-frequency filters of 10 and 500 Hz, respectively.

Esophageal recording site. In both initial and follow-up studies an electrode catheter with 22 mm electrode spacing was used. In infants, this interelectrode spacing constitutes a relatively long distance compared with atrial size. For example, the average left atrial dimension (at end-ventricular systole) in term infants is approximately 10 mm and in older infants (such as those in the follow-up study) it is approximately 17 mm. Thus,
the interelectrode spacing is large compared with the atrial dimension. In both initial and follow-up studies the electrode was positioned at a site, determined by infant height, which approximated the site of maximum esophagointal deflection and minimum atrial pacing threshold. We have assumed that in an individual infant this site is reproducibly identifiable; consequently, the esophagointal deflection on the esophageal electrocardiogram was recorded from the same relative site at both studies. Further, due to the relatively large spacing of the bipolar electrodes compared with atrial size, small changes in electrode catheter position should have little influence on the site of esophagointal recording. Thus, during tachycardia changes in VAeso intervals represent changes in ventriculointal conduction rather than changes in the site of recording.

**Statistical methods.** Student’s t test for paired or unpaired observations was used as appropriate for comparison of features of tachycardia as well as for comparison of characteristics of conduction and refractoriness.

**Results**

**Electrophysiologic features: initial study.** There was ECG evidence of ventricular preexcitation in 10 of 35 infants. Analysis during sinus rhythm showed QRS delta wave patterns suggesting left lateral (five infants), left posterior (four infants), and posterior septal (one infant) sites of ventricular preexcitation. In infants with preexcitation the minimum paced cycle length with one-to-one conduction via the accessory connection was either less than 230 msec (five infants) or greater than 350 msec (five infants).

Tachycardia with ECG features identical to spontaneously occurring episodes was initiated in each infant by transesophageal pacing. Tachycardia cycle lengths were 180 to 300 msec (mean, 227 msec); there was no evidence of AV dissociation and VAeso intervals ranged from 80 to 230 msec (mean, 115 msec). These features suggest the presence of orthodromic reciprocating tachycardia. Additionally, in 12 infants, transient left (11 infants) or right (one infant) bundle branch block was associated with prolongation of tachycardia cycle length or VAeso interval by 35 msec or more, suggesting participation of an ipsilateral free wall accessory AV connection in orthodromic tachycardia. In six infants with left free wall preexcitation sites, concordant tachycardia-related, transient left bundle branch block changes confirmed the presence of a left-sided accessory AV connection that was participating in tachycardia.

**Electrophysiologic features: follow-up study.** At the time of follow-up study, there was ECG evidence of preexcitation in four of 10 infants who had had preexcitation at initial study. In one infant preexcitation was absent in the baseline state, but during infusion of isoproterenol infusion evidence of preexcitation emerged. As shown in figure 1, an initial electrocardiogram from this infant was consistent with a left posterolateral site of ventricular preexcitation. At follow-up study at 8 months of age no evidence of preexcitation was present in the baseline state. When isoproterenol was administered, ECG changes consistent with left anterolateral preexcitation were noted. Also, ECG evidence of preexcitation emerged during the infusion of isoproterenol in two infants who had not shown preexcitation at initial study or during recording.

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**FIGURE 1.** Surface ECG leads I, II, and III at 11 days and 8 months in infant 12. During normal sinus rhythm (NSR) at 11 days of age, preexcitation was present, while at 8 months of age, it was absent. However, during infusion of isoproterenol at 8 months, preexcitation was again present. When pacing was performed at a cycle length 400 msec, the QRS morphology became normal. The electrocardiogram recorded at 11 days of age suggested a left posterolateral preexcitation site, whereas at 8 months a left anterolateral site is was indicated. It is not known whether this observation reflects maturational ECG changes resulting from preexcitation at the same site, or whether it is a manifestation of preexcitation at a second site.
of the baseline electrocardiogram at the time of follow-up study. During the infusion of isoproterenol, electrocardiograms from these two infants showed posterior septal (figure 2) and left lateral sites of preexcitation, respectively. Thus, at follow-up preexcitation was present in four infants during baseline studies and in an additional three infants during infusion of isoproterenol.

Sustained tachycardia (exceeding 50 complexes) was initiated in 18 infants during baseline studies. During infusion of isoproterenol, sustained tachycardia was initiated in six additional infants. Tachycardia was considered to be “noninducible” in 11 infants, since in spite of administration of isoproterenol and atropine, no tachycardia was initiated (five infants) or only echo beats or nonsustained tachycardia (less than five beats) was initiated (six infants). The transient nature of the latter episodes precluded any evaluation of AV or VAeso intervals. Thus, in 24 of 35 infants sustained tachycardia was initiated at the follow-up study. Tachycardia cycle lengths were 210 to 340 msec (mean, 252 msec). In 11 infants, the VAeso interval during tachycardia was ±10 msec compared with that at initial study, while in eight infants the VAeso interval increased by 20 to 40 msec, and in five infants it increased by 50 to 80 msec (figure 3). In the latter group, isoproterenol was required for initiation of tachycardia in four of five infants. In infants in whom tachycardia was initiated at follow-up, six had preexcitation during sinus rhythm. On the other hand, among infants in whom sustained tachycardia could not be initiated, only one infant had preexcitation and it was elicited only during the infusion of isoproterenol.

Changes in tachycardia cycle length. Tachycardia cycle length changed by −15 to 85 msec when the initial study was compared with the follow-up study. The average tachycardia cycle length increased from 223 ± 29 to 252 ± 33 msec (p < .05). Cycle length shortened by 4 to 15 msec in three infants (one of whom was receiving isoproterenol), but increased in every other infant. Since tachycardia cycle length is the sum of AV and VAeso intervals, we examined the relative changes that occurred in each. The mean change in VAeso interval was 24 msec and the mean change in AV interval was 5 msec. This suggests the importance of changes in VAeso interval in determin-

**FIGURE 2.** Surface ECG leads V4, V5, and V6 at 17 days and 10 months in infant 13. During normal sinus rhythm (NSR) at 17 days of age, there is no evidence of preexcitation since the ECG traces are virtually identical in sinus rhythm and tachycardia (PAT). At 10 months, tachycardia was inducible, but the VAeso had increased from 80 to 110 msec (not shown). During sinus rhythm the QRS morphology was normal, but when isoproterenol was administered, preexcitation became apparent.
ing age-related changes in tachycardia cycle length. As shown in figure 4, positive and negative AV interval changes were observed (middle panel), whereas the VAeso interval remained unchanged or increased (top panel). Further, the change in cycle length was positively correlated with both changes in AV interval (r = .61) and those in VAeso interval (r = .49; p < .05). However, the change in VAeso interval was negatively correlated with the change in AV interval (r = −.4; p < .05). These findings demonstrate that increases in tachycardia cycle length from the initial to the follow-up study were determined primarily by changes in VAeso interval.

Changes in preexcitation. Among 10 infants with ventricular preexcitation at initial study, only five of 10 infants exhibited preexcitation at follow-up study. In each case of persistent preexcitation the minimum cycle length with one-to-one conduction via the accessory connection had been less than 230 msec at the initial study. At follow-up study, the minimum paced cycle length with one-to-one conduction via the accessory connection continued to be less than 230 msec in three of five infants, but was greater than 350 msec at this time in the remaining two. Also, at follow-up two infants exhibited preexcitation during infusion of isoproterenol that had not been present at initial study or during baseline at follow-up. In both infants the minimum paced cycle length with one-to-one conduction via the accessory connection was greater than 350 msec.

Among the seven infants who had preexcitation at follow-up study, the antegrade effective refractory period of the accessory connection was less than 250 msec in two and greater than 400 msec in the remaining five.

Characteristics of infants with noninducible PAT. Sustained tachycardia could not be induced in 11 infants who were 6 to 26 (mean, 12) months of age at follow-up. Before the initial study, congestive heart failure had developed during prolonged tachycardia in three infants (prenatal, two infants; postnatal, one infant). Tachycardia cycle length, AV interval, and AVeso interval at initial study were not significantly different when the 11 infants with noninducible PAT at follow-up were compared with the 24 infants with inducible PAT (table 1). Preexcitation was present in three of 11 infants at initial study but at follow-up only 1 of 11 infants demonstrated preexcitation, and it was elicited during infusion of isoproterenol. Treatment had been similar to that received by the 24 infants with induc-
Table 1: Characteristics of tachycardia at initial study

<table>
<thead>
<tr>
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<th>Tachycardia at follow-up (n = 24)</th>
<th>No tachycardia at follow-up (n = 11)</th>
<th>p value</th>
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<tbody>
<tr>
<td>CL</td>
<td>223 ± 29</td>
<td>237 ± 34</td>
<td>NS</td>
</tr>
<tr>
<td>AV</td>
<td>110 ± 26</td>
<td>120 ± 39</td>
<td>NS</td>
</tr>
<tr>
<td>VAeso</td>
<td>113 ± 36</td>
<td>117 ± 30</td>
<td>NS</td>
</tr>
</tbody>
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CL = tachycardia cycle length at initial study; AV = AV interval during tachycardia at initial study.

months of age at follow-up study, were noted to have spontaneous recurrence of PAT following hospital discharge after the initial study. In each case, PAT recurred a minimum of 6 months after the initial PAT episode. When first recognized, tachycardia had resulted in congestive heart failure in four infants (prenatal, no infants; postnatal, four infants) and no symptoms in two infants. Preexcitation was present initially in two infants, but at follow-up only one had preexcitation. Long-term treatment began after the initial study consisted of digoxin (two infants), digoxin and propranolol (one infant), or procainamide (one infant), and two infants received no treatment. All infants with spontaneous recurrence of PAT had inducible PAT at follow-up study.

AV conduction characteristics at follow-up. Comparisons were made between the 24 infants with inducible tachycardia at follow-up study and the 11 infants with noninducible tachycardia to evaluate possible differences in AV nodal function. In the baseline state, the cycle length during sinus rhythm was 420 to 510 msec (mean, 468 msec); thus, no rate correction was used for determinations of refractory period. Effective and functional refractory periods of the AV conduction system and the minimum paced cycle length with one-to-one AV conduction were not significantly different in the two groups (table 2).

Table 2: AV conduction and refractory periods at follow-up study

<table>
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<tr>
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<th>Tachycardia at follow-up (n = 24)</th>
<th>No tachycardia at follow-up (n = 11)</th>
<th>p value</th>
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<tr>
<td>Min CL 1:1</td>
<td>243 ± 26</td>
<td>242 ± 22</td>
<td>NS</td>
</tr>
<tr>
<td>ERP AVCS</td>
<td>217 ± 40</td>
<td>208 ± 28</td>
<td>NS</td>
</tr>
<tr>
<td>FRP AVCS</td>
<td>285 ± 25</td>
<td>287 ± 18</td>
<td>NS</td>
</tr>
</tbody>
</table>

Min CL 1:1 = minimum paced cycle length with 1:1 atrioventricular conduction via normal atrioventricular conduction system; ERP AVCS = effective refractory period of atrioventricular conduction system; FRP AVCS = functional refractory period of atrioventricular conduction system.
Discussion

The principal finding of this study is that in 24 of 35 infants (68%) with spontaneous and inducible PAT during the first 2 months of life, inducible tachycardia persisted to an average age of 12 months, even though clinically apparent spontaneous tachycardia had ceased in 29 of 35 infants (83%). Changes in antegrade conduction properties of accessory AV connections were documented in some infants by loss of preexcitation or prolongation of the minimum paced cycle length with preexcited QRS morphology, while changes in retrograde conduction properties of accessory AV connections were suggested in some infants by prolongation of the VAso interval during tachycardia. AV nodal function was not different in those with inducible versus those with noninducible PAT. These findings suggest the possibility that changes in conduction properties of an accessory AV connection may explain the loss of susceptibility to spontaneous and/or inducible PAT in some infants.

Natural history of PAT and WPW in infants. The natural history of PAT and WPW syndrome in infants has been only partially described, and serial electrophysiologic studies of these disorders have not been previously reported. Thus, little is known of either the functional or morphologic changes that occur in accessory AV connections. There has been a paucity of information about the natural history of morphologic characteristics of accessory connections. Accessory AV connections appear to occur during cardiogenesis. Based on morphologic studies, the prevailing view is that accessory AV connections are uncommon in infants greater than 6 months of age, and they are not present in asymptomatic adults. It has been postulated that failure to lose accessory AV connections during normal cardiac development accounts for their postnatal presence.

While there have been no serial studies of electrophysiologic characteristics of accessory connections, ECG features of the expression of accessory connections have been the basis of serial studies: preexcitation during sinus rhythm and PAT recurrences. These studies have been reviewed recently, but several should be recounted here. Serial study of the electrocardiogram in young patients with preexcitation has usually demonstrated the loss of ECG evidence of preexcitation in about one-half. For example, Lundberg reported long-term follow-up (mean, 24 years) of 23 patients with WPW syndrome that was first noted in infancy. ECG evidence of preexcitation was present in 13 of 23 patients between the ages 2 and 10 years and continued to be present after age 10 in these 13 patients. In two patients ECG evidence of preexcitation was not present at between 2 and 10 years, but it became apparent after 10 years of age. The intermittent nature of preexcitation has been noted by others. As shown in the infants we evaluated, the antegrade refractory periods of accessory AV connections are prolonged and conduction characteristics diminished in young patients with PAT compared with those reported in older patients undergoing electrophysiologic study. Przybylski et al. evaluated 21 older patients (mean, 46 years) in whom previous ECG evidence of preexcitation had disappeared. In nine of 21 patients carotid sinus massage–induced bradycardia resulted in reappearance of preexcitation, while in five of 12 patients the infusion of isoproterenol restored preexcitation. In the infants we studied, isoproterenol infusion was similarly useful in unmasking preexcitation.

Among studies on follow-up of PAT recurrences, the study by Lundberg is remarkable because of the long follow-up period, which exceeded two decades. In 40 infants with PAT, the number of recurrences of tachycardia varied with patient age and the presence or absence of preexcitation. During the first year of life, tachycardia recurrence rates were similar for those with and without preexcitation. On the other hand, between the ages of 1 and 10 years, tachycardia recurred in about one-half of patients with preexcitation, while very few patients without ventricular preexcitation had recurrence of PAT. A similar situation existed in the second decade of follow-up, but during the third decade about one-half with and one-fourth without preexcitation suffered recurrence of tachycardia. In the infants we evaluated, the association between persistent preexcitation and spontaneous recurrence of PAT was not as strong as previously reported. This is not unexpected since preexcitation is determined in part by antegrade conduction properties of the accessory connection, whereas occurrence of tachycardia is determined in part by retrograde conduction properties.

Indications for recurrence of PAT. In patients susceptible to PAT (orthodromic reciprocating tachycardia), complex factors affecting anatomy and electrophysiologic function interact to permit recurrence of tachycardia. Dunnigan et al. recently evaluated the types of initiating events for PAT and noted age-related differences in the types of initiating events. Thus, age-related changes in initiating events may contribute to age-related changes in PAT recurrence. However, results reported in this study demonstrate changes in the functional properties of accessory connections that
may also help to explain differences in PAT recurrence rates among infants.

References
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Circulation. 1987;75:542-549
doi: 10.1161/01.CIR.75.3.542
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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