Sites of Congenital and Surgical Heart Block as Defined by His Bundle Electrocardiography


SUMMARY

Catheter recordings of His bundle electrograms were obtained in seven patients with congenital heart block (CHB) and in two with surgical heart block (SHB). In the latter two patients block occurred following total correction of tetralogy of Fallot. In six patients with CHB block occurred proximal to H. In one CHB patient block occurred in the His bundle with "split" H potentials. Intraventricular conduction was normal in five of the patients with CHB who had narrow QRS and H-V intervals ranging from 35 to 45 msec. H-V intervals were short in two CHB patients (25 and 30 msec), one of whom had QRS widening with initial slowing. In the latter patient a functioning infranodal bypass (Mahaim tract) inserting into the right ventricular septum could explain the findings. In the two patients with SHB block was distal to H with P-H intervals of 125 msec and 160 msec, respectively.

The degree of bradycardia and the occurrence of symptoms partially correlated with a location of block in or distal to the His bundle. It is suggested that recording of H potentials is useful in the evaluation of children with complete atrioventricular block.

Additional Indexing Words:
Bilateral bundle-branch block
Wolff-Parkinson-White syndrome
Mahaim tract
Intraventricular conduction
Tetralogy of Fallot

The His bundle recording technique has proved useful in delineating the site of acquired complete atrioventricular (A-V) block in adults. In most cases of chronic heart block as well as in block complicating acute anterior wall myocardial infarction the site of delay has been distal to the His bundle, probably reflecting bilateral bundle-branch block. In A-V block secondary to both digitalis intoxication and acute inferior wall infarction the site of delay has been proximal to the His bundle, presumably reflecting A-V-nodal conduction disturbance. The clinical behavior of patients with conduction disease may be partially related to the site of block.

In the present study we have applied the His bundle recording technique to delineating the site of block in children with both congenital and surgical heart block. In addition, observations are made regarding the mechanism of QRS widening which was present in one of the patients with congenital heart block.
### Table 1

**Clinical and Electrocardiographic Data in Patients with Congenital Heart Block and Surgical Heart Block**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Associated disease and surgery</th>
<th>AR/min</th>
<th>VR/min</th>
<th>QRS duration (sec)</th>
<th>QRS morphology</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>16</td>
<td>M</td>
<td>None</td>
<td>64-80</td>
<td>40-50</td>
<td>0.08</td>
<td>Normal</td>
<td>Syncope at age 12</td>
</tr>
<tr>
<td>2</td>
<td>9</td>
<td>F</td>
<td>None</td>
<td>70-114</td>
<td>35-45</td>
<td>0.08</td>
<td>Normal</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>M</td>
<td>Secundum ASD (1.7:1 shunt)</td>
<td>90-100</td>
<td>40-50</td>
<td>0.06</td>
<td>Normal</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>11</td>
<td>F</td>
<td>None</td>
<td>80-110</td>
<td>40-50</td>
<td>0.08</td>
<td>Absent Q in V₁</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>12</td>
<td>F</td>
<td>None</td>
<td>60-150</td>
<td>40-50</td>
<td>0.12</td>
<td>Delta wave in V₄</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>37</td>
<td>M</td>
<td>None</td>
<td>65-100*</td>
<td>20-40*</td>
<td>0.08*</td>
<td>Normal</td>
<td>Syncope, dizzy spells</td>
</tr>
<tr>
<td>7</td>
<td>17</td>
<td>F</td>
<td>None</td>
<td>70-180</td>
<td>32-40</td>
<td>0.06</td>
<td>Normal</td>
<td>Syncope at age 13, light-headedness, dyspnea on exertion</td>
</tr>
<tr>
<td>8</td>
<td>8</td>
<td>F</td>
<td>Total surgical correction</td>
<td>60-80*</td>
<td>10-40*</td>
<td>0.12*</td>
<td>RBBB pattern*</td>
<td>Syncope</td>
</tr>
<tr>
<td>9</td>
<td>12</td>
<td>F</td>
<td>Total surgical correction</td>
<td>70-100*</td>
<td>28-60*</td>
<td>0.12*</td>
<td>Varying RBBB* and LBBB pattern</td>
<td>Syncope, dizzy spells</td>
</tr>
</tbody>
</table>

*Prior to pacemaker insertion.

Abbreviations: AR = atrial rates; VR = ventricular rates; ASD = atrial septal defect; RBBB = right bundle-branch block; LBBB = left bundle-branch block.

### Methods

**Patients**

Seven patients with congenital heart block (CHB) and two patients with surgical heart block (SHB) were studied. Clinical and electrocardiographic data of the study group are summarized in table 1. Ages ranged from 5 to 37 years. One patient with CHB had an associated small secundum atrial septal defect. The two patients with SHB developed permanent block at the time of total correction of tetralogy of Fallot. The surgical procedure in both patients included pulmonary infundibulectomy, pulmonary valvulotomy, and repair of ventricular septal defect with a Teflon patch.

Electrocardiograms in all patients revealed complete A-V dissociation secondary to A-V block. The observed ranges of atrial and ventricular rates are listed in table 1. Six of the patients with CHB had escape rhythms with narrow QRS. QRS morphology was normal in all of these except one (patient 4), who had absence of septal Q waves in V₄ and QS complexes in V₁. One patient with CHB (patient 5) had QRS widening (fig. 1). The QRS in this patient was 0.12 second in duration and was characterized by initial slowing which was positive in lead V₄ and negative in lead V₁. The QRS pattern could be interpreted as either incomplete left bundle-branch block or type B preexcitation; in the absence of any conducted beats electrocardio-

**Figure 1**

Electrocardiogram from patient 5 demonstrating QRS widening with initial slowing suggestive of either incomplete left bundle-branch block or type B preexcitation. Note complete A-V dissociation.

graphic differentiation between these two possibilities is impossible.
Prior to surgery the SHB patients had narrow QRS with right ventricular hypertrophy. One of the patients (patient 9) had P-R prolongation. Following surgery the escape rhythms were characterized by wide QRS.

Four patients with CHB were completely asymptomatic. Two patients with CHB had histories of transient syncope several years prior to study. One patient with CHB and both patients with SHB had recurrent syncope necessitating permanent pacing.

Electrophysiologic Studies

Informed consent was obtained. Studies were performed during diagnostic cardiac catheterization in six patients with CHB and one patient with SHB. One patient with CHB and one with SHB were studied at the time of battery change.

His bundle electrograms (H) were recorded with a bipolar catheter passed via femoral vein and positioned at the tricuspid valve.3,4 Recordings were made at filter frequencies of 40 to 500 Hz on a multichannel oscillographic photographic recorder at paper speeds of 100 and 200 mm/sec. Single or multiple simultaneous electrocardiographic leads were also recorded.

P-H interval was measured when block was distal to H, and was defined as the interval from the onset of the P wave to the first high-frequency deflection of the H potential (normal 80 to 140 msec). H-V was measured when block was proximal to H and was defined as the interval from the first high-frequency deflection of the H potential to the onset of ventricular activation as measured from the earliest deflection recorded from either the electrocardiographic leads or the ventricular electrogram (normal 35 to 55 msec).

Care was taken to distinguish the right bundle-branch electrogram from the His bundle electrogram, utilizing both catheter location and the timing of recorded potentials.4 The recording of large atrial and ventricular electrograms suggests that the electrodes are atroventricular in location (position for recording of H potentials), while the recording of minimal or absent atrial electrograms and large ventricular electrograms suggests an intraventricular location of the electrodes (position for recording of RB potentials). In addition, the H-V interval was considered the longest interval recorded upon withdrawal of the electrode catheter back toward the atrium. His bundle pacing was not utilized for validation of potentials because of the possible hazards of electrical stimulation with large milliamperage with a free-floating catheter.

In six patients with CHB intervals were also recorded after the administration of 0.1 to 0.2 mg/kg of atropine intravenously. Isoproterenol was administered by intravenous drip in two of these patients (prior to atropine).

Results

Site of Block (Table 2)

Complete A-V block was present in all patients. Atrial and ventricular rates during the study are listed in table 2. Block was proximal to the H recording site in six patients with CHB (fig. 2). In these patients P waves were not accompanied by related H potentials. Escape rhythms were A-V junctional with H potentials preceding all QRS complexes.

One patient with CHB had block in the His bundle (figs. 3 and 4). In this patient each P wave was accompanied by a high-frequency electrogram (H1) with a fixed P-H1 interval of

| Table 2 |

Results of Electrophysiologic Studies

<table>
<thead>
<tr>
<th>Control heart rates</th>
<th>Intervals</th>
<th>Heart rates after drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient AR/min VR/min</td>
<td>P-H (msec) H-V (msec) Site of block</td>
<td>AR/min VR/min</td>
</tr>
<tr>
<td>1 60 48</td>
<td>37</td>
<td>Proximal to H</td>
</tr>
<tr>
<td>2 68 45</td>
<td>45</td>
<td>Proximal to H</td>
</tr>
<tr>
<td>3 62 49</td>
<td>36</td>
<td>Proximal to H</td>
</tr>
<tr>
<td>4 66 46</td>
<td>30</td>
<td>Proximal to H</td>
</tr>
<tr>
<td>5 60 44</td>
<td>25</td>
<td>Proximal to H</td>
</tr>
<tr>
<td>6 65 36</td>
<td>39</td>
<td>Proximal to H</td>
</tr>
<tr>
<td>7 68 36</td>
<td>145 (P-H1) 35 (H-V)</td>
<td>In H (“split” H potentials)</td>
</tr>
<tr>
<td>8 90 70 (paced)</td>
<td>125</td>
<td>Distal to H</td>
</tr>
</tbody>
</table>
| 9 95 70 (paced) | 160 | Distal to H | }

Abbreviations: AR = atrial rate; VR = ventricular rate; H = His bundle; isp = isoproterenol.

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Congenital A-V block proximal to H. Top tracing in each panel is the electrocardiogram and the bottom, the His bundle electrogram. Paper speed is 200 mm/sec. Time lines on this and all subsequent illustrations are at 1 second. P-wave and atrial electrograms are labeled P; QRS and ventricular electrogram are labeled R; and H potential is labeled H. Atrial rates (AR) and ventricular rates (VR) are listed, as is the H-V interval (HV). (A) Record from patient 1. Note the H potential preceding the QRS and P blocked proximal to H. H-V is within normal limits. (B) Similar recording from patient 2. H-V is within normal limits. (C) Recording from patient 4. Note slightly shortened H-V of 30 msec. (D) Record from patient 5. Note short H-V of 25 msec.

Block in the His bundle with “split” H potentials in patient 7. Shown are ECG leads V6, I, II, and III, and His bundle electrograms (HBE). A high-frequency potential (H1) follows every P wave with a P-H1 (PH1) of 145 msec. Another high-frequency potential precedes every QRS (H2) with an H2-V (H2V) interval of 35 msec. Paper speed 100 mm/sec. Other abbreviations as in figure 2.

145 msec. A second high-frequency potential (H2) was present preceding every QRS with an H2-V interval of 35 msec. P-H1 shortened with administration of isoproterenol and atropine, while H2-V remained constant (fig. 4). The findings suggested block in the His bundle with “split” H potentials as previously described by Narula and co-workers.5

In both patients with SHB the site of block was distal to the H recording site (fig. 5). Both patients were studied during ventricular pacing. Each P wave was followed by an H
potential, with a P-H interval of 125 msec in patient 8 and 160 msec in patient 9. These P waves were not conducted to the ventricles. The prolonged P-H in the latter patient presumably reflected congenital first-degree A-V block at the A-V node which predated surgery.

In summary, block in CHB was proximal to H in six patients and in the His bundle in one patient. Block in SHB was distal to the H recording site.

**Intraventricular Conduction (Table 2)**

In the five CHB patients with narrow QRS of normal configuration H-V (or H2-V) intervals ranged from 35 to 45 msec and were in the normal range (fig. 2A and B; fig. 3). Conduction distal to the site of block was considered normal.

H-V intervals were short in two patients with CHB. Patient 4 (H-V 30 msec) had a narrow QRS with loss of normal septal forces (fig. 2C). Patient 5 (H-V 25 msec) had a QRS suggestive of either incomplete left bundle-branch block or type B preexcitation (figs. 1 and 2D). The prominent initial slowing, which is directed leftward, suggests that right ventricular activation precedes that of the left. There are several possible explanations for the findings in this patient: (1) block proximal to

Response of block in the His bundle ("split" H potentials) to drug administration. Recordings from patient 7. Paper speed 100 mm/sec. (A) Control recordings. (B) Recordings following atropine administration. Note decrease in P-H1 (PH1) to 135 msec despite increase in atrial rate (AR). The ventricular rate (VR) is unchanged. (C) Recordings during isoproterenol administration (prior to atropine). Note increase in atrial and ventricular rates. The P-H1 shortens to 125 msec. Other abbreviations as in figures 2 and 3.

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H and A-V junctional escape with left bundle-branch block; (2) block proximal to H and A-V junctional escape with utilization of a preferential pathway to the right ventricle (longitudinal dissociation in the His bundle); (3) block proximal to H with right bundle-branch escape rhythm producing retrograde H potentials; (4) block proximal to H and A-V junctional escape with a functioning infranodal accessory pathway (Mahaim tract) producing right septal preexcitation. While 1 and 2 would not account for H-V shortening, 4 would. For 3 one would have to postulate, in addition, enhanced right bundle-branch automaticity or depressed A-V junctional automaticity allowing emergence of a right bundle-branch pacemaker. This seems unlikely. The increase in ventricular rate with atropine administration in this patient (see below), is also not suggestive of a right bundle-branch pacemaker. Although the data do not allow us to reach a definite conclusion, we favor 4 as the most reasonable explanation for the findings in this patient.

The findings in patient 4 have some similarity to those in patient 5 in that the H-V was slightly shortened and normal septal forces were absent. It is not certain that the abnormalities in patients 4 and 5 are due to similar mechanisms.

Ventricular Rates and Drug Responses (Table 2)

Junctional escape rates in the patients with CHB varied from 36 to 49/min (figs. 2 and 3). The slowest rates (36/min) were noted in patients 6 and 7. Both of these patients were noteworthy, the former being the oldest CHB patient in the study, and the latter for having block in the His bundle. Increased age may lead to less intrinsic rhythmicity in the escape focus. A lower escape rhythm in the patient with His bundle block may account for his slower heart rate. The escape rhythm of the patients with SHB could not be recorded because of control of the ventricles by artificial pacemakers.

Six patients with CHB received atropine. Atrial rates increased in all and ventricular rates in five. Ventricular rate did not increase in patient 7 with His bundle block (fig. 4B), suggesting that the escape rhythm originated from the more distal His bundle, an area with minimal parasympathetic control.

Patients 6 and 7 also received isoproterenol by intravenous drip, with increase in ventricular rates to 52 and 44 beats/min, respectively (fig. 4C), demonstrating that the A-V junctional pacemakers in both patients were responsive to humoral catecholamines.

Discussion

Pathologic Correlations

Congenital heart block exists as an isolated abnormality of the conduction system or may be associated with other congenital cardiac malformations. Pathologic findings accounting for A-V block have included lack of connection of the atrium with the more distal conduction system,7-11 separation of the A-V node and His bundle,12 imperfect formation or absence of the His bundle,13 and disruptive lesions in the His bundle.14-16

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Six of the patients in the present report had isolated CHB. The site of block in five of these was proximal to the His bundle recording site. This location of block is consistent with discontinuity of conduction tissue in the A-V-nodal approaches, in the A-V node, or in the A-V-nodal—His bundle connection. The His bundle recording technique cannot distinguish among these possibilities. One patient with CHB had block in the His bundle with “split” H potentials, suggesting disruption of the His bundle.

One patient with CHB had block proximal to H with an associated secundum atrial septal defect. Previous pathologic studies in two patients with secundum defects and CHB revealed lack of connection with the atrium and A-V node in one case, and necrosis of the A-V bundle and bifurcation in the second case. We would postulate that our patient had the former lesion.

The normal QRS and H-V intervals in five of the patients with CHB are consistent with a normally formed intraventricular conduction system distal to the site of block. The two patients with short H-V intervals will be discussed below.

In patients with SHB following total correction of tetralogy of Fallot, pathologic studies have revealed traumatic lesions to the His bundle and bundle branches. The demonstration of block distal to H in our two cases of SHB is consistent with these pathologic observations.

Escape Rhythms

The escape rhythms present in patients with complete A-V block are dependent on both the site of block and the relative rhythmicity of conduction tissues distal to the block. In this series all patients with CHB had A-V junctional escape rhythms with H potentials preceding each QRS. The His bundle recording technique does not allow differentiation of where in the A-V junction these beats arose other than suggesting a site of origin in the His bundle in the patient with “split” H potentials. Although catheter recordings of A-V-nodal potentials have been reported, the validity of these as actually representing A-V-nodal depolarization is still open to dispute. Thus, we have not attempted to determine the exact site of origin of escape rhythms using the presence or absence of A-V-nodal potentials. Action-potential studies suggest that A-V-nodal cells are not automatic and that automaticity in the A-V junction resides in His-Purkinje cells. Because of this, it is likely that the escape rhythms in patients with CHB originated in the His bundle.

Functional properties of the escape rhythms may be of some value in determining their site of origin. Ventricular rates in five of six patients with CHB and block proximal to H were between 44 and 49/min during the study. The oldest patient with CHB (and block proximal to H) and the patient with His bundle block had rates of 36/min. It may be that A-V junctional automaticity decreases with aging, as does that of the sinus node, accounting for the relatively slow rate in the former patient. The slow rate in the latter patient probably reflects lesser rhythmicity of a more distal His bundle pacemaker.

The atropine responses are also of interest. Five patients with CHB and block proximal to H developed increased ventricular rates with atropine. The patient with block in the His bundle did not experience an increase in rate with this drug, suggesting lack of parasympathetic control of a more distal His bundle pacemaker. Preliminary work in other laboratories using the His bundle technique has also suggested that the more distal His bundle pacemakers are unresponsive to atropine.

The increase in ventricular rate in one patient with block proximal to H and in the patient with block in the His bundle with isoproterenol is consistent with the enhancement of automaticity in His-Purkinje cells with catecholamines. The response to isoproterenol would not appear to differentiate between proximal and distal His bundle rhythms.

The two patients with SHB were studied while the ventricles were being paced, thus not allowing us to determine the nature of the escape rhythm. However, electrocardiograms prior to pacemaker insertion revealed wide
QRS complexes with slow ventricular rates, suggesting idioventricular origin. The demonstration of block distal to H is consistent with these electrocardiographic findings.

Preexcitation

Patient 5 had block proximal to H, QRS widening with initial slowing, and a short H-V interval. Although several explanations could be given for these findings (see Results), we favor right septal preexcitation via an accessory infranodal pathway (Mahaim tract) as the most likely. The findings in this patient somewhat resemble those in a patient with Wolff-Parkinson-White syndrome recently reported by Castillo and Castellanos.26 Their patient manifested a short H-V interval and delta wave in some beats. These findings were interpreted as supporting the existence of Mahaim tract conduction.

The occurrence of delta waves with complete A-V dissociation is rare. In previous cases delta waves were not persistent.27–29 If our case is an example of preexcitation with complete A-V block, it is the only case that we are aware of where this occurs as a persisting phenomenon.

Clinical Implications

Both the severity of bradycardia and the presence of symptoms referable to slow heart rates appeared to be partly related to the location of block in or distal to the His bundle. Two of the three patients needing permanent pacing had block distal to the H recording site. In addition, the patient with block in the His bundle had a history of previous syncope and very slow escape rates. Four patients with block proximal to H were totally asymptomatic. However, the correlation was not absolute in that one patient with block proximal to H needed permanent pacing because of Stokes-Adams attacks, and another had a history of two syncopal episodes.

It does seem reasonable to suggest that His bundle recording in children with heart block is of value in prognosticating individual cases and of help in determining the need for pacemaker therapy.

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