Significance of Block Distal to the His Bundle
Induced by Atrial Pacing in Patients
with Chronic Bifascicular Block

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SUMMARY Twenty-one of 496 (4%) patients with chronic bifascicular block, studied and followed prospectively, had block distal to the His bundle (BDH) induced by atrial pacing during initial electrophysiologic studies. In six, BDH was noted during pacing-induced atrioventricular (AV) nodal Wenckebach periods (at paced rates of 150–190 beats/min), with BDH in the short HH cycles after the AV nodal blocked P (long cycle). The AH interval was normal in all six patients and HV was normal in four. None of the six patients has developed AV block during a mean follow-up of 5.33 ± 0.48 years.

In 15 patients, pacing-induced BDH was noted during intact AV nodal conduction (paced rate of 80–200 beats/min). The AH interval was prolonged in one, and HV was prolonged in 10 of the 15 patients. During a mean follow-up of 3.4 ± 0.59 years, seven of these patients developed AV block, one had treadmill-provoked AV block, and two died suddenly (major morbid event in 10 of 15 patients).

In conclusion, BDH induced by atrial pacing is an infrequent finding in patients with bifascicular block, and can be a functional as well as a pathologic response. The latter is associated with a high risk of major morbid events (AV block and sudden death).

ROUTINE ELECTROPHYSIOLOGIC STUDY in patients with bifascicular block includes recording of conduction intervals (using His bundle [H] electrograms), and atrial incremental pacing. The usual response to incremental atrial pacing, is the development of atrioventricular (AV) nodal Wenckebach periodicity (block proximal to H). Block distal to H induced by atrial pacing is a much less commonly encountered phenomenon. Theoretically, this finding might suggest conduction abnormality in the His-Purkinje system, and could be associated with high risk for subsequent AV block or sudden death. There are no data concerning the prevalence of block distal to H induced by atrial pacing or its significance in terms of future course in patients with chronic bifascicular block.

In this study, we present a prospective analysis of this finding in a large group of patients with chronic bifascicular block undergoing electrophysiologic study. We report data concerning the incidence, types of block induced by atrial pacing, and the natural history of patients with this finding.

Materials and Methods

Definitions

Definitions are based on the recommendations of the criteria committee of the New York Heart Association. The criteria for electrocardiographic diagnosis of right bundle branch block included a QRS duration of 0.12 second or greater with an rSR, qR or a tall R wave in lead V1. The criteria for diagnosis of left bundle branch block were 1) QRS duration of 0.12 second or greater, 2) the presence of a broad monophasic R or Rs in lead V6, and 3) ST-segment depression and T-wave inversion in lead V6.

In patients with right bundle branch block, left anterior hemiblock was diagnosed when the mean frontal QRS axis was more negative than −30°, with a small q and tall R wave in lead I and small r and deep S wave in lead III. Left posterior hemiblock was diagnosed when the mean frontal QRS axis was more positive than +90°, with a small r and deep S wave in lead I, and small q and tall R wave in lead III and absence of right ventricular hypertrophy.

Bifascicular block was defined as right bundle branch block with left anterior hemiblock, right bundle branch block with left posterior hemiblock or left bundle branch block.

Patient Selection

Five hundred thirty-one patients with chronic bifascicular block have been detected, studied and followed in our conduction disease clinics (Cook County Hospital, West Side Veterans Administration Hospital, and the University of Illinois Hospital) between January 1970 and February 1979. These patients were detected through screening of inpatient
and outpatient ECGs in the three hospitals. In addition, some patients with bifascicular block were referred to us for electrophysiologic studies and follow-up in our conduction clinics.

Criteria for inclusion in this prospective study of intraventricular conduction defects included the following: 1) presence of chronic established bifascicular block with intact AV conduction, 2) signing of informed consent for and performance of electrophysiologic studies, 3) absence of prior second- or third-degree AV block, and 4) voluntary agreement to periodic follow-up in a conduction disease clinic. Patients with a history of prior second- or third-degree AV block and patients with acute myocardial infarction were excluded.

Initial evaluation of patients included history, physical examination, chest X-ray, serial electrograms, and routine laboratory tests. A clinical diagnosis was established for each patient using previously described criteria. Patients with normal heart size on chest roentgenogram and without signs or symptoms of organic heart disease were diagnosed as having "primary conduction disease."

Electrophysiologic Studies

Informed written consent was obtained before electrophysiologic study. His bundle electrograms were recorded in all patients using previously described catheter techniques. Cardiac drugs were withheld for at least 48-72 hours before the study. Measurements of AH (AV nodal conduction) and HV (His-Purkinje conduction) intervals were made at paper speeds of 200 mm/sec, and reflected the mean of 10 consecutive beats. The normal value (mean ± 2 sd) for the AH interval was 92 ± 38 msec, and 43 ± 12 msec for the HV interval. Atrial pacing at increasing rates, with rates from 80-210 beats/min, was performed. We noted the rate at which second-degree AV block proximal and/or distal to H occurred.

Follow-up

The follow-up was similar to that previously described. After initial study, all patients were prospectively followed in conduction disease clinic at intervals of 1-3 months. Patients were hospitalized on development of symptoms and prolonged portable electrocardiographic monitoring was used to detect suspected transient bradyarrhythmias. In patients who developed AV block, a presumptive site of block was diagnosed, using clinical and surface electrocardiographic data. Second-degree AV nodal block was presumably diagnosed when type I block was present in any combination with type II block or 2:1 block. Third-degree AV nodal block was thought to be present when the escape rhythm was relatively slower with or without QRS complexes different from previously conducted complexes. Confirmation of site of block was obtained with repeat His bundle recording, preferably during episodes of AV block. Treadmill exercise testing was performed in patients with suspected bradyarrhythmias. If AV block was provoked by exercise, the rate at which the block occurred was noted.

All deaths were thoroughly investigated and classified as sudden or nonsudden. Sudden death was defined as unexpected death due to natural causes within 24 hours of the onset of acute symptoms or within 24 hours of being seen alive without symptoms.

Information obtained during initial evaluation and subsequent follow-up was key-punched and stored on data base system computer discs. Specifically designed computer programs were used for data recall and detailed statistical analysis. Patients with pacing-induced block distal to H were compared with other patients undergoing atrial pacing studies who did not manifest block distal to H. We used a standard t test to test the significance of differences in means and a 2-by-2 chi-square method for frequency data analysis.

Results

The general characteristics of the study group have been described in detail. Patients with chronic bifascicular block (361 with right bundle branch block and left anterior hemiblock, 46 with right bundle branch block and left posterior hemiblock, and 124 with left bundle branch block). There were 424 males and 107 females, ages 18-93 years (mean ± sd 63 ± 21.2 years). Four hundred thirteen patients had clinical organic heart disease and 118 had primary conduction disease. The follow-up in these patients ranged from 21 days to 8.7 years (mean ± sem 3.5 ± 0.3 years).

In 496 patients, initial electrophysiologic study included incremental atrial pacing. Of these, 21 patients (4%) developed AV block distal to H during atrial pacing. Two patterns of pacing-induced block distal to H were discernible. Six of the 21 patients (1% of the total patients undergoing atrial pacing and 29% of patients with pacing-induced block distal to H) developed block during AV nodal Wenckebach periodicity induced by atrial pacing (see below). The remaining 15 patients (3% of the patients undergoing atrial pacing and 71% of those with pacing-induced block distal to H) developed block during intact AV nodal conduction (see below). Because of major differences in life history, these groups are described separately.

Group 1 — Block Distal to H During Pacing-induced AV Nodal Wenckebach Periodicity (tables 1 and 2; figs. 1 and 2)

Of these six patients with block distal to H during pacing-induced AV nodal Wenckebach periodicity, five were males and one was female, ages 18–73 years
TABLE 1. Data in Six Patients with Pacing-induced Block Distal to the His Bundle during AV Nodal Wenckebach

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age/sex (years)</th>
<th>Diagnosis</th>
<th>ECG</th>
<th>Electrophysiology</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>PR (sec)</td>
<td>QRS morphology</td>
</tr>
<tr>
<td>1</td>
<td>44/F</td>
<td>HCVD</td>
<td>0.16</td>
<td>RBBB, LAH</td>
</tr>
<tr>
<td>2</td>
<td>73/M</td>
<td>PCD</td>
<td>0.20</td>
<td>RBBB, LAH</td>
</tr>
<tr>
<td>3</td>
<td>58/M</td>
<td>IHSS</td>
<td>0.24</td>
<td>LBBB</td>
</tr>
<tr>
<td>4</td>
<td>18/M</td>
<td>PCD</td>
<td>0.16</td>
<td>RBBB, LPH</td>
</tr>
<tr>
<td>5</td>
<td>67/M</td>
<td>PCD</td>
<td>0.16</td>
<td>RBBB, LAH</td>
</tr>
<tr>
<td>6</td>
<td>24/M</td>
<td>PCD</td>
<td>0.20</td>
<td>RBBB, LAH</td>
</tr>
</tbody>
</table>

Abbreviations: HCVD = hypertensive cardiovascular disease; PCD = primary conduction disease; IHSS = idiopathic hypertrophic subaortic stenosis; RBBB = right bundle branch block; LAH = left anterior hemiblock; LPH = left posterior hemiblock; CL = cycle length; BDH = pacing-induced block distal to His bundle; ERP = effective refractory period; VSCS = ventricular specialized conduction system; AV = atrioventricular.

(mean 47 ± 9.2 years). Two patients (33%) had organic heart disease and four (67%) primary conduction disease. Four had right bundle branch block with left anterior hemiblock, one right bundle branch block with left posterior hemiblock and one left bundle branch block.

The AH interval in these patients was 68–100 msec (mean 84 ± 5.1 msec) and was normal in all (table 1). The HV interval was 38–69 msec (mean 50 ± 5.6 msec) and was prolonged in two of six patients (33%). The effective refractory period of ventricular specialized conduction system was 380–500 msec (mean 427 ± 19.3 msec). The longest cycle length producing AV nodal Wenckebach periodicity in these patients was 333–400 msec (heart rate 150–190 beats/min; mean ± SEM 355 ± 13.4 msec). Block distal to H (during AV nodal Wenckebach periodicity) was observed at cycle lengths of 300–400 msec (mean 352 ± 14.2 msec).

The electrophysiologic mechanism of pacing-induced block distal to H during Wenckebach periodicity has been described.16 This pattern of block is schematically shown in figure 1. Block distal to H in these six patients was functional and reflected a long

![BDH DURING ATRIAL PACING](image)

**Figure 1.** Ladder diagram showing electrophysiologic mechanisms of pacing-induced block distal to the His bundle (BDH). The upper level of each ladder diagram reflects atrium (A), the middle two levels reflect the atrioventricular node (AVN) and H and the bottom reflects the ventricle (V). The upper panel shows block distal to H during AV nodal Wenckebach periodicity. The lower two panels represent pacing-induced block distal to H with intact AV nodal conduction, the middle being type II and the bottom type I block.

TABLE 2. Comparison of Group 1 Patients with 490 Patients Undergoing Atrial Pacing

<table>
<thead>
<tr>
<th>Patients with BDH during AV nodal Wenckebach (n = 6)</th>
<th>Patients undergoing atrial pacing (n = 490)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age (mean = SEM) (years)</td>
<td>47 ± 9.2</td>
</tr>
<tr>
<td>2. Organic heart disease (%)</td>
<td>33%</td>
</tr>
<tr>
<td>3. ECG</td>
<td></td>
</tr>
<tr>
<td>A) RBBB + LAH</td>
<td>64%</td>
</tr>
<tr>
<td>B) RBBB + LPH</td>
<td>18%</td>
</tr>
<tr>
<td>C) LBBB</td>
<td>18%</td>
</tr>
<tr>
<td>4. AH interval (msec) &gt; 130 msec</td>
<td>84 ± 5.1</td>
</tr>
<tr>
<td>5. HV interval (msec) &gt; 55 msec</td>
<td>50 ± 5.6</td>
</tr>
<tr>
<td>6. CL producing Wenckebach periodicity (msec)</td>
<td>355 ± 13.4</td>
</tr>
</tbody>
</table>

Abbreviations: BDH = block distal to His bundle; RBBB = right bundle branch block; LAH = left anterior hemiblock; LPH = left posterior hemiblock; LBBB = left bundle branch block; CL = cycle length; AV = atrioventricular.
HH cycle in the His-Purkinje system due to failure of impulse propagation in the AV node, with block distal to H of the next short HH cycle (fig. 2).

Compared with the other 490 patients undergoing atrial pacing (table 2) these six patients were younger and had less organic heart disease ($p < 0.05$). There were no significant differences in sex, incidences of specific varieties of bifascicular block, mean AH, mean HV and mean paced cycle length producing Wenckebach periodicity.

**Group 2 — Block Distal to H During Atrial Pacing with Intact AV Nodal Conduction**

(Tables 3 and 4; figs. 1, 3 and 4)

In 15 of the patients, block distal to H induced by atrial pacing occurred while AV nodal conduction was intact. In 12 patients, the initiation of block could be recorded. In all of these patients, two or more paced impulses were conducted from H to ventricle before block distal to H (fig. 3). In these patients, the block

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**TABLE 3. Data in 15 Patients with Pacing-induced Block Distal to the His Bundle with Intact Atrioventricular Nodal Conduction**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age/sex (years)</th>
<th>Diagnosis</th>
<th>PR (sec)</th>
<th>QRS morphology</th>
<th>AH (msec)</th>
<th>HV (msec)</th>
<th>ERP of VSCS (msec)</th>
<th>Rate (CL in msec) for BDH (beats/min)</th>
<th>Type of block</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>49/M</td>
<td>PMD</td>
<td>0.22</td>
<td>RBBB, LAH</td>
<td>85</td>
<td>93</td>
<td>—</td>
<td>130 (462)</td>
<td>I</td>
</tr>
<tr>
<td>2</td>
<td>56/F</td>
<td>HCVD</td>
<td>0.17</td>
<td>RBBB, LAH</td>
<td>91</td>
<td>45</td>
<td>—</td>
<td>160 (375)</td>
<td>II</td>
</tr>
<tr>
<td>3</td>
<td>58/F</td>
<td>HCVD</td>
<td>0.20</td>
<td>RBBB, LAH</td>
<td>128</td>
<td>41</td>
<td>700</td>
<td>80 (750)</td>
<td>2:1</td>
</tr>
<tr>
<td>4</td>
<td>63/F</td>
<td>ASHD</td>
<td>0.18</td>
<td>RBBB, LAH</td>
<td>86</td>
<td>80</td>
<td>560</td>
<td>90 (666)</td>
<td>II and 2:1</td>
</tr>
<tr>
<td>5</td>
<td>63/M</td>
<td>HCVD</td>
<td>0.24</td>
<td>RBBB, LAH</td>
<td>68</td>
<td>78</td>
<td>330</td>
<td>200 (300)</td>
<td>II</td>
</tr>
<tr>
<td>6</td>
<td>52/F</td>
<td>PMD</td>
<td>0.20</td>
<td>RBBB, LAH</td>
<td>70</td>
<td>85</td>
<td>—</td>
<td>120 (500)</td>
<td>II</td>
</tr>
<tr>
<td>7</td>
<td>63/M</td>
<td>ASHD</td>
<td>0.24</td>
<td>RBBB, LAH</td>
<td>90</td>
<td>85</td>
<td>—</td>
<td>130 (462)</td>
<td>II</td>
</tr>
<tr>
<td>8</td>
<td>65/M</td>
<td>PMD</td>
<td>0.16</td>
<td>RBBB, LAH</td>
<td>80</td>
<td>60</td>
<td>490</td>
<td>140 (430)</td>
<td>II and 2:1</td>
</tr>
<tr>
<td>9</td>
<td>54/F</td>
<td>HCVD</td>
<td>0.24</td>
<td>LBBB</td>
<td>150</td>
<td>75</td>
<td>—</td>
<td>110 (545)</td>
<td>II</td>
</tr>
<tr>
<td>10</td>
<td>53/M</td>
<td>ASHD</td>
<td>0.16</td>
<td>LBBB</td>
<td>93</td>
<td>74</td>
<td>—</td>
<td>130 (462)</td>
<td>II</td>
</tr>
<tr>
<td>11</td>
<td>58/M</td>
<td>HCVD</td>
<td>0.16</td>
<td>RBBB, LAH</td>
<td>79</td>
<td>77</td>
<td>—</td>
<td>90 (666)</td>
<td>I</td>
</tr>
<tr>
<td>12</td>
<td>65/F</td>
<td>HCVD</td>
<td>0.16</td>
<td>LBBB</td>
<td>74</td>
<td>76</td>
<td>490</td>
<td>190 (316)</td>
<td>II</td>
</tr>
<tr>
<td>13</td>
<td>15/M</td>
<td>TOF</td>
<td>0.16</td>
<td>RBBB, LAH</td>
<td>105</td>
<td>44</td>
<td>—</td>
<td>140 (430)</td>
<td>2:1</td>
</tr>
<tr>
<td>14</td>
<td>75/M</td>
<td>PCD</td>
<td>0.18</td>
<td>RBBB, LAH</td>
<td>77</td>
<td>42</td>
<td>530</td>
<td>150 (400)</td>
<td>2:1</td>
</tr>
<tr>
<td>15</td>
<td>75/M</td>
<td>HCVD</td>
<td>0.18</td>
<td>RBBB, LAH</td>
<td>75</td>
<td>55</td>
<td>—</td>
<td>140 (430)</td>
<td>2:1</td>
</tr>
</tbody>
</table>

**Abbreviations:** BDH = pacing-induced block distal to His bundle; CL = cycle length; HCVD = hypertensive cardiovascular disease; ASHD = arteriosclerotic heart disease; PMD = primary myocardial disease; PCD = primary conduction disease; TOF = tetralogy of Fallot; RBBB = right bundle branch block; LBBB = left bundle branch block; LAH = left anterior hemiblock; ERP = effective refractory period; VSCS = ventricular specialized conduction system.
was type I in two, type II in seven, 2:1 in one, and type II as well as 2:1 in two. In the remaining three patients, initiation of pacing-induced block was not recorded; only 2:1 block distal to H was recorded (fig. 4). Because the initiation of block in these three patients was not recorded, the mechanisms of block remain uncertain. It is possible that 2:1 block in them reflected a functional phenomenon, with the long-short sequence being established by the coupling interval of the first of the sequence of atrial paced beats.17

Of these 15 patients, nine were males and six females, ages 18–75 years (mean 59 ± 3.6 years). One patient (7%) had primary conduction disease and 14 had organic heart disease (93%). Patients 3, 5 and 7 had a history of syncope (single episode), patients 9 and 13 had intermittent dizziness, and patients 4 and 15 had chronic congestive failure. The cycle length producing block distal to H with intact AV nodal conduction in the 15 patients ranged from 300–750 msec (heart rate 80–200 beats/min), with a mean of 478 ± 20.1 msec. Twelve had right bundle branch block with left anterior hemiblock and three had left bundle branch block.

The AH interval in these 15 patients was 68–150 msec (mean 91 ± 5.8 msec) and was prolonged in one. The HV interval was 41–93 msec (mean 67 ± 4.6

<table>
<thead>
<tr>
<th>TABLE 4. Comparison of Group 2 Patients with 481 Patients Undergoing Atrial Pacing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with BDH during intact AV nodal conduction (n = 15)</td>
</tr>
<tr>
<td>--------------------------------------------------------------</td>
</tr>
<tr>
<td>Age (mean ± SEM) (years)</td>
</tr>
<tr>
<td>Organic heart disease (%)</td>
</tr>
<tr>
<td>ECG</td>
</tr>
<tr>
<td>A) RBBB + LAH</td>
</tr>
<tr>
<td>B) LBBB</td>
</tr>
<tr>
<td>AH interval (msec)</td>
</tr>
<tr>
<td>&gt; 130 msec</td>
</tr>
<tr>
<td>&gt; 55 msec</td>
</tr>
</tbody>
</table>

Abbreviations: BDH = block distal to His bundle; RBBB = right bundle branch block; LAH = left anterior hemiblock; LBBB = left bundle branch block; CL = cycle length; AV = atrioventricular.

Figure 3. His bundle (H) recordings that show pacing-induced block distal to H with intact atrioventricular (AV) nodal conduction. The upper panel shows type II and lower panel type I second-degree AV block at pacing rates of 130 beats/min and 90 beats/min, respectively. HBE = His bundle electrogram; HR = heart rate.
msec) and was prolonged in 10 of 15 patients (67%). The effective refractory period of the ventricular specialized conduction system measured in six patients was 330–700 msec (mean 517 ± 48.9 msec), and was more than 600 msec in one patient.

Comparison of these 15 patients with the remaining 481 patients undergoing atrial pacing (table 4) revealed no significant differences in age, sex, varieties of bifascicular block and mean AH interval. However, these 15 patients had a significantly longer mean HV interval and greater incidence of HV prolongation ($p < 0.05$).

**Natural History**

**Group 1 — Block Distal to H During AV Nodal Wenckebach Periodicity**

The follow-up in this group was 4.1–7.4 years (1496–2687 days), with a mean of 5.3 ± 0.5 years. All six patients are living and have not developed AV block. In one of the patients, a prophylactic pacemaker was implanted at another medical center by his family physician for HV prolongation. This patient has not developed documented AV block.

**Group 2 — Block Distal to H During Atrial Pacing with Intact AV Nodal Conduction (table 5)**

The follow-up in this group was 0.25–7 years (91–2544 days), with a mean of 3.4 ± 0.6 years. Spontaneous second- or third-degree AV block developed in seven of these 15 (47%) patients on days 5, 17, 60, 141, 995, 1727 and 1854 of follow-up. The site of block (presumptive, as determined by clinical and electrocardiographic data or definite, as determined by His bundle studies) was trifascicular (distal to H) in six and AV nodal (proximal to H) in one. In the latter patient, the diagnosis of the site of block had been based upon ECG criteria only (documentation of type I, second-degree AV block). The site of block could very well be distal to H, because this patient had

**Table 5. Follow-up Data in Patients with Pacing-induced Block Distal to the His Bundle During Intact Atrioventricular Nodal Conduction**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Day of AVB</th>
<th>Presenting symptom with AVB</th>
<th>Degree (type) of AVB</th>
<th>Site of AVB</th>
<th>Therapy</th>
<th>Death (sudden or nonsudden)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1854</td>
<td>CHF</td>
<td>2° (I)</td>
<td>AV Nodal*</td>
<td>PMR</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>1727</td>
<td>Syncope</td>
<td>2° (II) and 2:1</td>
<td>Trifascicular*</td>
<td>PMR</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>Syncope</td>
<td>2° (II)</td>
<td>Trifascicular*</td>
<td>PMR</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>995</td>
<td>CHF</td>
<td>2° (II)</td>
<td>Trifascicular†</td>
<td>PMR</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>60</td>
<td>Syncope</td>
<td>3°</td>
<td>Trifascicular*</td>
<td>PMR</td>
<td>(Sudden, day 173)</td>
</tr>
<tr>
<td>6</td>
<td>17</td>
<td>Syncope</td>
<td>2° (II)</td>
<td>Trifascicular†</td>
<td>PMR</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>141</td>
<td>Syncope</td>
<td>2° (II)</td>
<td>Trifascicular†</td>
<td>PMR</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td>3</td>
<td>—</td>
<td>2° (II) (exercise-provoked)</td>
<td>Trifascicular*</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>(Sudden, day 1240)</td>
</tr>
<tr>
<td>10</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>(Sudden, day 1377)</td>
</tr>
</tbody>
</table>

*Site of AV block was established by clinical and electrocardiographic findings.
†Site of AV block was established by His bundle studies.

Abbreviations: AV = atrioventricular; AVB = atrioventricular block; CHF = congestive heart failure; PMR = permanent pacemaker insertion.
markedly prolonged HV interval (93 msec) and type I atrial-paced block distal to H at initial electrophysiologic study. One additional patient had second-degree AV block on treadmill exercise upon achieving a sinus rate of 140 beats/min, which was identical to the paced rate that induced block distal to H. Several 24-hour Holter recordings during subsequent follow-up have not revealed spontaneous AV block in this patient.

All of the seven patients who developed spontaneous AV block were permanently paced (five for syncope and two for cardiac failure) and are without symptoms. Six of the seven patients with AV block are currently alive; one died suddenly (while paced). The patient with treadmill-provoked block is not paced and is alive and well. Of the seven patients who did not develop spontaneous AV block or treadmill-provoked block, three are alive and four dead. Of the latter, two died suddenly, on days 1240 and 1377. One of the two patients (no. 9) who died suddenly had multiple premature ventricular contractions (without any documented episode of ventricular tachycardia) on various resting ECGs.

In summary, of 15 patients who developed pacing-induced block distal to H with intact AV conduction, 10 (67%) have developed either spontaneous AV block, treadmill-provoked block or died suddenly. The HV interval was prolonged in eight of these 10 patients (80%) and was normal in two (20%) (NS). The effective refractory period of the ventricular specialized conduction system was more than 600 msec in only one of these 10 patients.

AV block (including treadmill-provoked block) or sudden death occurred in eight of 10 patients (80%) with block distal to H induced by atrial pacing at rates of 140 beats/min or less and in two of five patients (40%) at rates greater than 140 beats/min (NS).

**Relationship of Pacing-induced Block Distal to H to Spontaneous Occurrence of Trifascicular Block**

In our series of 496 patients, 10 (2%) had developed spontaneous block distal to H as of February 1979. Of these 10, six (60%) were predicted by demonstration of pacing-induced block distal to H during intact AV nodal conduction. In contrast, the incidence of spontaneous block distal to H in patients undergoing atrial incremental pacing study without manifesting block distal to H was less than 1% (four of 496 patients).

**Discussion**

The progression of bifascicular block to advanced or complete AV block has been shown by many workers. The reported incidence of progression has ranged from 1–62% in a large number of retrospective studies on the natural history of bifascicular block. We have recently reported a 4.4% incidence (20 of 452 patients) of spontaneous AV block in a large group of prospectively followed patients with chronic bifascicular block. Similar studies by Scheinman et al., Kulbertus et al., and McAnulty et al. confirm the low incidence of progression in these patients.

Attempts to delineate factors determining future risk of advanced AV block and sudden death in patients with bifascicular block have not proved useful. Some of the factors include: presence of disease in the remaining functioning fascicle, associated organic heart disease, advanced age and neurologic symptoms.

Recording of His bundle electrograms in patients with bifascicular block provides a means for assessing conduction properties in the remaining functioning fascicle. The HV interval in these patients is a measure of conduction in the distal His bundle and the third functioning fascicle. Theoretically, an abnormally prolonged HV interval associated with bifascicular block could relate to the future occurrence of AV block and sudden death. The value of HV prolongation as predictor of future development of AV block in these patients is controversial. Studies by Narula et al., Vera et al., and Scheinman et al. suggest that HV prolongation in patients with chronic bifascicular block is associated with increased risk of advanced AV block and sudden death. Studies by Denes et al. and McAnulty et al. reported an identical incidence of spontaneous AV block in bifascicular block patients with and without HV prolongation. The incidence of sudden death was also reported to be identical in the two groups. In another study from our laboratory, it was shown that even a markedly prolonged HV interval (80 msec or greater) in patients with chronic bifascicular block was associated with a low incidence (5%) of subsequent AV block. These observations suggested that the measurements of HV interval in patients with bifascicular block did not predict future development of AV block.

Little is known about the effect of associated cardiovascular disease on progression of conduction disease in patients with chronic bifascicular block. In a recent study comparing bifascicular patients with and without organic heart disease, the incidence of spontaneous AV block was found to be higher in patients with organic heart disease. In another recent study, it was concluded that severity and progression of conduction disease were independent of advanced age.

The occurrence of syncope in patients with chronic bifascicular block could suggest intermittent failure of the remaining functioning fascicle with subsequent complete AV block. Recent studies suggest that syncope in patients with chronic bifascicular block reflects various cardiac and noncardiac causes. AV block was not the most common cause of syncope. The electrophysiologic measurements as well as incidences of sudden and nonsudden deaths were identical in patients with and without syncope, suggesting that syncope in patients with bifascicular block was also of little prognostic value.

Atrial pacing at incremental rates is often performed as part of electrophysiologic studies of patients with bifascicular block in the cardiac catheterization laboratory. This provides provocative
stress of the conduction system with rapid rates, allowing demonstration of conduction disease not apparent during sinus rhythm. Atrial pacing at rapid heart rates generally produces type I second-degree AV block (AV nodal) proximal to H. Rosen et al. suggested that development of block distal to H with rapid atrial pacing is an abnormal finding implying conduction abnormality in the His-Purkinje system. This was based upon observations during atrial pacing in four patients with first-degree AV block and left bundle branch block, all having prolonged HV intervals. In a retrospective study of 50 patients with intermittent second- or third-degree AV block and bifascicular block, Vera and co-workers showed pacing-induced block distal to H in four patients during 1:1 conduction. No information based on prospective analysis of the significance of pacing-induced block distal to H in patients with bifascicular block is available.

In the present study, two mechanisms of block distal to H induced by atrial pacing were observed. One was the occurrence of block during pacing-induced AV nodal Wenckebach periodicity with block distal to the H in the short cycles after completion of Wenckebach periods. Failure of impulse propagation at the AV node (during Wenckebach periodicity) produced long cycle lengths in the His-Purkinje system (long HH interval), with associated long His-Purkinje refractory period in the subsequent cycle (Ashman phenomenon). This prolongation of His-Purkinje refractoriness resulted in block distal to H of the second beat of the next Wenckebach sequence (short HH cycle). This long-short sequence giving rise to functional block distal to H was observed in approximately 25% of the patients with block distal to H induced by atrial pacing (1% of our total series). Our results suggest that this response does not represent a pathologic process in the third functioning fascicle because it is not associated with risk of subsequent AV block or sudden death mortality. Several electrophysiologic features were of interest in this group: normal AV nodal function, relatively normal HV interval and paced rates of 150 beats/min or greater inducing block distal to H.

The second mechanism was the occurrence of pacing-induced block distal to H with intact AV nodal conduction. This was the most frequent response, occurring in approximately 75% of the patients with pacing-induced block distal to H. This response presumably reflects intermittent failure of conduction through the functioning fascicle leading to intermittent trifascicular block. In most of the patients (10 of 15), the atrial rates producing AV block were 140 beats/min or less. Other important features in this group were relatively normal AV nodal function and presence of prolonged HV interval in most of the patients (67%). The mean HV interval and incidence of HV prolongation were significantly longer in this group than in patients undergoing atrial pacing study who did not manifest block distal to H.

The results of the present study show that pacing-induced block distal to H with intact AV nodal conduction is associated with relatively high risk of subsequent AV block and sudden death. It was not surprising that the site of subsequent AV block was trifascicular (distal to H) in all except one. A definite site of block was not delineated in all the patients developing AV block. Therefore, our conclusions regarding the frequency and implications of site of AV block remain tentative. However, the prior presence of prolonged HV interval in five of our seven patients developing trifascicular block suggests that our presumptions about the site of block were probably accurate. Sudden death in two of the patients could have reflected sudden development of trifascicular block with resultant asystole. The absence of significant morbidity and mortality in the three patients with 2:1 block distal to H induced by atrial pacing, in whom initiation of block was not recorded, may have reflected a functional finding, with long-short phenomenon being established by initiation of atrial pacing.

As mentioned above, our large prospective study of patients with chronic bifascicular block has not yet shown that HV interval prolongation is a predictor of future trifascicular block. The results of the present study suggest that pacing-induced block distal to H during intact AV conduction is a useful prognostic indicator. In our series, this finding predicted over half of total patients eventually developing trifascicular block. It is likely that the HV interval and pacing-induced block are measurements of different aspects of conduction. While HV is a measure of His-Purkinje conduction time at basal states, the pacing-induced block is a measure of the ability of repetitive conduction under stress. Conduction system disease can produce abnormalities of conduction velocity (time) and/or ability to conduct repetitively at rapid heart rates. Discordance of conduction time and refractoriness in the His-Purkinje system have been reported by Wu and co-workers.

Although the pacing-induced block distal to H with intact AV nodal conduction implies disease in the His-Purkinje system, failure to show similar block with pacing does not necessarily imply the absence of such disease. In some patients, AV nodal dysfunction may limit AV conduction and prevent rapid impulse penetration into the His-Purkinje system, thus preventing demonstration of block. In other patients, the diseased His-Purkinje system may be unpredictable and may conduct at rapid rates on one occasion but fail to conduct at similar rates on another occasion.

Clinical Implications

Block distal to H induced by atrial pacing is an infrequent finding, occurring in approximately 4% of electrophysiologic studies in patients with chronic bifascicular block. The pacing-induced block distal to H can be both a functional response (group 1) and a pathologic response (group 2). When pacing-induced block distal to H occurs during pacing-induced AV nodal Wenckebach periodicity, it appears not to be associated with risk of AV block or sudden death.
When it occurs with intact AV nodal conduction, pacing-induced block distal to H is associated with a high risk of development of trifascicular block and sudden death. This finding appears more sensitive for predicting trifascicular block than HV prolongation, marked HV, prolongation or presence of symptoms.\(^6\), \(^8\), \(^9\) Most of the patients in the present series were hospitalized patients. The incidence and implications of pacing-induced block distal to H might be different in a group of healthy subjects with chronic bifascicular block or in patients with neurologic symptoms.

The results of the present study raise several difficult questions regarding indication for routine electrophysiologic study in patients with chronic bifascicular block. Because block distal to H induced by atrial pacing appears to predict subsequent AV block, one can ask whether all patients with chronic bifascicular block should be subjected to His bundle recording, including atrial incremental pacing. Using the present series as an example, we would have had to study 496 patients with chronic bifascicular block in six cases (of 10 patients developing spontaneous trifascicular block), with a mean follow-up of approximately 4 years. Prophylactic pacing would have been initiated in 15 patients shortly after the initial electrophysiologic study. This would probably have prevented the syncopal episodes only in five patients and congestive failure in two patients (presenting symptoms associated with development of AV block). Because two patients with pacing-induced block distal to H died suddenly, it is possible that prophylactic pacing might have prevented these sudden deaths. Prophylactic pacing would also have been initiated in six other patients who have not yet developed spontaneous AV block. Although electrophysiologic study, including atrial pacing, appears very promising, the diagnostic yield is too low to use these tests in asymptomatic patients.

Electrophysiologic study, including atrial pacing, would be of value in patients with chronic bifascicular block and unexplained dizziness and/or syncope. Pacing-induced block distal to H with intact AV nodal conduction would strongly implicate AV block as the cause of symptoms, and would then be an indication for permanent pacing.

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Electrophysiologic Identification of Dual Atrioventricular Nodal Pathway Conduction in Patients with Reciprocating Tachycardia Using Anomalous Bypass Tracts

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SUMMARY In 67 consecutive patients with reciprocating tachycardia using an anomalous atrioventricular (AV) or nodoventricular (NV) bypass tract, electrophysiologic findings suggested the coexistence of dual AV nodal pathway conduction in eight patients. The evidence of coexistent dual AV nodal pathways and anomalous bypass tracts took three forms. In four patients, alternating short and long AV nodal conduction time (AH intervals), presumably caused by rate-dependent 2:1 conduction in the fast AV nodal pathway, were recorded during AV reciprocating tachycardia. Intravenous administration of atropine invariably resulted in 1:1 fast AV nodal pathway conduction during tachycardia in all patients. In three other patients who had anomalous AV bypass tracts capable of only retrograde conduction, discontinuous AV nodal conduction curves (A1A2, H1H2) were generated during atrial extrastimulation. The remaining patient had an anomalous NV bypass tract bridging the slow AV nodal pathway and the right ventricle. During atrial extrastimulation, antegrade block in the fast AV nodal pathway caused antegrade conduction across a pathway composed of the slow AV nodal pathway and the NV bypass tract. This suddenly produced right ventricular preexcitation with inscription of the His bundle deflection within the QRS complex, disrupting the AV nodal conduction curves (A1A2, H1H2). A sustained reciprocating tachycardia with a complete left bundle branch block pattern was subsequently initiated using the slow AV node-NV bypass tract pathway for antegrade conduction and the fast AV node-His-Purkinje system pathway for retrograde conduction. These observations suggest that dual AV nodal pathway conduction can be identified electrophysiologically in patients with reciprocating tachycardia involving anomalous bypass tracts, but its manifestations may take several forms.

DUAL ATRIOVENTRICULAR (AV) nodal pathway conduction is a common electrophysiologic phenomenon. Characteristically, discontinuous AV nodal conduction curves (A1A2, H1H2) are generated with programmed atrial extrastimulation. However, with an anomalous bypass tract, anomalous AV or nodoventricular conduction may preclude the induction of such characteristic AV nodal conduction curves (A1A2, H1H2). Therefore, the dual AV nodal pathway conduction may not be recognized during electrophysiologic evaluation of patients with anomalous AV or nodoventricular bypass tracts.

In this study we examined 67 consecutive patients with reciprocating tachycardia using an anomalous bypass tract. We have tried to identify and characterize the electrophysiologic findings suggestive of coexisting dual AV nodal pathway conduction in these patients.

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