His-Purkinje Conduction Findings After Cardiac Surgery in Children

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SUMMARY Conduction abnormalities of the His-Purkinje (HP) system are common sequelae of surgical repair of heart defects. Standard electrophysiologic recordings may fail to demonstrate abnormalities of the HP system when routine intervals are measured. When the conduction system is stressed, certain HP features that may indicate latent conduction disease within the HP system can be revealed. These features include split His potentials (SH) with spontaneous tachycardia or during rapid atrial pacing (RAP); SH with atrial extrastimuli (AES); and long HV intervals with AES associated with block distal to His during RAP. These findings may represent pathologic or functional properties of the HP system, and were identified in seven of 35 patients after surgical repair of tetralogy of Fallot (29 patients) or ventricular septal defect (six patients) who had electrophysiologic studies 1–8 years after operation. In five of the seven patients, these findings were revealed only when the conduction system was stressed. This incidence does not represent the true frequency of these features in the 35 patients because they were not evaluated under the same electrophysiologic stresses. Clinically, these patients did not differ from those manifesting these HP findings. The electrocardiographic patterns were as follows: normal QRS duration and normal axis (n = 1), right bundle branch block (RBBB) and normal axis (n = 1), RBBB and right-axis deviation (n = 4) (left anterior fascicular block [LAB] with tachycardia developed in one, RBBB and LAB (n = 1). The LAB was diagnosed on the basis of superior axis shift compared with the preoperative tracings. All had normal PR intervals. HP features may be unmasked by stressing the conduction system. The significance of these elicited conduction findings is unclear because data on the normal electrophysiologic properties of the conduction system in children are limited. Therefore, these features may be functional or pathologic.

CONDUCTION DISTURBANCES often occur after repair of congenital heart defects. The majority of patients who undergo transvenricular repair of tetralogy of Fallot (TOF) and ventricular septal defect (VSD) show the pattern of right bundle branch block (RBBB) on the ECG. In a smaller number (7–22%), associated left anterior fascicular block (LAB) may develop. The prognostic significance of this pattern, as well as the RBBB pattern alone, has evoked great controversy; however, the picture is becoming clarified as detailed analysis of patients with congenital heart disease and postoperative intra-ventricular conduction defects has been made and electrophysiologic studies have allowed refinements of the surface ECG interpretations. Krongrad, by comprehensive analysis of previously published studies, identified two groups at highest risk: patients in whom transient complete atrioventricular block (AV) developed postoperatively with subsequent conduction with LAB and RBBB, and patients who had trifascicular disease. Thirty percent of the patients in these groups had complete AV block or experienced sudden death.

The first group (transient AV block and subsequent RBBB-LAB pattern) can be identified on the basis of early and late postoperative ECGs. The second group (trifascicular disease) cannot be defined as easily: Although the surface ECG may show the RBBB-LAB pattern, it may not reveal the presence of AV conduction delay, because the PR interval may be normal while the HV interval is prolonged. Moreover, "trifascicular" disease may be present in the absence of LAB if there is a His bundle lesion producing nearly equal delay in all three fascicles. For the sake of simplicity, the term "trifascicular" is used in the historical sense and assumes that the intra-ventricular conduction system is composed of three discrete fascicles.

We anticipated that patients with the worst prognosis (transient AV block with LAB-RBBB pattern or trifascicular disease) would have proximal conduction disorders. That discrete lesions in the bundle of His can produce these patterns has been shown experimentally. Intracardiac electrophysiologic studies may help distinguish proximal from distal lesions; these abnormalities may be concealed, becoming manifest only when the conduction system is stressed by the use of drugs or pacing techniques. The significance of these provoked manifestations is not known and they may represent functional conduction characteristics.

In this report, we describe HP findings produced by stressing the conduction system. Although these findings may indicate HP disease, data on the normal conduction characteristics of the HP system in children are limited, precluding certitude in separating functional from pathologic findings.
Methods

Patient Population

The seven children who are the subjects of this report were selected from a series of 35 children who underwent postoperative hemodynamic and electrophysiologic studies. Patient 1 underwent transatrial closure of a VSD, while the remaining patients had a ventricular approach. Not all of the 35 patients were studied with the same electrophysiologic stresses to the conduction system, so the occurrence of abnormalities in seven of 35 does not reflect a true frequency. Stresses imposed upon the conduction system in the study group consisted of: (1) atropine administration (patient 1), (2) awake and crying recordings (patients 1 and 2), (3) rapid atrial pacing and atrial extrastimuli (patients 3–7). One catheter was used for the recordings in patients 1 and 2 during rest, atropine infusion or crying. Two catheters were used for patients 3–7 — one for recording His bundle electrograms and the other for recording and pacing from the high right atrium. Surface electrocardiographic leads I, II and III and intracardiac electrograms were recorded on a multichannel photographic recorder (Electronics for Medicine DR-16) at a paper speed of 100 mm/sec and filter settings of 40–500 Hz for the intracardiac recordings. In patients 3–7, His bundle deflections were recorded during rapid atrial pacing with atrial extrastimuli coupled to sinus and/or paced depolarizations by means of a programmed digital stimulator. (Bioelecronic and Medtronics Programmable Stimulator).

Definitions

A, H and V or A,H,V and V,V refer to low right atrium, His and ventricular deflections, respectively, originating from the patient’s basic or constant atrial paced rhythm. A2, H2 and V2 refer to deflections induced by premature atrial extrastimuli. H’ refers to a second His deflection during the patient’s basic rhythm; H’’ and H’’’ refer to second and third His deflections induced by atrial extrastimuli. A2A2, H1H2 and V1V2 intervals and functional and effective refractory periods are defined according to Wit et al. The relative refractory period of the HP system is defined according to Denes et al.22 The HV interval (normal for our laboratory 25–50 msec) is defined according to Castellanos et al.24 and the V-RV apex interval (normal for our laboratory 0–30 msec) is defined according to Sung et al.25

Results (table 1)

Preoperatively, all patients had right-axis deviation, which is the usual finding in patients with these lesions and does not indicate left posterior hemiblock. RBBB developed postoperatively in all patients, but the QRS duration in patient 3 reverted to normal by the time of the study. Left-axis deviation developed in patient 1 in conjunction with the RBBB, and left-axis deviation with tachycardia developed in patient 2. Both of these patients were presumed to have left anterior hemiblock (LAB) on the basis of superior axis shift compared with the preoperative tracings. The V-RV apex time was prolonged in five of

<table>
<thead>
<tr>
<th>Pt</th>
<th>QRS axis</th>
<th>V-RV apex interval (msec)</th>
<th>HV interval at rest (msec)</th>
<th>Maximum HV interval during stress (msec)</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>−30°</td>
<td>Not recorded</td>
<td>60</td>
<td>80</td>
<td>Split His deflections at rest (HH’ 25 msec)</td>
</tr>
<tr>
<td>2</td>
<td>+90°</td>
<td>(LAB with tachycardia)</td>
<td>50</td>
<td>45</td>
<td>Split His deflections with tachycardia (HH’ 20 msec) and normal H’V</td>
</tr>
<tr>
<td>3</td>
<td>+30°</td>
<td>39</td>
<td>35</td>
<td>130</td>
<td>AES produced three His deflections (H2, H’ and H’’) and a normal H’’’V2</td>
</tr>
<tr>
<td>4</td>
<td>+150°</td>
<td>50</td>
<td>40</td>
<td>155</td>
<td>AES produced split His deflections (H2H’2) and a normal H’’’V2</td>
</tr>
<tr>
<td>5</td>
<td>+150°</td>
<td>30</td>
<td>58</td>
<td>75</td>
<td>Long HV at rest. RAP produced a varying HV interval (45–75 msec) during AV nodal Wenekbach; HV interval was inversely proportional to the AH interval</td>
</tr>
<tr>
<td>6</td>
<td>+30°</td>
<td>50</td>
<td>40</td>
<td>100</td>
<td>AES produced prolongation of H2V2; RAP (cycle length 350 msec) produced block distal to His</td>
</tr>
<tr>
<td>7</td>
<td>+120°</td>
<td>85</td>
<td>45</td>
<td>140</td>
<td>AES produced prolongation of H2V2; RAP (cycle length 350 msec) produced Wenekbach in the node and HP system with block distal to His</td>
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Abbreviations: LAB = left anterior fascicular block; AES = atrial extrastimuli; RAP = rapid atrial pacing; AV = atrio-ventricular; HP = His-Purkinje.
Figure 1. Patient 1. Surface ECG leads I, II and III and the His bundle electrogram (HBE) at rest. There are two His deflections (H and H'). This continuous recording shows the constancy of the AH, HV and HH' intervals despite the changing amplitude of the two His deflections. In the first complex of this tracing only H' is recorded.

The six patients in whom it was measured, suggesting proximal RBBB.

Split His Deflections (patients 1–4)

Patient 1 had split His deflections (H and H') at rest (fig. 1). The AH interval was 70 msec. The HV interval was 60 msec and HH' interval was 25 msec, resulting in a normal H'V interval of 35 msec. After the administration of 0.6 mg of atropine, the AH interval shortened to 44 msec, the HV lengthened (80 msec), and the HH' remained constant (25 msec), resulting in a prolonged H'V interval of 55 msec. That the H originated from the His bundle and not from the atrium was validated by a change in the AH interval with the atropine effect. When the child awakened and cried, the AH interval shortened to 60 msec, the HV shortened to 40 msec and the HH' interval shortened to 15 msec (fig. 2).

The split His deflections at rest may indicate HB disease. The HV interval was prolonged but the H'V interval was normal at rest. The response to atropine with shortening of the AH interval is consistent with improved AV nodal conduction. Consequent upon this facilitation of AV nodal conduction, HP conduction was prolonged (HV of 80 msec with HH' of 25 msec and a prolonged H'V interval of 55 msec), because the HP system was partially refractory and conduction was slowed. The normalization of the HV and H'V intervals and the shortening of the HH' interval with crying reflects expected facilitation of conduction induced in the HP system by this naturally occurring sympathetic stimulus.

Patient 2 developed left-axis deviation and split His deflections during spontaneous tachycardia (awake recordings). The HV interval was normal (40 msec) at rest and prolonged (60 msec) with tachycardia, which represents an abnormal response.

Tachycardia-dependent LAB and RBBB in conjunction with a prolonged HV interval and a normal H'V interval in patient 2 suggested the presence of pathology in the His bundle itself. The split His deflections represent additional evidence of His bundle disease.

Patients 3 and 4 had split His deflections with atrial extrastimuli. Patient 4 had split His deflections with rapid atrial pacing. Both had normal HV intervals at rest. Figure 3 shows a series of recordings from patient...
3 during atrial extrastimulation coupled to sinus rhythm. There is (1) progressive prolongation of the H2V2 interval (figs. 3A–F); (2) decreasing amplitude and increasing duration of the H deflection (figs. 3A–F); (3) fractionation of the H deflection (H1' and H2'') (figs. 3E and F); and (4) progressive prolongation of HB conduction time (first-degree block with decremental extrastimulations). Mild sinus arrhythmia occurred during these recordings and may have contributed to the changing conduction patterns.

Patients 3 and 4 showed features that may indicate HB disease. As the HV interval prolonged there was a decrease in the amplitude and an increase in the duration of the His deflection, as described in HB conduction disease. Also, there were two or three distinct His deflections (H1, H1' and H2''). These two patients were studied by atrial extrastimuli coupled to sinus rhythm. Consequently, slight variation in cycle length may have contributed to the prolongation of the H2V2 interval when the cycle length preceding the extrastimulus was longer. It is unknown whether split His deflections induced by atrial extrastimuli are functional or pathologic.

Long HV Intervals and Pacing-induced Block Distal to His (Patients 5–7)

Patient 5 had a prolonged HV at rest (58 msec) and a normal V-RV apex time. With rapid atrial pacing he developed AV nodal Wenckebach and a varying HV interval (45–75 msec).

Patients 6 and 7 had long HV intervals with atrial extrastimuli. This first occurred at H1H2 intervals of 405 msec and 450 msec (the relative refractory period of the HP system), respectively. Figure 4 graphically shows this prolongation in patient 6. Both patients developed block distal to the His bundle deflection with rapid atrial pacing (cycle length 350 msec). Patient 6 had 2:1 block (fig. 5), while patient 7 had AV nodal Wenckebach with varying HV intervals with block distal to the His bundle.

Patient 5 had HP disease at rest (on the basis of a long HV interval) and pacing-induced block distal to the His bundle deflection, which has been regarded as functional in adult patients. Patients 6 and 7 had HP refractory periods that were somewhat longer than the values obtained.

**FIGURE 3.** Patient 3. Surface ECG leads I, II and III. His bundle electrogram (HBE) and high right atrial (HRA) electrogram during basic sinus rhythm (A1H1V1) and decremental atrial extrastimulations (A2).

(A) At a cycle length (CL) of 360 msec there is a normal H2V2 interval (35 msec). (B) At a CL of 340 msec, H2V2 prolongs (55 msec) and the H2 amplitude diminishes. (C) At a CL of 320 msec, H2V2 prolongs to 85 msec and the H2 deflection widens. (D) At a CL of 280 msec, H2V2 increases to 95 msec and the amplitude is markedly decreased. (E) At a CL of 260 msec, H2V2 increases to 130 msec and a second deflection (H2') is discernible with an H2H2' interval of 45 msec. (F) At a CL of 240 msec, the H2V2 is 130 msec and three distinct His deflections are discernible (H1, H1' and H2''). The H1H1' interval is 35 msec and the H2H2'' interval is 60 msec, resulting in a normal H1''V2 of 35 msec.
Discussion

His bundle disease induced in animals by coronary artery ligation and by direct trauma to the His bundle has resulted in a decrease in the amplitude and increase in the duration of the His deflection, as well as fractionation (splitting) into two or more deflections. Similar observations have been made in adult humans who have acquired conduction disease in the settings of trauma, arteriosclerosis, diabetes, hypertension and myocardial infarction. These studies indicate that identical electrocardiographic patterns may arise from distal bundle branch disease or from HB disease. Such electrophysiologic studies support the hypothesis of functional longitudinal dissociation of the His bundle; the fibers predestined for the bundle branch system are differentiated in the main bundle of His, and proximal injury may result in any form of bundle branch block or any combination thereof. The studies of James and Scherf provide an anatomic substrate for this functional pattern. It is likely, then, that unifascicular, bifascicular and trifascicular disease may arise from true interruption of the distal bundle branch system or from proximal interruption in the His bundle where bundle branch differentiation has already occurred. The patients in the present study had evidence of simulated trifascicular disease localized to the HB in four and to the HP or distal conduction system in three. Electrophysiologic studies on patients after repair of TOF have substantiated trifascicular disease on the basis of long HV intervals. In a limited number, the HP system has been studied by rapid atrial pacing or atrial extrastimuli.

There is evidence that trifascicular HP disease is associated with complete AV block or sudden death. But there is no evidence that trifascicular disease induced by maneuvers as reported here carries a

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**Figure 4.** Patient 6. $H_1H_2$ is plotted against $V_1V_2$ (solid circles) and $H_2V_2$ (open circles). At $H_1H_2$ intervals of 400-600 msec, the HV interval is constant (40-50 msec). At $H_1H_2$ intervals of 390, 380 and 370 msec, the HV intervals prolong to 80, 95 and 100 msec, respectively.

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**Figure 5.** Patient 6. Surface ECG leads I, II and III and His bundle electrograms (HBE) during atrial pacing at a cycle length of 345 msec. The AH interval is constant at 150 msec and there is 2:1 block distal to His.
similarly poor prognosis. In fact, long HV intervals and split His deflections with atrial extrastimuli reflect partial refractoriness of the HP system and may represent functional characteristics; however, these features are also found in association with cardiac disease. All of the patients in this study have been well during a follow-up period of 2–12 years. At present, with the limited available data on normal HP conduction in children, it is impossible to separate functional from pathologic characteristics.

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References

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