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References


Dual Atrioventricular Nodal Pathways:
A Common Electrophysiologic Response in Children

Mohinder K. Thapar, M.D., and Paul C. Gillette, M.D.

SUMMARY Electrophysiologic investigation was performed on 61 children and young adults during evaluation of either cardiac dysrhythmia or pre- or postoperative congenital heart disease. The results of these studies were reviewed retrospectively to determine if longitudinal dissociation of the atrioventricular node (AVN) was present. Dual AVN pathways were detected by the atrial extrastimulus technique, using His bundle electrograms. A discontinuous $H_1H_2$ response curve indicated the presence of dual AVN pathways. There was a higher incidence of dual AVN pathways in patients with clinically evident paroxysmal supraventricular tachycardia (PSVT) than in those without PSVT. Dual AVN pathways were equally prevalent in children with corrected and uncorrected congenital cardiac defects.

FUNCTIONAL longitudinal dissociation of the atrioventricular node (AVN) has been well documented in both animals and human adults. The presence of these fast and slow conduction pathways has been shown frequently in adults with paroxysmal supraventricular tachycardias (PSVT), in which the slow pathway conduction is usually antegrade and the fast pathway conduction retrograde, thus establishing the reentry circuit for PSVT. This is a separate group from those with extranodal accessory connections, which are also found commonly in patients with PSVT. Dual AVN pathways have been found in asymptomatic adults who have undergone electrophysiologic studies. In one series, 10% of the adults undergoing electrophysiologic studies had dual AVN pathways. Although the presence of dual AVN pathways was documented using His bundle electrograms for the first time in a child, there have been no studies to determine the frequency with which dual AVN pathways occur in children with or without congenital cardiac defects or PSVT. Therefore, we analyzed the records of children studied electrophysiologically in our laboratory to determine the frequency of occurrence of dual AVN pathways in subjects with an anatomically normal heart, with or without PSVT, and those with a congenital cardiac defect, both before and after surgery.

Materials and Methods

The records of all 61 children and young adults who had atrial extrastimulus studies in our laboratory between January 1976 and December 1977 were analyzed. These children were not included in our
previous reports, and were 8 months to 17 years old (mean ± SD 7.8 ± 5.0 years). These children were divided into three groups. Group 1 included 27 children who had anatomic normal hearts, with or without PSVT. Group 2 consisted of 16 children who had had open heart surgery for the repair of congenital cardiac defects. Group 3 included 17 children who had uncorrected congenital cardiac defects. These patients underwent electrophysiologic evaluation of either suspected or documented cardiac dysrhythmia or pre- and postoperative evaluation for congenital cardiac disease. The nature of the procedure was explained and a signed consent was obtained in all cases.

Children were sedated with meperidine 2 mg/kg, promethazine 0.5 mg/kg, and chlorpromazine 0.5 mg/kg. Cardioactive medications were discontinued at least 24 hours before the study.

A tripolar electrode catheter was introduced percutaneously into the right femoral vein and placed across the tricuspid valve for His bundle potential recording, as described previously. A quadrupolar electrode catheter was introduced percutaneously into the right or left femoral vein and positioned into the high right atrium near its junction with the superior vena cava. Three surface electrocardiographic leads and intracardiac electrograms from high right atrium and His bundle were recorded on multichannel oscillographic recorders (Electronics for Medicine) at a paper speed of 100 mm/sec. Atrial extrasystoles were given at a decreasing coupling interval of 10–20 msec during sinus rhythm and/or after nine atrial paced beats by a programmable digital stimulator (F. Bloom & Co., Philadelphia, Pennsylvania). The stimuli were twice the diastolic threshold and 2 msec in duration. During the entire procedure, femoral arterial pressure was monitored by means of an indwelling cannula.

**Electrophysiologic Definitions**

HRA, A, H, and V were high right atrial, low septal right atrial, His bundle, and ventricular responses, respectively, to either a sinus or driven beat preceding the premature stimuli. HRA, A, H, and V were high right atrial, low septal right atrial, His bundle and ventricular responses, respectively, to the premature beat. HRA, A, H, and V were high right atrial, low septal right atrial, His bundle and ventricular responses, respectively, of either single or multiple echo beats. HRA, A, H, and V were high right atrial, low septal right atrial, His bundle and ventricular responses, respectively, of the sinus beat after the premature beat.

The response of the AVN to the increasingly more premature stimulation was plotted on a graph with A intervals on the horizontal axis and H intervals on the vertical axis. A smooth curve represented a physiologic response of an AVN without functional longitudinal dissociation. In subjects with dual AVN pathways, the curve was discontinuous, as first shown by Rosen et al. (fig. 1). Dual AVN pathways were considered present if the H intervals increased by 40 msec for a decrement of 10 msec or less in the A interval, with a concomitant increase in the conduction time represented by the A interval, with no change in H intervals (figs. 2 and 3). This discontinuity of the curve was reproducible with repeated scanning. Sometimes, with similar A intervals, both short and long H intervals were observed. After the jump, the slope of the curve could be either positive or negative, clearly establishing the discontinuity of the curve. The effective refractory period (ERP) of the AVN was defined as the longest A interval that was not propagated to the bundle of His. The functional refractory period (FRP) of the AVN was the shortest H interval ob-
Figure 2. Simultaneous recording of leads I and aVF with intracardiac electrogram from high right atrium (HRA) and bundle of His (HBE₁ and HBE₁₀). Atrial premature stimulation was delivered at a decreasing interval during sinus rhythm. A) H₁H₂ of 440 msec in response to A₁A₂ of 300 msec. B) In response to an increased prematurity of 10 msec, i.e., A₁A₂ of 290 msec, the fast fibers reached their effective refractory period. However, the impulse was still conducted by the slow fibers, resulting in increase of the H₁H₂ interval by 100 msec and demonstrating the presence of dual AVN pathways.

Figure 3. Atrial extrastimulation during sinus rhythm. Premature atrial stimulation with A₁A₂ of 230 msec resulted in multiple echo beats (Aₑ) in a patient with dual atrioventricular node (AVN) pathways. The site of reentry was in the region of the AVN, as suggested by the retrograde atrial activation sequence. The low right atrium was the first to be activated, followed by the left atrium and the high right atrium. LRA = low septal right atrium; CS = coronary sinus, distal and proximal; FAP = femoral artery pressure; other abbreviations as in figure 2.
tained in response to the premature stimulation.
Criteria for the diagnosis of types of tachycardias have been described before.12, 13, 16

Results

Among the 61 children in this study, 28 (45.9%) had dual AVN pathways and 21 had PSVT. Thirteen of the 21 (61.9%) children with PSVT had evidence of dual AVN pathways. Table 1 lists the mechanism of PSVT in these subjects, as determined electrophysiologically.

Group 1 (Normal Hearts)

In this group of 27 children with structurally normal hearts, 17 presented with PSVT. Ten of these 17 had evidence of dual AVN pathways. In seven, the mechanism of tachycardia was due to reentry within the AVN. Atrial muscle reentry tachycardia was present in one and concealed accessory ventriculoatrial connection in two. The other seven with PSVT did not have demonstrable dual AVN pathways. Tachycardia in these seven was due to reentry in the AVN in one, reentry in the sinoatrial node in two, accessory atrioventricular connection in two, and ectopic focus tachycardia in two.

Of the 10 children without PSVT, seven had dual AVN pathways. A short PR interval with a normal QRS complex, sick sinus syndrome, ventricular premature depolarization and ventricular tachycardia were present in one each. Three had no associated electrocardiographic abnormalities. In the three who had no evidence of dual AVN pathways, a prolonged QT interval, sick sinus syndrome and atrial premature depolarizations were present in one each.

The ERP and FRP of the AVN of subjects with anatomically normal hearts, with and without dual AVN pathways, are shown in Table 2. There was no difference in the refractory periods at a similar cycle length between subjects with and without dual AVN pathways. Only one child in this group had a prolonged AVN ERP. He also had dual AVN pathways and AVN reentry PSVT.

Group 2 (Postoperative)

In this group of 16 patients, four had PSVT. Two of the four had dual AVN pathways. Both had PSVT due to reentry in the AVN, one after the repair of an

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**Table 1. Mechanism of Tachycardia in Subjects with Paroxysmal Supraventricular Tachycardia**

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (Normal hearts)</th>
<th>Group 2 (Postoperative)</th>
<th>Group 3 (Congenital cardiac defects)</th>
</tr>
</thead>
<tbody>
<tr>
<td>With dual AVN pathways</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AVN reentry tachycardia</td>
<td>10</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Atrial muscle reentry</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Concealed accessory ventriculoatrial connection</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Without dual AVN pathways</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AVN reentry tachycardia</td>
<td></td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Ectopic focus atrial tachycardia</td>
<td></td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Sinoatrial node reentry tachycardia</td>
<td></td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Accessory atrioventricular connection</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

Numbers refer to number of patients.
Abbreviation: AVN = atrioventricular node.

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**Table 2. Mean Effective and Functional Refractory Periods of the Atrioventricular Node in Subjects with Normal Hearts and Corrected and Uncorrected Congenital Cardiac Defects, with and Without Dual Atrioventricular Node Pathways**

<table>
<thead>
<tr>
<th></th>
<th>Effective refractory period (msec)</th>
<th>Dual AVN Functional refractory period (msec)</th>
<th>Cycle length (msec)</th>
<th>Effective refractory period (msec)</th>
<th>No dual AVN Functional refractory period (msec)</th>
<th>Cycle length (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>224 ± 53</td>
<td>341 ± 67</td>
<td>540 ± 210</td>
<td>252 ± 65</td>
<td>355 ± 76</td>
<td>603 ± 272</td>
</tr>
<tr>
<td>Group 2 (Postoperative)</td>
<td>238 ± 51</td>
<td>372 ± 45</td>
<td>602 ± 99.8</td>
<td>249 ± 66</td>
<td>368 ± 59</td>
<td>610 ± 81.5</td>
</tr>
<tr>
<td></td>
<td>(n = 6)</td>
<td>(n = 6)</td>
<td>(n = 6)</td>
<td>(n = 10)</td>
<td>(n = 10)</td>
<td>(n = 10)</td>
</tr>
<tr>
<td>Group 3 (Congenital cardiac defects)</td>
<td>236 ± 43</td>
<td>389 ± 93</td>
<td>624 ± 87.3</td>
<td>201 ± 41</td>
<td>333 ± 43</td>
<td>558 ± 89</td>
</tr>
<tr>
<td></td>
<td>(n = 5)</td>
<td>(n = 5)</td>
<td>(n = 5)</td>
<td>(n = 12)</td>
<td>(n = 12)</td>
<td>(n = 12)</td>
</tr>
</tbody>
</table>

Values are mean ± sd.
Abbreviation: AVN = atrioventricular node.
atrial septal defect and the other after aortic valve commissurotomy for aortic stenosis. The remaining two with PSVT had no evidence of dual AVN pathways. The mechanism of tachycardia in these was due to reentry in the sinoatrial node in the one who had undergone repair of tetralogy of Fallot and ectopic atrial focus tachycardia in the other, who had undergone repair of ventricular septal defect.

Of the 12 children without PSVT, four had dual AVN pathways. Two of these four with dual AVN pathways had right bundle branch block with left anterior hemiblock after closure of ventricular septal defect, and the other two had had closure of atrial septal defect, resulting in sick sinus syndrome. The remaining eight children had no evidence of dual AVN pathways. Two of these had sick sinus syndrome, one after closure of atrial septal defect and the other after repair of atroventricular canal. The remaining six had no rhythm disturbances.

ERP and FRP at similar cycle lengths (table 2) were not different in patients with and without dual AVN pathways, nor was there any difference from those in group 1. Two patients in this group had prolonged ERP and FRP of the AVN. Neither had dual AVN pathways.

**Group 3 (Congenital Heart Defects)**

In this group of 17 children with uncorrected congenital heart defects, none had PSVT. Five had dual AVN pathways with no rhythm disturbances. One each had pulmonary stenosis, ventricular septal defect, endocardial cushion defect, double outlet right ventricle, and aortic stenosis with aortic insufficiency.

None of the 12 remaining children in group 3 without evidence of dual AVN pathways had rhythm disturbances. Three had ventricular septal defect, two pulmonary stenosis, two atrial septal defect, two tetralogy of Fallot, one corrected transposition of the great arteries and one cardiomyopathy.

The FRP and ERP at similar cycle lengths were not different in those with and without dual pathways, nor was there any difference between this group and those with normal hearts and children with corrected congenital heart defects (table 2). Two patients had prolonged ERP and FRP of the AVN, one with and one without dual AVN pathways.

**Discussion**

Evidence of functional longitudinal dissociation of the AVN was first shown in experimental animals. A similar phenomenon was postulated in man to explain the ventricular echo beats, elicited by ventricular premature stimulation. Using His bundle electrograms and the atrial extrastimulus technique, Rosen et al. clearly demonstrated two pathways with different refractory periods in a child with two different PR intervals. Since then there have been several reports in adults.

Using the same method, Denes et al. found a 10% occurrence of dual AVN pathways in adult patients who underwent extrastimulus testing. The incidence of dual AVN pathways in our study group was 46%. This difference in the frequency of occurrence might be related to the selective nature of our material or different electrophysiologic properties in children than in adults. In animals, electrophysiologic properties of the conduction system have been shown to change with age. Histopathologic studies have also shown that the conduction system is immature at birth and undergoes a process of moulding and cell death until early childhood. Another possible reason for difference in the apparent frequency of dual AVN pathways is that demonstration of dual AVN pathways by the atrial extrastimulus technique depends upon the physiologic properties of these pathways. In order to show a discontinuous curve, the ERP of the fast fibers must be longer than that of slow fibers. If the ERP of fast fibers is shorter than that of slow fibers, the slow fibers could not be detected by atrial premature stimulation. Also, the FRP of the atrium must be shorter than the ERP of the slow pathway of the AVN; otherwise, dual pathways would be masked. In this situation, pacing the atria at a faster rate would shorten the FRP of the atrium, perhaps helping to unmask the presence of dual AVN pathways. DuBrow et al. showed that advancing age results in an increase of the ERP and FRP of the atria and AVN, which is independent of the cycle length. This difference in the refractory period may explain why our results differ from those of Denes.

Supraventricular tachycardia may be due to either an ectopic focus or reentry at one of several sites. The latter is more frequent in both adults and children, and reentry in the AVN is the most common, usually due to the presence of dual AVN pathways. There may, however, be another mechanism for reentry in the AVN, such as reflection, which has not been clearly defined in human beings. This study showed that the most common mechanism of PSVT in the presence of dual AVN pathways in children, as in adults, was reentry in the AVN. The presence of concealed septal atrioventricular accessory connections are difficult to exclude. Tachycardia may, however, be due to reentry at other sites, despite the presence of the dual AVN pathways, as one of 10 had reentry in the atrial muscle and two of 10 had reentry through concealed ventriculoatrial connections. In subjects without dual AVN pathways, the mechanism of tachycardia was due to reentry in the sinoatrial node or accessory atrioventricular connection or to ectopic atrial foci. There was only one child with AVN reentry tachycardia in whom dual AVN pathways were not demonstrated. These observations suggest that dual AVN pathways are frequently the reentry mechanism in AVN reentry tachycardia. We do not know if the presence of dual AVN pathways in a child would predispose to PSVT. A longer follow-up is required to answer this question.

In adults, injury to the AVN secondary to diaphragmatic myocardial infarction or intracardiac surgery may manifest itself as AVN dysfunction. AVN dysfunction may be due to functional and/or anatomic disintegration of the node. This may result
in a situation similar to that of dual AVN pathways. Comparison of the patients who had intracardiac surgery for repair of congenital cardiac defects with those who had uncorrected congenital cardiac defects showed a similar incidence of dual AVN pathways in the two groups. Therefore, our data suggest that intracardiac surgical procedures did not alter the frequency of dual AVN pathways.

Dual AVN pathways were a common electrophysiologic response in children in our study, particularly in those with PSVT. The mechanisms of PSVT in the presence of dual AVN pathways were due to reentry in the AVN. However, dual AVN pathway may also be present in asymptomatic children. Longitudinal follow-up is necessary to understand the importance of these dual AVN pathways in asymptomatic children.

References

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