Dual Atrioventricular Nodal Pathways in Children without Supraventricular Tachycardia

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Summary

Atrioventricular conduction characteristics were examined by atrial extrastimulus technique in 45 children aged 50 days to 14 years following diagnostic cardiac catheterization. None of the patients had a history of supraventricular tachycardia and none had evidence of pre-excitation on routine electrocardiogram. By plotting the A2H2 and H1H2 intervals against corresponding S1S2 intervals 2 types of conduction curve were obtained. Eight of the 45 children (17.7%) showed discontinuous conduction curves, characteristic of dual atrioventricular nodal pathways, whereas 37 patients had continuous curves. Of the 8 patients with discontinuous curves 3 showed unsustained echo beats following extrastimuli, after the effective refractory period of the fast pathway was reached. This study showed that, as in adults, the atrioventricular node was frequently functionally dissociated in children.

Additional Indexing Words:
Reentrant tachycardia Reentry Atrioventricular conduction Atrioventricular node

Supraventricular tachycardia (SVT) may be due to: 1-An automatic ectopic focus in the atrium or atrioventricular (AV) junction, or 2-A reentry mechanism. The reentry pathway may involve almost any portion of the conduction system, however it frequently utilizes the AV node. Thus in a majority of patients with SVT reentry utilizes a slow nodal pathway for antegrade conduction, and a fast nodal pathway for retrograde conduction. The purpose of this communication is to report that dual AV nodal pathways are commonly present in children without evidence of SVT or pre-excitation.
MATERIALS AND METHODS

AV conduction characteristics were studied in 45 children aged 50 days to 14 years ($7.96 \pm 3.40$ years, mean $\pm$ 1SD), during diagnostic cardiac catheterization. The underlying diseases consisted of 33 cases with congenital heart defects (tetralogy of Fallot, membranous ventricular septal defect, secundum atrial septal defect, pulmonary stenosis and various miscellaneous disorders) and 12 cases with chronic rheumatic heart disease, primarily mitral regurgitation and/or mitral stenosis. None of the patients had rhythm disturbances. The studies were performed to determine the electrophysiologic effects of various antiarrhythmic drugs on the cardiac conduction system. The results of the control extrastimulation studies were reviewed retrospectively to determine whether or not longitudinal dissociation of the AV node was present. Informed consent was obtained from the parents after complete explanation of the procedure involved. None of the children were on cardioactive drugs before the study. The studies were performed in the postabsorptive state.

All patients received premedication consisting of meperidine 1.5 mg/Kg (max 30 mg), promethazine 0.4 mg/Kg (max 10 mg), and chlorpromazine 0.4 mg/Kg (max 10 mg), 1 hour before the study. Up to 3 bipolar electrode catheters were introduced into either the superficial saphenous vein or the femoral vein on one side. Occasionally a right antecubital vein was also used. One bipolar catheter was positioned across the tricuspid valve to record the His bundle electrogram. Another catheter was used for high right atrial pacing, and delivering timed extrastimuli by a model 5325 Medtronic programmable stimulator. The third catheter was used for recording intra-atrial electrogram. All intracardiac lines as well as the surface EKG were connected to a VR8 Electronics for Medicine recorder via an isolator box. External lead I or aVF was simultaneously displayed with the intraatrial and His bundle electrograms. All recordings were obtained at a paper speed of 100 mm/sec. The patients' atria were paced ($S_1$ = pacing signal) at a rate fast enough to capture the heart and extrastimuli ($S_2$ = extrastimulus signal) were delivered in 10 msec decrements following every 8th paced beat, and continued until no intracardiac response occurred. The stimuli were twice the diastolic threshold. The $A2H2$ intervals ie the intervals between premature atrial responses ($A2$) and premature His responses ($H2$), as well as the $H1H2$ intervals ie the intervals between the last normally paced His ($H1$) and premature His responses ($H2$) were measured, for every timed extrastimulus interval ($S1S2$). Subsequently AV conduction curves were constructed by plotting the $A2H2$ and $H1H2$ intervals against corresponding $S1S2$ intervals.

Two types of AV conduction curves were recognized as first described by Rosen et al.$^{(2,3)}$: 1-Discontinuous conduction curves representing functional longitudinal AV nodal dissociation, and 2-Continuous conduction curves representing a physiologically single AV nodal pathway. Discontinuous curves were considered present, when the $H1H2$ and $A2H2$ intervals increased by 40 msec or more with a decrement of 10 msec in the $S1S2$ interval, without prolongation of $H2V2$ intervals.
Results

Eight of the 45 patients (17.7%) showed discontinuous conduction characteristic of dual AV nodal pathways (Figs. 1 and 2). Whereas 37 patients had continuous curves. Three of the 8 patients with dual AV nodal pathways showed unsustained echo beats following S2, after the effective refractory period of the fast pathway was reached. All patients tolerated the procedure well.

Fig. 1. A: This figure shows EKG, His bundle electrogram (HBE) and intra-atrial electrogram (IAE) of an 11-year-old boy with pulmonic valvular stenosis. The atrium was paced at a cycle length of 400 msec (S1S1 = 400 msec). With an atrial extrastimulus delivered at 250 msec, the A2H2 and H1H2 intervals were 110 and 300 msec respectively. B: Upon 10 msec decrement in extrastimulus timing (S1S2 = 240 msec), the A2H2 and H1H2 intervals increased remarkably to 190 and 380 msec respectively, thus pointing to the presence of dual AV nodal pathways.
Fig. 2. A and B. AV nodal conduction curves constructed for the same patient as in Fig. 1. This figure shows the response of the AV node to timed extrastimuli at 10 msec decrements (S1S2). H1H2 and A2H2 intervals are plotted against S1S2 intervals. The curves are discontinuous with a sudden “jump” in H1H2 and A2H2 intervals, pointing to the presence of dual AV nodal pathways.

DISCUSSION

Supraventricular tachycardia (SVT) may be due to: 1-An automatic ectopic focus in the atrium,4) or atrioventricular junction,5) or, 2-A reentry mechanism.1) The reentry pathway may involve the sinoatrial node,6)-8) atrium,9) AV node,3),10),11) an anomalous bypass (concealed,12) or active as in the WPW syndrome),13),14) or the His-Purkinje system.6),15),16) Despite the variable mechanisms involved, SVT is frequently due to reentry within the AV node,3),16) Reentry within the AV node is usually due to functional longitudinal dissociation of the AV node, or dual AV nodal pathways.10),11) Thus in the majority of adults and children reentry utilizes a slow AV nodal
pathway for antegrade conduction, and a fast AV nodal pathway for retrograde conduction.\textsuperscript{20,21} Occasionally however the reverse may be true.\textsuperscript{22} Electrophysiologically dual AV nodal pathways may be distinguished by a discontinuous AV conduction curve, as demonstrated by Rosen et al.\textsuperscript{21,3} Thus 2 pathways are distinguished within the AV node, i.e. a fast pathway with a long refractory period and a fast conduction velocity; and a slow pathway with a short refractory period and a slow conduction velocity. Denes et al\textsuperscript{23} reported that 10\% of adult patients who underwent electrophysiologic studies had dual AV nodal pathways. In this report we studied the functional characteristics of the AV node in 45 children with various types of congenital or acquired heart disease. None of the patients had a history of SVT and none had evidence of pre-excitation on routine electrocardiogram. The results of this study showed that 8 of the 45 children i.e 17.7\% had discontinuous AV conduction curves characteristic of dual AV nodal pathways (Figs. 1 and 2), whereas 37 patients had continuous curves. Of the 8 children with dual AV nodal pathways three had unsustained echo beats following timed extrastimuli after the effective refractory period of the fast pathway was reached. Thapar and Gillette\textsuperscript{24} recently reported the prevalence rate of dual atrioventricular nodal pathways in 61 children, divided into 3 groups. They found an overall rate of 46\%. However of their 3 groups of patients only the third group i.e those with congenital heart disease without supraventricular tachycardia is comparable to our patient material. In their third group, 5 out of 17 patients (29\%) showed dual AV nodal pathways. Like our study none of these patients (with or without dual AV nodal pathways) had supraventricular tachycardia. The reason for the different rates reported by Thapar and Gillette and the present study are not clear, however, the total number of patients, the inclusion of children with acquired heart disease and different physiological properties of the cardiac conduction system at the time of the electrophysiological studies might be responsible for the difference in the apparent frequency of dual AV nodal pathways. In summary, this study showed that as in adults, children also frequently showed longitudinal AV nodal dissociation, without clinical evidence of SVT. Despite the presence of dual AV nodal pathways, the relationships of the conduction velocities and effective refractory periods of the pathways are such that reentry is not encouraged. Only when using electrophysiologic techniques the presence of dual AV nodal pathways could be demonstrated. It is not known whether or not the presence of dual AV nodal pathways in a child would predispose to development of SVT.

Neuss et al reported that by administration of atropine or pacing the heart at various rates, cases with continuous curves could be converted into
discontinuous curves. Thus it is probable that, had provocative maneuvers been employed the prevalence rate of dual AV nodal pathways in this group of children with heart disease but without history of SVT would have been even higher than 17.7%. As these studies were performed on children with heart disease (congenital defects or acquired, mostly of rheumatic origin), these results may not be extrapolated to children with normal hearts.

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REFERENCES